45/12

1980

NEWARK PUBLIC LIBRARY

AUG 1 9 1981

U. S. GOVT. PUBS.
DEPOSITORY 376

Bibliography on Smoking and Health

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20.11:

ISSN: 0067-7361

1980

Bibliography on Smoking and Health

DEPARTMENT OF HEALTH & HUMAN SERVICES USA

WORLD HEALTH ORGANIZATION
COLLABORATING CENTRE
FOR REFERENCE ON
SMOKING AND HEALTH

Compiled by:
Informatics Inc.
6011 Executive Boulevard
Rockville, MD 20852
Under Contract No. 282-79-0101-LG

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402

PREFACE

Abstracts of current literature are published by the Office on Smoking and Health (OSH) in the *Smoking and Health Bulletin* every 8 weeks and are printed annually with a cumulative author and subject index as the *Bibliography on Smoking and Health.* The current edition of the Bibliography has 1,850 abstracts. All items cited are part of the permanent holdings of OSH and are maintained in its Technical Information Center (TIC). The technical collection consists of over 30,000 documents, the majority published since 1960. Earlier editions of the Bibliography, from 1968 through 1979, are available from the TIC as long as supplies last.

The Technical Information Center offers bibliographic and reference service to researchers and others, as well as publishing and distributing a number of titles in the field of smoking. In addition to the Bulletin and the Bibliography, these publications are the biennial *Directory of On-Going Research in Smoking and Health,* the annual *Health Consequences of Smoking* report, and *State Legislation on Smoking and Health.* Current copies of each can be obtained by writing or calling the TIC. Back issues of some titles are also available.

The Technical Information Center has computer capability, through its Automated Search and Retrieval System, to generate comprehensive bibliographic print-outs on topics of current interest in smoking and health. Persons wishing to make use of this free literature search service are requested to complete a Search Request Form for this purpose. Forms can be found in current issues of the *Smoking and Health Bulletin* or may be requested from the TIC.

Information regarding the availability of articles listed in the Bibliography or services and publications of the Office on Smoking and Health may be obtained by writing or calling the Technical Information Center.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Phone: 301-443-1690

CONTENTS

AGRICULTURE 1,46,114,149,191,235,290,352

CHEMISTRY 2,50,150,191,236,290,354

PHARMACOLOGY AND TOXICOLOGY 7,57,154,194,240,294,357

MORTALITY AND MORBIDITY 14,67,163,202,251,307,370

NEOPLASTIC DISEASES 16,69,164,202,252,309,371

NON-NEOPLASTIC RESPIRATORY DISEASES 19,75,166,206,257,312,374

CARDIOVASCULAR DISEASES 23,81,171,211,263,318,380

PREGNANCY AND INFANT HEALTH 27,91,175,218,270,326,388

OTHER DISEASES AND CONDITIONS 29,93,176,218,271,329,390

BEHAVIORAL AND EDUCATIONAL RESEARCH 30,95,178,220,273,331,392

SMOKING CESSATION AIDS 145

SMOKING CESSATION METHODS 34,104,183,223,277,341,402

TOBACCO ADDITIVES 106,130,278,405

TOBACCO CONSTITUENT ALTERATION 126

TOBACCO MANUFACTURING AND PROCESSING 37,107,115,185,225,279,343,406

TOBACCO SUBSTITUTES 140

TOBACCO ECONOMICS 37,107,186,225,281,344,408

BILLS AND LEGISLATION 38,108,186,230,346

GENERAL 38,109,146,187,230,283,347,409

AUTHOR AND ORGANIZATIONAL INDEX 413

SUBJECT INDEX 439

AGRICULTURE

80-0001. DeBaets, A.; Weber, K. H. **The Decline of Residues From Plant Protectives on Tobacco.** *Tabak-Journal International* 6: 403-404, 406, December 1977.

There has been a drastic decline in DDT residues (from 22.8 mg/kg in 1969 to 0.5 mg/kg in 1976) in tobacco imports from 19 important suppliers of the European community. Similar trends have been observed in the decrease of residues from Aldrin, Chlordane, Dieldrin, Endrin, Heptachlor, and Heptachlor-epoxide. Benzenehexachloride (BHC), however, is a special case and still remains a problem. More and more tobacco growing countries are now using organophosphorus insecticides (Malathion, Parathion). Limited data are available for residues, but usually residues are below 1 mg/kg. Carbamates and dithiocarbamates are also widely used. Residue levels from these insecticides are also believed to be low. Provided that the rules of good agricultural practice are observed, tobacco can be purchased from the most important tobacco producing countries largely free from undesirable residues. In order to reach this target all over the world, suggestions for uniform international measures have been submitted to the European Community Commission.

80-0002. Giambiagi, N.; Couto, M. R.; Figueroa, P. **Dynamic of Nitrification in Tobacco Soil of Lerma Valley, Salta, Rep. Argentina.** *Agrochimica* 21(1/2): 79-89, January-March, 1977.

The dynamics of nitrification in tobacco soil was studied at the Experimental Station of Salta during 1970-71 at two different depths: 0-20 and 20-40 cm. Nitrification was low in winter, began to increase in August, reached maximum values in December, and decreased to initial levels by the end of the summer. Annual averages of nitrates and organic nitrogen obtained at 0-20 cm were 21.4 ppm and at 20-40 cm were 10.8 ppm. Clean fallow had a greater effect on nitrogen mineralization than black fallow, whereas stubble introduced 1 month prior to tobacco transplant inhibited nitrification. Rotation with gramineous cultures seemed to extend the maximum nitrate production period 2 months. The principal ecological factors that seemed to influence the seasonal variation were temperature, humidity, and oxygen. (Auth. Abs. Mod.)

80-0003. Gopalachari, N. C.; Murty, K.S.N.; Prasad Rao, J.A.V.; Prasad, S. S. **Study on the Relationship of Fertilizer Levels and Seasonal Variations to the Chemical Composition and Nutrient Uptake by Flue-Cured Tobacco.** *Tobacco Research* 3(2): 95-101, 1977.

The influence of continuous application of plant nutrients in a multiple cropping system on the quality of flue-cured tobacco did not show any significant differences in the chemical composition of tobacco leaf, dry matter production (paddy and/or tobacco), or uptake of nutrients by tobacco for different fertilizer dosages. However, the seasonal variations significantly influenced the chemical composition and nutrient uptake of tobacco. (Auth. Abs. Mod.)

80-0004. Ishizaki, H.; Akiya, T. **Effects of Chlorine on Growth and Quality of Tobacco.** *Japan Agricultural Research Quarterly* 12(1): 1-6, January 1978.

With an increase of chlorine (Cl) application (0-20 kg/10 acres), the amount of Cl absorbed by leaf and stem increased from 6.04 to 14.59 kg/10 acres; but the utilization rate decreased from 92.2 to 42.7 percent at 5 and 20 kg/10 acres respectively. The Cl leaf content increased at all leaf positions, and at lower leaves in particular, with increased Cl application. The contents of total carbohydrates, cations, sodium, and phosphate at the middle (20-21) leaf positions increased with increased Cl content. The rate of a lighted cigarette dying out under natural burning conditions was determined by chlorine and potassium content, and by leaf position. Chlorine and potassium were antagonistically related to burning quality. The higher the leaf position, the lower the chlorine content, and the greater dying out rate of lighted cigarettes.

80-0005. Long, R. C.; Woltz, W. G. **Environmental Factors Affecting the Chemical Composition of Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 116-163.

Environmental factors such as air and soil temperature, solar radiation, humidity, carbon dioxide concentration, nutrition, and soil moisture; and their effects on tobacco plant development and chemical composition of tobacco leaf are discussed in this review.

80-0006. Markose, V. T.; Patel, G. J. **Effect of Soil Fumigation and Nitrogen Manuring on Bidi Tobacco.** *Tobacco Research* 3(1): 7-10, June 1977.

The effect of fumigation and nitrogenous fertilizer on the yield and quality of bidi tobacco was studied using ten treatment combinations of five nitrogen (N) levels with 25 liters/hectare 1-2-Dibromo—3—chloropropane (DBCP) and without. Application of DBCP gave about 43.72 percent higher yield and thereby an increased profit of 1086 Rupees/hectare over no fumigation. The beneficial effect of fumigation was due to better plant growth resulting from control of root knot nematodes (*Meloidogyne javanica and*

Meloidogyne incognita). Nitrogenous manuring per application up to 300 kg N/hectare was advantageous. Nonsignificant interaction of DBCP and nitrogenous fertilizer revealed that the fertilizer efficiency was not influenced by nematicide. (Auth. Abs.)

80-0007. Moore, L. D.; Reilly, J. J.; Terrill, T. R. **The Effect of Soil Fertility on Weather Fleck, Growth, and Chemical Composition of Flue-Cured Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 269-289.

The effects of nitrogen (N), phosphorus (P), and potassium (K) fertilization rates on the sensitivity of flue-cured tobacco to ambient levels of air pollution, and the effects of fertilization and fleck damage on growth and nitrogen, nicotine, and sugar composition of cured leaves were determined. Field experiments were conducted with NC88, which is sensitive to air pollution, and with either Coker 319, or 347, as the tolerant flue-cured cultivar. Oxidant levels increased as the growing season progressed, but leaf fleck did not increase at the same rate. No simple linear relationship was found between pollutant levels and flecking intensity. Significant relationships were found for applied N-P-K fertilization rates and both plant growth and leaf composition. N-P-K rates were also significantly related to air pollution injury ratings with higher rates of N and P associated with lower injury ratings. No consistent associations were found between the degree of flecking and growth or chemical composition. High rates of P fertilization resulted in high percentages of reducing sugars, and were associated with lower fleck ratings. High rates of N fertilization also resulted in lower injury rates and sugar percentages in cured leaves.

80-0008. Peedin, G. F.; McCants, C. B. **Influence of Soil Applications of Calcium on Selected Agronomic and Chemical Characteristics of Flue-Cured Tobacco.** *Tobacco Science* 21: 17-21, 1977.

Six field experiments, designed to study the influence of soil applications of calcium (Ca) on selected agronomic and chemical characteristics of two flue-cured tobacco (*Nicotiana tabacum L.*) cultivars, were conducted on soils and in environments representative of those where the crop is grown in North Carolina. In each experiment, rates of banded fertilizer Ca (as $CaSO_4$) from 0 to 150 lbs/acre(A) were applied to cultivars NC 2326 and Coker 254. At one location, the cultivars and fertilizer Ca treatments were superimposed on broadcast applications of 0 and 1000 lbs/A dolomitic limestone for two successive years. Yield and value per acre were increased both years by lime application to a Norfolk fine sandy loam with 40 percent aluminum saturation and pH 5.3. In the remaining experiments, a significant yield response to fertilizer Ca was obtained only on a soil with 0.40 meq exchangeable Ca/100 cc soil. The average price of cured leaves was not adversely affected by lime application, whereas the 150 lbs/A rate of fertilizer Ca decreased average price in two experiments; inferior leaf quality as measured by average price was not associated with excessively high leaf concentrations of Ca and magnesium (Mg). Cured leaves from limed plots contained higher concentrations of Ca, Mg, and nitrogenous constituents, and generally lower concentrations of potassium (K) and reducing sugars than those from nonlimed plots, but the balance of total alkaloids to nitrogen was not substantially influenced by lime application. Concentrations of Mg, nitrogenous constituents, and sugars in cured leaves were not consistently affected by rate of fertilizer Ca, while concentrations of Ca and K generally were increased by rates up to 75 lbs/A. Significant cultivar—node position interactions were obtained for each leaf constituent measured, whereas interactions of cultivars with lime and/or fertilizer Ca were rarely significant for either agronomic or chemical characteristics. (Auth. Abs.)

80-0009. Reddy, N. S. **Accent on Ripeness—A Basic Requirement for Production of Quality Tobacco.** *Tobacco Research* 2(1): 1-6, June 1976.

Ripe tobacco is characterized by such physical characteristics as graininess, fluffiness, high filling value, elasticity, hygroscopic properties, color, and aging. Chemically, ripe tobaccos are characterized by favorable sugar/nicotine, nitrogen/nicotine ratios and low levels of soluble nitrogen. Harvesting time, varieties grown, curing techniques, cultural practices, temperature, moisture, pests, and disease are also important.

80-0010. Shoop, G. J.; Rogers, W. E.; Lynn, L. B.; Jones, M. L. **Oryzalin, A Pretransplant Surface Applied Herbicide for Burley and Dark Tobacco.** In: Proceedings of the 31st Meeting of the North Central Weed Conference, Omaha, December 7-9, 1976. North Central Weed Control Conference, Inc., Champaign, Illinois, 1976, pp. 93-95.

Oryzalin was evaluated as a pretransplant surface application in burley and dark tobacco research trials conducted in Maryland, Kentucky, and Wisconsin. Weed control ratings, transplant survival counts, crop injury ratings, rate of flowering as an indication of crop maturity, yield, and quality of tobacco were data collected from the various experiments. Normal cultural practices including cultivation were utilized throughout the growing season. The tobacco types Wis 503, MD 872, burley, and dark, all showed excellent tolerance to the X rate of oryzalin for soil types. There was no significant difference observed in stand survival. No detectable crop injury symptoms were observed. The Wisconsin trial indicated crop maturity was delayed in the untreated plots due to weed competition. The green weight and cured weight yield of all research trials indicated that these tobacco types had excellent tolerance to oryzalin. In the Maryland location, three U.S. Department of Agriculture research specialists graded the quality of tobacco from all chemical treatments slightly above the untreated control.

CHEMISTRY

80-0011. Abedinzadeh, Z.; Razeghi, M.; Khalkhali, Z. **Determination of Trace Elements in Cigarette Paper by Neutron Activation Analysis.** *Radiochemical and Radioanalytical Letters* 27(1): 35-42, September 14, 1976.

The concentration of 21 trace elements in cigarette wrapping paper used for manufacturing Iranian cigarettes was measured by neutron activation analysis employing a high resolution Ge(Li) detector. γ-ray spectra of the samples were taken by a 4096- and an 800-channel pulse height analyzer. The trace elements found were sodium, potassium, scandium, chromium, manganese, iron, cobalt, zinc, selenium, bromine, strontium, rubidium, silver, cesium, tin, antimony, lanthanum, europium, gadolinium, terbium, mercury, and thorium. The presence of elements such as mercury, antimony, and selenium is of particular interest in view of their suspected toxic properties.

80-0012. Andersen, R. A.; Kasperbauer, M. J.; Lowe, R. H.; Smiley, J. H. **Comparison of Carbonyls and Nitrogenous Compounds During Bulk Versus Air Curing of Burley Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 184-216.

Total carbonyls, free amino acids, total protein, and alkaloid concentrations were compared in bulk-cured and conventionally air-cured burley tobacco. A method for measuring total carbonyls in tobacco was developed to facilitate this study. Significant concentration differences were found for total carbonyls, amino acids, proteins, and alkaloids in lamina and midveins of leaves cured by the two methods. The differences were discussed in terms of their effects upon the quality and usability of the cured tobacco. An introductory review of the chemical changes that occur during the air curing of tobacco was also presented. (Auth. Abs.)

80-0013. Ball, K. **Carbon Monoxide Yield of Cigarettes. (Letter).** *British Medical Journal* 2(6192): 731, September 22, 1979.

While filter cigarettes with their lower tar levels may have contributed to a fall in lung cancer rates in men under age 60, they may also have led to more deaths from coronary heart disease due to their higher carbon monoxide (CO) levels (if the filters are not ventilated). Knowledge of the CO yield of individual cigarette brands is therefore very important. The British Government is urged to publish this information (which is already routinely collected by the Department of Health and Social Security), in addition to the tar and nicotine levels of cigarettes.

80-0014. Baretta, E. D.; Stewart, R. D.; Graff, S. A.; Donahoo, K. K. **Methods Developed for the Mass Sampling Analysis of CO and Carboxyhemoglobin in Man.** *American Industrial Hygiene Association Journal* 39: 202-209, March 1978.

Gas chromatography was used to quantitate carbon monoxide (CO) in air and also as an indirect means of determining the percentage of carboxyhemoglobin (COHb) in blood. The blood was then used to calibrate four CO-Oximeters used in a survey to determine average COHb levels in various segments of the U.S. population. Mean differences, both between the two methods of analysis and between pairs of CO-Oximeters, were less than 0.1 percent COHb saturation. COHb values obtained on consecutive days using one CO-Oximeter were repeatable within a S.D. ±0.13 percent COHb. Carbon monoxide is normally found in the atmosphere and in the alveolar breath of nonsmokers at concentrations ranging from 1-10 ppm. Unless exposed to elevated ambient levels, the nonsmoker normally has a COHb saturation of <2 percent. Continuous exposure to CO at the present ambient air standard of 10 mg/m^3 (8.7 ppm) would result in a COHb level of about 1.5 percent. CO concentrations resulting from the analysis of blood samples collected from nonsmokers were well within the analytical capabilities of this method. A typical series of 10 repetitive determinations resulted in a COHb saturation of 1.2 percent S.D. 0.10 (coefficient of variation = 8.3 percent) with a range of 1.08-1.35 percent COHb. (Auth. Abs. Mod.)

80-0015. Brunnemann, K. D.; Stahnke, G.; Hoffmann, D. **Chemical Studies on Tobacco Smoke. LXI. Volatile Pyridines: Quantitative Analysis in Mainstream and Sidestream Smoke of Cigarettes and Cigars.** *Analytical Letters* 11(7): 545-560, 1978.

A quantitative analytical method has been developed for volatile pyridines in tobacco smoke using pyridine-^{14}C as an internal standard. Mainstream and sidestream smoke are trapped in 0.05N sulfuric acid and partitioned with chloroform. The acidic portion is then concentrated, adjusted to pH 9, and extracted with chloroform. Methanol and a trace of 2N sulfuric acid are added to this chloroform portion and the mixture is carefully concentrated. A trace of sodium hydroxide is added and the final concentrate is analyzed by gas chromatography. Twenty-one volatile pyridines have been identified. The mainstream smoke of a popular 85 mm U.S. cigarette without filter tip contained the following major pyridines: 32.4 μg pyridine, 12.3 μg α-picoline, 24.2 μg $\beta+\gamma$-picoline, 7.6 μg 3-ethylpyridine, 5.9 μg 4-ethylpyridine, and 23.3 μg 3,4-lutidine + 3-vinylpyridine. The sidestream smoke of this cigarette contained up to 28 times higher concentrations of pyridines. Volatile pyridines were also determined in the mainstream smoke of other nonfilter cigarettes, filter cigarettes, little cigars, and a nontobacco cigarette. Of special interest were the much greater quantities of volatile pyridines in the mainstream and sidestream of cigars in comparison to those of cigarettes. The ratio of pyridines in sidestream to those in mainstream was 5 to 50 in large cigars. Selective filtration was observed with both cellulose acetate and charcoal filter tips. The analytical method resulted in reproducible values for pyridine, with an average recovery rate of more than 90 percent. In addition to pyridines, two pyrazines were also isolated from the smoke. Processed tobacco contained minute amounts of some volatile pyridines. (Auth. Abs.)

80-0016. Court, W. A.; Hendel, J. G. **Determination of Nonvolatile Organic and Fatty Acids in Flue-Cured Tobacco by Gas-Liquid Chromatography.** *Journal of Chromatographic Science* 16(7): 314-317, July 1978.

A method is described for the analysis of the major nonvolatile organic acids and fatty acids of flue-cured tobacco (*Nicotiana tabacum L.*). The tobacco acids are simultaneously extracted from tobacco and esterified with sulfuric acid in methanol. The methyl esters are then partitioned into chloroform and determined by gas-liquid chromatography. (Auth. Abs.)

80-0017. DeBardeleben, M. Z.; Claflin, W. E.; Gannon, W. F. **Role of Cigarette Physical Characteristics on Smoke Composition.** In: *Recent Advances in Tobacco Science*. Series 4, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1978, pp. 85-111.

The various physical characteristics which describe the cigarette have been examined with regard to their effect on the composition of the smoke generated. The effects of cigarette length, circumference, weight and packing density, tobacco filler cut width, butt length, and filler moisture content have been reviewed. Mathematical modelling is viewed as a partial answer to controlling the complex variables which prescribe the ultimate smoke composition. It is possible to juxtapose the influences of many variables at once and judge their relationships and counterbalancing effects. Assuming that puff count is the parameter to be tested, its dependence on packing density is first determined experimentally with everything closely controlled except for density. An equation is derived to portray this relationship. Then, variances in circumference are studied and differences between the circumference results and the density function are used to derive a second equation. The same procedure is followed for the third variable, butt length. The net result of these three studies is an equation which allows the prediction of effects of changes in up to three variables simultaneously. This predictive capability is a powerful asset in the design of new cigarettes.

80-0018. De Salles de Hys, L. **La Fraction Hydrosoluble du Condensat de Fumee de Cigarette: Identification des Principaux Constituants. [The Water Soluble Fraction of Cigarette Smoke Condensate: Identification of the Principal Components.]** *Annales du Tabac* Section 1(14): 119-124, 1976, French.

The water-soluble fraction of cigarette smoke condensate has been partitioned on ion exchange resin to separate neutral and basic constituents before analysis by gas-chromatography and mass-spectrometry. Nitrogen compounds have been identified, some of them for the first time: cyclic amides, pyridine and pyrazine derivatives, and nicotine n′-oxide. (Auth. Abs.)

80-0019. Durocher, D. F.; Mattina, C. F.; Selke, W. A. **Diffusion of Gaseous Components Through the Wrapper of a Cigarette.** *Beitrage zur Tabakforschung International* 9(4): 201-207, July 1978.

The diffusion of carbon monoxide (CO) and carbon dioxide (CO_2) through the wrapper of cigarettes was studied by direct measurement under steady state conditions, simulating the conditions which obtain during puffing. Cigarettes made with porous paper, with perforated paper, and with ventilated filters were studied. The diffusion of CO and CO_2 from cigarettes was found to be strongly influenced by the mode of cigarette ventilation. The greatest effect of diffusion on CO delivery was found for cigarettes where ventilation was achieved by ventilating the filter. The influence on diffusion of the type and amount of burn chemical applied to the paper and of changing the average pressure drop across the cigarette is described. It was found that normal levels of burn chemicals added to the paper will only slightly decrease the fraction of CO diffusing through the wrapper. (Auth. Abs.)

80-0020. Einolf, W. N.; Ferguson, R. N.; Whidby, J. F.; DeBardeleben, J. F. **Isolation and Identification of 2,6-Dimethyl- and 2,2,6-Trimethyl-4-piperidone, Artifacts Produced by the Reaction of Cigarette Smoke Condensate and Acetone.** *Beitrage zur Tabakforschung International* 9(4): 208-213, July 1978.

Cigarette smoke condensate (CSC) processed with acetone was found to contain 2,2,6-trimethyl-4-piperidone and the *cis* and *trans* isomers of 2,6-dimethyl-4-piperidone. The compounds were found to be artifacts formed by the reaction of ammonia and acetaldehyde in the CSC with the solvent acetone. Conditions which affected the yield of the artifacts from the processed CSC include pH, temperature, reaction time, and levels of ammonia and acetaldehyde present in CSC. (Auth. Abs.)

80-0021. Gopalachari, N. C.; Kameswara Rao, B. V. **A Semi-Micro Method for the Determination of Total Nitrogen in Tobacco.** *Tobacco Research* 2(2): 163-165, 1976.

A simple, rapid, and reliable modification of the conventional Kjeldahl method for the determination of total nitrogen in plant materials is described. Tobacco samples of different varieties and types were analyzed for total nitrogen by the usual Kjeldhal method and the new method. The mean difference between the two methods was tested by means of students t test for paired observations. The observed mean difference was not significantly different from zero; hence, it may be concluded that the values of total nitrogen estimated by the two methods are not significantly different from each other at the 5 percent level. The method may therefore be adopted for the determination of total nitrogen in plant samples, as it is simple, efficient, and time-saving.

80-0022. Hecht, S. S.; Schmeltz, I.; Hoffmann, D. **Nitrogenous Compounds in Cigarette Smoke and Their Possible Precursors.** In: *Recent Advances in Tobacco Science*. Series 3, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1977, pp. 59-93.

In this review, the modes of formation of some major classes of nitrogenous compounds in tobacco smoke are discussed. Certain groups of compounds such as pyridines, indoles, nitriles, nitro compounds, aromatic amines, and volatile nitrosamines appear to be present in smoke principally as a result of complex pyrolytic reactions during smoking. Others, such as the tobacco alkaloids (e.g., nicotine and nornicotine), amino acids, and aliphatic amines are transferred from tobacco. Some groups, including pyrroles, pyrazines, nonvolatile nitrosamines (e.g., N′-nitrosonornicotine), and hydrazines may arise as a result of both transfer and pyrolytic reactions. Hydrogen cyanide and ammonia are two other important smoke components. Major leaf precursors for nitrogenous products in smoke include nitrates, proteins, amino acids, tobacco alkaloids, and pigment. The manipulation of these factors to produce smoke with desirable characteristics is a complex task. For example, reduction of tobacco nitrate would appear to be a feasible method for reducing nitrosamine and ammonia levels in smoke. However, high nitrate tobaccos give smoke with reduced levels of polynuclear aromatic hydrocarbons. Some components, such as pyrroles and pyrazines, may be added to tobacco to improve flavor, but their possible effects on levels of undesirable smoke components must be determined. (Auth. Abs.)

80-0023. Higgins, C. E.; Gayle, T. M.; Stokely, J. R. **Sensor for Detection of Tobacco Smoke Particulates in Inhalation Exposure Systems.** *Beitrage zur Tabakforschung International* 9(4): 185-189, July 1978.

A commercially available light-emitting diode/phototransistor array was investigated for detection and analysis of tobacco smoke in the Walton Horizontal Smoking Machine (WHSM) animal inhalation exposure system. The diode element of the device emits light into the smoke aerosol, and light back-scattered by the smoke particles is detected by the phototransistor element. The sensor provides a continuous, nearly instantaneous graphical visualization of smoke particulates in the immediate vicinity of the array. Experimental studies showed that sensor readout is linearly related to smoke particulate concentration of the smoke and is independent of the composition of the smoke. Smoke particle agglomeration associated with aging of smoke causes observable but minor changes in sensor response. (Auth. Abs.)

80-0024. Hoffmann, D.; Hecht, S. S.; Ornaf, R. M.; Wynder, E. L.; Tso, T. C. **Chemical Studies on Tobacco Smoke. XLII. Nitrosonornicotine: Presence in Tobacco, Formation and Carcinogenicity.** In: Walker, E. A.; Bogovskii, P.; Griciute, L. (Editors). *Environmental N-Nitroso Compounds Analysis and Formation*, IARC Science Publications No. 14, Lyon, 1976, pp. 307-320.

Nitrosonornicotine (NNN), the first organic carcinogen isolated from unburned tobacco, has been found in smoking tobaccos, chewing tobaccos, and snuff in concentrations between 0.3 and 90 μg. This appears to be an unusually high concentration for a nitrosamine in an environmental agent. Data are presented which suggest that NNN, and possibly other unknown nitrosamines, are formed during the curing of tobacco and that the nitrate content of tobacco is an important factor in nitrosamine formation. In mice, NNN induced adenomas of the lung. Bioassays with rats have shown that NNN is carcinogenic to the esophagus and the nasal cavity. These chemical and biological data are consistent with the observation that tobacco chewers face an increased risk of cancer of the esophagus. (Auth. Abs. Mod.)

80-0025. Hseih, R. H. **Differences in Chemical Components Between Taiwan Tobacco and Imported American Tobacco.** *Bulletin of the Taiwan Tobacco Research Institute* 6: 39-44, April 1977, Chinese.

In order to study the difference in chemical composition between Taiwan and United States tobacco varieties, samples of comparable leaf grades were taken for analysis of their major chemical components. The following results were obtained: (1) Taiwan tobacco was higher in total alkaloids than United States tobacco; (2) crude ash was higher in Taiwan tobacco than United States tobacco, especially for C group leaves; (3) Taiwan tobacco showed slightly higher content of reducing sugar than United States tobacco; (4) United States tobacco was shown to contain twice the amount of petroleum ether extracts as Taiwan tobacco; and (5) approximately the same level of total nitrogen and chlorine content was observed in the two tobacco varieties. (Auth. Abs.)

80-0026. Ishiguro, S.; Sugawara, S. **Gas Chromatographic Analysis of Cigarette Smoke by Trimethylsilylation Method.** *Beitrage zur Tabakforschung International* 9(4): 218-221, July 1978.

A gas chromatographic method was developed to quantitatively determine the major components in the total particulate matter (TPM) of cigarette smoke. Tobacco smoke condensate was collected on a glass fiber filter and the components were converted to their trimethylsilyl derivatives and then subjected to glass capillary column gas chromatography. By this method, volatile and nonvolatile tobacco smoke components, including unstable phenolic substances and water-soluble polyhydroxy compounds, were determined simultaneously. (Auth. Abs.)

80-0027. Ishiguro, S.; Sugawara, S. **Comparisons of Smoke Components in the Semivolatile Phase From Lamina and Midrib Cigarettes of Flue-Cured Tobacco Leaves.** *Agricultural and Biological Chemistry* 42(8): 1527-1531, August 1978.

Dichloromethane extracts of lamina and midrib cigarette smoke condensates were subjected to glass capillary column gas chromatography. Most peaks in the gas chromatogram were identified and the major components were quantitatively determined. The compositional differences between the two types of smoke were then compared. Among the major components (excluding nicotine) in lamina cigarette smoke were the isoprenoids, furans, phenols, acetic acid, pyridines, acetol, and 2,3-dihydro-3,5-dihydroxy-6-methyl-4H-pyran-4-one. In midrib cigarette smoke, 2-cylopentenones, furfural, 5-methylfurfural, phenols, acetic acid, and 2,3-dihydro-3,5-dihydroxy-6-methyl-4H-pyran-4-one were the major constituents. In midrib cigarette smoke the major components appear to be derived from cell wall substances by pyrolysis while lamina cigarette smoke consists of many sugar degradation products and essential oil components which would be directly transferred from tobacco. (Auth. Abs. Mod.)

80-0028. Jenkins, R. W., Jr.; Bass, R. T.; Newman, R. H.; Chavis, M. K. **Cigarette Smoke Formation Studies. V. The Effects of the Cigarette Periphery on Mainstream Smoke Formation.** *Beitrage zur Tabakforschung* 9(3): 126-130, October 1977.

The effect of the cigarette periphery on smoke formation has been studied. A series of cigarettes was prepared with carbon-14 labeled materials placed in different radial positions within the rods. These cigarettes were smoked and the distribution of the radioactive label in total smoke was measured. The periphery played a large contributing role (though not the sole role) in the formation of the (1) mainstream particulate phase, (2) sidestream particulate phase (derived from nondistillable/nonsublimable materials), and (3) mainstream gas phase (derived from nondistillable/nonsublimable materials). The central portion of the cigarette rod played a large contributing role in (1) sidestream gas phase and (2) sidestream particulate phase (derived from distillable/sublimable compounds). The mainstream gas phase yields from the distillable/sublimable compounds were due in large part to the pyrolysis/combustion products formed during the static burn period. These products were carried into mainstream smoke by the incoming air at the beginning of the pull and by diffusion due to the pressure gradient existing in the cigarette during static burn. These experiments have demonstrated the significant effect that the central portion of the cigarette rod has on sidestream smoke formation and, conversely, the effect of the periphery on mainstream smoke. (Auth. Abs. Mod.)

80-0029. Kiefer, J. E. **Ventilated Filters and Their Effect on Smoke Composition.** In: *Recent Advances in Tobacco Science* , Series 4, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1978, pp.69-83.

The rapid increase in sales of ventilated-filter cigarettes has been paralleled by studies directed toward defining the effects of ventilation on smoke chemistry. Filter ventilation not only dilutes the smoke stream, but it also effects physical changes at the burning coal, within the tobacco column, and within the filter. Ventilation of a filter cigarette alters the pyrolytic conditions and changes the amount and composition of the smoke. The compressed temperature gradient behind the burning coal may also influence aerosol formation as well as the rate of transfer of volatile components (such as nicotine) from the tobacco to the smoke aerosol. The slower flow rate through the cigarette column enables significant amounts of low molecular weight gases, such as carbon monoxide, to diffuse out of the cigarette while the lower flow rate through the filter facilitates increased filtration efficiencies. Consequently, the amounts of most smoke components are reduced except for oxygen and nitrogen which are increased, and the chemical composition of the smoke is altered. Perhaps the most interesting effects are reduced total particulate matter, high filtration efficiencies, and considerably lower amounts of carbon monoxide and hydrogen cyanide deliveries. The composition of the semivolatile fraction is also modified. (Auth. Abs.)

80-0030. Murty, K.S.N.; Gopalachari, N. C.; Subramanyam, M.; Ramachander, P. R.; Sastry, A. S. **Inter Relationship of Physical and Chemical Attributes in FCV Tobacco.** *Tobacco Research* 2(1): 45-51, June 1976.

By working out correlations between physical and chemical quality characters of tobacco, it was observed that sugars and the quality ratios of total sugars/total nitrogen, total sugars/nicotine and total sugars/protein are positively correlated to equilibrium moisture content and negatively correlated to filling value, pore volume, and thickness. The reverse was the case with the nitrogenous constituents. As an extension of these studies, attempts were made to build up prediction equations of chemical quality characters based on physical quality characters and vice versa. The results showed that prediction of physical characters using chemical characters was not satisfactory, while the chemical characters could be predicted with good precision using physical characters. Among the physical characters, filling value appeared to be more related to these chemical characters. (Auth. Abs.)

80–0031. Nagaraj, G.; Chakraborty, M. K. **Lipophilic Constituents of *NATU* Tobacco: I. N-Paraffinic Hydrocarbons.** *Tobacco Research* 3(1): 18-22, June 1977.

N-Paraffins in *Natu* tobacco have been isolated and analyzed by gas-liquid chromatography. The dry leaf contains 0.17 percent paraffinic hydrocarbons, hentriacontane being the major component accounting for 55.6 percent of the total. Odd numbered n-paraffins constitute 91 percent of the total paraffins. Dotriacontane is the major even numbered compound representing 7.6 percent of the total paraffins. (Auth. Abs.)

80–0032. Neurath, G. B.; Dunger, M.; Pein, F. G. **Interaction of Nitrogen Oxides, Oxygen and Amines in Gaseous Mixtures.** In: Walker, E. A.; Bogovskii, P.; Griciute, L. (Editors). *Environmental N-Nitroso Compounds Analysis and Formation.* IARC Scientific Publications No. 14, Lyon, International Agency for Research on Cancer, 1976, pp. 215-225.

The rate of decrease in nitric oxide content in aging cigarette smoke, calculated to contain 10 percent oxygen, is of the same order but slightly higher ($k = 3.24 \times 10^6$) than that produced with secondary amines, the higher value probably being due to an oxygen content greater than 10 percent. Thus, the formation of nitrosamines in tobacco smoke under normal conditions is shown to be possible. However, results obtained with various trapping and extraction systems should be verified by calculations of their kinetics to estimate the probability of their occurring under natural conditions. Aging of tobacco smoke, particularly in the presence of air, will unavoidably enhance the formation of nitrogen dioxide and, as a consequence, of *N*-nitroso compounds. The marked effect of oxygen concentration and the negative effects of temperature and of increasing water content on the oxidation of nitric oxide indicate important sources of error in studies on the nitrosamines present in tobacco smoke. Smoke actually drawn in by a smoker immediately enters the respiratory tract, where the temperature is about 37°C and the relative humidity about 100 percent. (Auth. Abs.)

80–0033. Owens, W. F., Jr. **Effect of Cigarette Paper on Smoke Yield and Composition.** In: *Recent Advances in Tobacco Science* , Series 4, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1978, pp.3-23.

With increasing permeability, inherently porous papers reduce puff counts and, with the exception of low molecular weight vapor phase compounds, give the expected nonselective reductions in tar, nicotine, carbon monoxide, and nitric oxide. Increasingly porous perforated papers increase puff count, selectively increase nicotine, and give lower vapor phase deliveries along with reductions in tar, carbon monoxide, and nitric oxide. Thermal analytical techniques do not fully explain the nature of burning chemical effect on puffing, permeability, and smoke composition. Work with high levels of additives can be used to design per puff yields without increasing per cigarette carbon monoxide deliveries. How paper permeability, inherent and perforated, is related to paper structure is reviewed. (Auth. Abs.)

80–0034. Perinelli, M. A.; Carugno, N. **Determination of Trace Metals in Cigarette Smoke by Flameless Atomic Absorption Spectrometry.** *Beitrage zur Tabakforschung International* 9(4): 214-217, July 1978.

A flameless atomic absorption method has been developed which permits the quick determination of the levels of trace metals in cigarette smoke. The total particulate matter was collected by electrostatic precipitation and dissolved in methanol before analysis. In order to trap the last remaining traces of metals, the gas phase was passed through microporous filters which were analyzed directly. The sensitivity of this method for the metals studied (zinc, lead, cadmium, nickel) is good in both the particulate and gas phase samples, with the sole exception of nickel in the gas phase. Results obtained by the flame and flameless methods are compared. (Auth. Abs.)

80–0035. Rogers, J. D.; Mitchem, A. R. **Physical Properties of Leaf as Indicators of Chemical and Smoking Properties.** In: *Recent Advances in Tobacco Science.* Series 2, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1976, pp. 112-126.

Leaf buyers for tobacco companies make sensory evaluations of blends for mouth and throat impact, and for characteristic fullness, balance, and amplitude of flavor. Other evaluations of the smoking properties of tobacco are based on physical and chemical properties. The reverse is also true. Color, pH, harvest time, and leaf position are considered, as are nitrogenous compound, nicotine, and reducing sugar contents. Body, or smoking strength, is expressed by high total nitrogen and nicotine levels. It is positively correlated with α-amino nitrogen and negatively correlated with sugars. The percentage of these components is used to evaluate not only smoking characteristics but also the effect of agricultural practices such as fertilization on tobacco quality.

80-0036. Sabir, S. S.; Barbooti, M. M. **Determination of Some Trace Metals in Iraqi Tobacco Leaves by Atomic Absorption Spectrophotometry.** *Annales du Tabac* 13(2): 181-185, 1976.

Atomic absorption spectrophotometry has been used for the determination of nine elements: sodium (Na), potassium (K), magnesium (Mg), calcium (Ca), iron (Fe), manganese (Mn), nickel (Ni), copper (Cu), and zinc (Zn) in Iraqi tobacco leaves grown in four districts representing plain and mountainous regions. In general, tobaccos grown in mountainous regions exhibit a lower percentage of Na, K, Mg, and Ca than those grown in the plain. Compared with other tobaccos grown in the U.S.A., Iraqi tobaccos contain a much higher content of these elements. While Fe is high and Mn is low in these tobaccos, the sum of the two is comparable to those of other tobaccos. With regard to Cu, the content of Iraqi tobacco is more than 20-fold higher than American tobacco. This is attributed to a correspondingly high copper content in the soil. (Auth. Abs. Mod.)

80-0037. Schlotzhauer, W. S.; Walters, D. B., Snook, M. E.; Higman, H. C. **Characterization of Catechols, Resorcinols, and Hydroquinones in an Acidic Fraction of Cigarette Smoke Condensate.** *Journal of Agricultural and Food Chemistry* 26(6): 1277-1281, 1978.

A biologically active, catechol-containing subfraction of a weakly acidic fraction of cigarette smoke condensate was further separated by gel filtration chromatography. Individual constituents of the gel fractions were identified by gas chromatography-mass spectrometry and ultraviolet spectroscopy. The principal dihydroxybenzene components were catechol, resorcinol, hydroquinone, and their alkyl-substituted derivatives. Other components of this fraction were 4-pyrone, vanillins, dimethoxyphenols, phenylphenols, and naphthols. (Auth. Abs.)

80-0038. Snook, M. E.; Severson, R. F.; Arrendale, R. F.; Higman, H.C.; Chortyk, O. T. **Multi-Alkylated Polynuclear Aromatic Hydrocarbons of Tobacco Smoke: Separation and Identification.** *Beitrage zur Tabakforschung International* 9(4): 222-247, July 1978.

The methyl, multi-methyl, and ethyl derivatives of the polynuclear aromatic hydrocarbons (PAH) of cigarette smoke condensate (CSC) were isolated from the neutrals by silicic acid chromatography, solvent partitioning, and gel chromatography. The procedure yielded a relatively pure PAH isolate amenable to further identifications. The multi-alkylated PAH were concentrated in the early gel fractions with parent and higher ring PAH found in subsequent gel fractions. It was shown that CSC is very rich in alkylated PAH, and their successful identification required extensive use of gas and liquid chromatography and ultraviolet and gas chromatography (GC)-mass spectrometric techniques. High-pressure liquid chromatography separated individual isomers of the alkylated PAH in complex gas chromatographic peaks. PAH from indene to pentamethyl chrysene were found. Collectively, approximately 1,000 PAHs of cigarette smoke condensate have been identified and have led to the development of methods for the routine quantitation of PAH in small quantities of cigarette smoke condensate. (Auth. Abs.)

PHARMACOLOGY AND TOXICOLOGY

80-0039. Autrup, H.; Essigmann, J. M.; Croy, R. G.; Trump, B. F.; Wogan, G. N.; Harris, C. C. **Metabolism of Aflatoxin B_1 and Identification of the Major Aflatoxin B_1-DNA Adducts Formed in Cultured Human Bronchus and Colon.** *Cancer Research* 39(3): 694-698, March 1979.

Aflatoxin B_1 and benzo(*a*)pyrene were activated by both cultured human bronchus and human colon as measured by binding to cellular DNA and protein. The binding of aflatoxin B_1 to DNA was dose-dependent, and the level of binding was higher in cultured human bronchus than it was in the colon. When compared to aflatoxin B_1, the binding level of benzo(*a*)pyrene to both bronchial and colonic DNA was generally higher. (Auth. Abs. Mod.)

80-0040. Chance, W. T.; Kallman, M. D.; Rosecrans, J. A.; Spencer, R. M. **A Comparison of Nicotine and Structurally Related Compounds as Discriminative Stimuli.** *British Journal of Pharmacology* 63(4): 609-616, August 1978.

Of seven nicotine-like compounds tested as discriminative stimuli in the rat, only 3-pyridyl-methylpyrollidine (3-PMP) generalized to the stimulus effects of nicotine. At a dose (800 μg/kg) approximately 4 times that used for the original nicotine discrimination (200 μg/kg), 3-PMP caused an equivalent nicotine-like response. The ED_{50} for 3-PMP was about 5 times that for nicotine. Testing of the compounds as possible antagonists of the nicotine-elicited cue were negative. The nicotine-like cue produced by an 800 μg/kg injection of 3-PMP was effectively blocked by mecamylamine but not by hexamethonium or atropine. Thus, 3-PMP appears to produce generalization to the nicotine cue via action on central nicotinic-cholinoceptors as has been previously reported for the nicotine discriminative stimulus. Mecamylamine blocked the stimulus-effects of 3-PMP (800 $\mu g/kg$) and of nicotine (200 $\mu g/kg$) with an ED_{50} of 0.32 and 0.20 mg/kg respectively. (Auth. Abs.)

80-0041. Drath, D. B.; Karnovsky, M. L.; Huber, G. L. **The Effects of Experimental Exposure to Tobacco Smoke on the Oxidative Metabolism of Alveolar Macrophages.** *Journal of the Reticuloendothelial Society* 25(6): 597-604, June 1979.

In response to 30 days of experimental exposure to tobacco smoke, rat alveolar macrophages (AM) showed a doubling of oxygen consumption (QO_2) and hydrogen peroxide (H_2O_2) release during phagocytosis, while $NADP^+$-dependent glucose oxidation was depressed and superoxide release was unaffected. The glutathione cycle, thought to link peroxide-generating reactions with hexose monophosphate shunt activity, was unaffected by tobacco smoke. Glutathione peroxidase was present to the extent of 27.2±5.2 units/mg protein and 24.3±4.6 units/mg in AM of control and smoke-exposed rats, respectively. The corresponding levels for glutathione reductase were 1.8±0.32 units/mg protein and 1.5±0.29 units/mg. A peroxidase similar to the myeloperoxidase of neutrophils was not detectable in AM. Catalase activity, although not affected by tobacco smoke, was present to the extent of 3.0±0.8 units/mg protein and was found in both a cytoplasmic and granule-rich component, with the latter latent to the extent of an order of magnitude. Superoxide dismutase was affected by smoke exposure in that a 35 percent decrease in activity was demonstrable. (Auth. Abs.)

80-0042. Drath, D. B.; Karnovsky, M. L.; Huber, G. L. **Tobacco Smoke: Effects on Pulmonary Host Defense.** *Inflammation* 3(3): 281-288, 1979.

Tobacco smoke affected both the metabolism and function of pulmonary alveolar macrophages (PAM). Phagocytosis of viable *Staphylococcus aureus* and inert starch particles was minimally but consistently depressed in PAM from rats exposed to tobacco smoke for six months. Oxygen consumption, superoxide and hydrogen peroxide release, and hexose monophosphate shunt activity were elevated in cells from smokers. Oxidation of glucose, labeled in the carbon-six position, remained unchanged. All observed effects of tobacco smoke on oxygen metabolism occurred during phagocytosis and did not affect the basal metabolism of the nonstimulated cell. (Auth. Abs.)

80-0043. Elgerot, A. **Psychological and Physiological Changes During Tobacco-Abstinence in Habitual Smokers.** *Journal of Clinical Psychology* 34(3): 759-764, July 1978.

Effects of 4 days of abstinence from tobacco smoking were examined in 11 habitual smokers at their usual place of work and related to smoking habits and attitudes toward smoking as studied with the aid of a questionnaire. The results showed that adrenaline excretion was consistently lower during the abstinence than during the smoking period. The intra-pair mean difference between the two conditions was statistically significant for each day ($p<0.02$). When the mental tasks were performed, adrenaline excretion was higher than on other days, which is consistent with earlier findings that showed that adrenaline excretion is a sensitive indicator of task-induced stress. Noradrenaline excretion, which showed the same pattern as adrenaline, was consistently lower during abstinence than during smoking. However, the differences between conditions were less marked than for adrenaline and significant only during the third day ($p<.05$). The task-induced arousal was not reflected in the excretion rate of this amine, which is also in agreement with earlier data. Performance on cognitive tests was unaffected. Irritation, disturbances in social relations and activities, and lack of needed social support were shown and reported frequently.

80-0044. Fagerstrom, K.-O.; Gotestam, K. G. **Increase of Muscle Tonus After Tobacco Smoking.** *Addictive Behaviors* 2(4): 203-206, 1977.

Six subjects smoked half a cigarette with different nicotine levels and inter-puff intervals. Electromyograph (EMG) readings were taken before, during, and after smoking from the pars descendens of musculus trapezius. During smoking it was found that the muscle tonus increased in a dose-dependent fashion. Two to three minutes after smoking the EMG level had returned to the initial (presmoking) level. The two conflicting phenomena which follow smoking, i.e., increased autonomic activity and the feeling of relaxation, are discussed. (Auth. Abs.)

80-0045. Frear, D. S.; Swanson, H. R. **Behavior and Fate of (^{14}C)Maleic Hydrazide in Tobacco Plants.** *Journal of Agricultural and Food Chemistry* 26(3): 660-666, 1978.

The behavior and fate of $(4,5\text{-}^{14}C)$ and $(3,6\text{-}^{14}C)$maleic hydrazide (MH) have been studied in selected varieties of flue-cured and burley tobacco. Foliar-absorbed (^{14}C)MH was translocated rapidly to actively growing tissues in a "source-to-sink" pattern. Twenty-eight days after treatment, 30-40 percent of the absorbed (^{14}C)MH was translocated to the roots and released into the nutrient solution, 12-22 percent remained in the plant. Fourteen to eighteen percent was extracted as methanol-soluble metabolites, and 25-35 percent remained in the roots and other tissues as a methanol-insoluble residue. Only 2 percent of the absorbed ^{14}C was evolved as $^{14}CO_2$. Degradation of the MH heterocyclic ring structure was not a significant metabolic pathway. The major methanol-soluble metabolite in foliar tissues was isolated and identified as the β-D-glucoside of MH. Unchanged MH was isolated and identified as a hydrolysis product following acid and base treatment of isolated methanol-insoluble residues. The distribution of ^{14}C in methanol-soluble and methanol-insoluble residue fractions from freshly harvested young tobacco plants was not altered greatly when the tobacco was flue-cured or air-cured under laboratory conditions. (Auth. Abs.)

80-0046. Fung, K. H.; Luke, R.K.J.; Uren, N. C. **Uptake and Translocation of Methomyl by Tobacco Plants.** *Tobacco Science* 21: 131-133, 1977.

The uptake and translocation of methomyl, a pesticide used in Australia to control yellow dwarf disease in tobacco plants, were investigated following application of the pesticide to the substrate and foliage of pot grown tobacco plants. Methomyl was absorbed and rapidly translocated to various parts of the plants when the pesticide was applied to the substrate. Methomyl (0.2 ppm) was detected in the lower part of the plant 5 minutes after treatment, and 0.8 ppm was found in the growing tip 3 hours after treatment. The highest level of methomyl was detected 8 days after application. Methomyl was also absorbed by the foliage, but translocation of the pesticide was small when compared with the amount of methomyl retained by the treated leaves. The highest level of methomyl translocated was 12.7 μg, whereas that retained was 130 μg. (Auth. Abs.)

80-0047. Green, M. R.; Lin, J. S.; Berman, L. B.; Osman, M. M.; Cerreta, J. M.; Mandl, I.; Turino, G. M. **Elastolytic Activity of Alveolar Macrophages in Normal Dogs and Human Subjects.** *Journal of Laboratory and Clinical Medicine* 94(4): 549-562, October 1979.

The presence and amount of elastase activity in alveolar macrophages from human and animal lungs remain uncertain. In this study levels of elastase activity were determined in freshly lavaged alveolar macrophages from 6 normal dogs and 12 human subjects without history of previous lung disease who underwent diagnostic bronchoscopy. Two of the subjects had never smoked. Elastase was assayed against oxalic acid-solubilized ligamentum nuchae elastin and succinyl trialanyl p-nitroanilide. Activity was calculated per unit cell and per unit weight of total protein content of the cell extract. Mean elastase activity extracted from the dog lungs was $6.3 \pm 3.1 \times 10^4$ μU/10^7 cells and 1767 ± 169 μU/μg of protein against the elastin substrate and $1.5 \times 10^5 \pm 6.2 \pm 10^4$ μU/10^7 cells and 4174 ± 373 μU/μg of protein against succinyl trialanyl *p*-nitroanilide. In human subjects mean elastase activity was $1.6 \times 10^5 \pm 3.0 \times 10^4$ μU/10^7 cells and 891 ± 119 μU/μg of protein against solubilized elastin and $2.0 \times 10^5 \pm 3.0 \times 10^4$ μU/10^7 cells and 1411 ± 319 μU/μg of protein against succinyl trialanyl *p*-nitroanilide. No relationship was found between smoking history and elastase activity. Freshly lavaged macrophages were also grown in culture. Measurable and similar levels of elastase were detected in media replaced twice weekly for 3 months. Time-lapse cinematography showed cells undergoing mitosis. Dog and human macrophages in culture phagocytosed particulate dyes and albumin-coated polystyrene microbeads. Synthetic site-specific elastase inhibitors (chloromethylketones) completely suppressed elastase activity. The results demonstrate significant specific elastase activity in alveolar macrophages from normal dogs and human subjects, both smokers and nonsmokers. Elastase activity appears to be synthesized continuously by alveolar macrophages rather than internalized from external sources. (Auth. Abs.)

80-0048. Greer, C. A.; Alpern, H. P. **Maturational Changes Related to Dopamine in the Effects of *d*-Amphetamine, Cocaine, Nicotine, and Strychnine on Seizure Susceptibility.** *Psychopharmacology* 64(3): 255-260, September 1979.

The effects of four neural excitants (*d*-amphetamine, cocaine, nicotine, and strychnine) on myoclonic and clonic seizure susceptibility were investigated in two age groups (30 and 120 days) of short-sleep mice. Amphetamine and cocaine decreased susceptibility to myoclonus in young mice and increased susceptibility in mature mice. These effects were attenuated by pretreatment with haloperidol, indicating mediation by a dopaminergic system. Amphetamine did not alter clonic susceptibility in either age group of mice, whereas cocaine affected clonic susceptibility and myoclonus. These effects were not attenuated by haloperidol, indicating mediation by systems other than dopamine. Nicotine decreased susceptibility to myoclonus and increased susceptibility to clonus, whereas strychnine increased susceptibility to both types of seizure. Haloperidol, however, failed to alter any of these effects. These results are consistent with our previous work which suggests that a dopaminergic mechanism in these mice undergoes marked developmental changes between 30 and 120 days of age. (Auth. Abs.)

80-0049. Hammer, R. E.; Mitchell, J. A. **Nicotine Reduces Embryo Growth, Delays Implantation, and Retards Parturition in Rats.** *Proceedings of the Society for Experimental Biology and Medicine* 162(2): 333-336, November 1979.

During the first 5 days of pregnancy, twice daily injections of nicotine (5mg/kg body weight) reduced embryo growth (43 vs. 6 percent retained blastocyst cavities at 6 AM, Day 5), delayed implantation 9 hours, and retarded the onset of parturition 17.3 ± 2.0 hours vs. 9.8 ± 0.4 hours ($p < 0.001$). Prior to uterine entry, embryos of nicotine-treated rats underwent a 31 percent reduction in cell number. It is not known whether nicotine exerts its growth-suppressing effects by acting directly on the embryo and/or indirectly by modifying the interior of the reproductive tract.

80-0050. Hsu, I. C.; Harris, C. C.; Yamaguchi, M.; Trump, B. F.; Schafer, P. W. **Induction of Ouabain-Resistant Mutation and Sister Chromatid Exchanges in Chinese Hamster Cells With Chemical Carcinogens Mediated by Human Pulmonary Macrophages.** *Journal of Clinical Investigation* 64(5): 1245-1252, November 1979.

Pulmonary macrophages (PAM) from 15 patients with smoking histories metabolically activated benzo(*a*)pyrene (BaP) and its proximate carcinogenic metabolite (±)trans 7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene (7,8-diol), to ultimate mutagens that were detected in cocultivated Chinese hamster V79 cells. Increases in the frequency of ouabain-resistant (O^r) mutations and sister chromatid exchanges were found in V79 cells only when they were cocultivated with both PAM and the chemical procarcinogens. Higher frequencies of both O^r mutations and sister chromatid exchanges were noted with 7,8-diol than with the parent compound, BaP. When metabolically activated by PAM the mean O^r mutation frequency caused by BaP was 9 O^r mutants/10^6 surviving V79 cells per 10^6 PAM and a 10-fold interindividual variation (range, 2-21) was found. The mean O^r mutation frequency caused by 7,8-diol was 64 and a ninefold interindividual variation (range, 14-120) was found. In the absence of PAM, the O^r mutation frequency in V79 cells was one or less O^r mutant per 10^6 survivors. An inhibitor of mixed function oxidases, 7,8-benzoflavone, reduced the frequencies of O^r mutations and of sister chromatid exchanges in V79 cells caused by 7,8-diol and BaP. As expected, 7,8-benzoflavone did not influence the frequency of O^r mutations caused by one of the ultimate mutagens derived from BaP and 7,8-diol, (±)7β, 8α-dihydroxy-9α, 10α-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene. These data are consistent with the hypothesis that PAM may play a role in the activation of environmental chemical procarcinogens. (Auth. Abs.)

80-0051. Hulshoff, A.; Lubawy, W. C.; Kostenbauder, H. B. **The Effect of Some Cigarette Smoke Constituents and Other Compounds on the Metabolism of Benzo(*a*)pyrene in Rabbit Lung 9000 *g* Supernatant.** *Xenobiotica* 8(11): 711-717, 1978.

The inhibitory effects of a number of compounds on the formation of two groups of benzo(*a*)pyrene metabolites in rabbit lung homogenates (9000 *g* supernatant) have been investigated. The inhibitors comprised two groups: those inhibiting primarily benzo(*a*)pyrene hydroxylase activity, and those inhibiting the activity of the epoxide-metabolizing enzymes as well as hydroxylase activity. Phenol, 1-naphthol, nicotine, and acetone belong to the former group; naphthalene, anthracene, and chlorpromazine to the latter group. The most active inhibitors also caused a significant decrease in the amount of benzo(*a*)pyrene bound covalently to tissue macromolecules. (Auth. Abs.)

80-0052. Husain, M. M.; Dehnen, W.; Monch, W. **Comparative Metabolism of Benzo(*a*)pyrene in Some Rodents.** *Toxicological European Research* 1(2): 75-83, March 1978.

Basal and methylcholanthrene-induced levels of lung microsomal aryl hydrocarbon hydroxylase (AHH) activities in male and female rats, mice, and hamsters were compared using a sensitive fluorimetric assay. Metabolic profiles of lung and liver from these species were simultaneously analysed by high performance liquid chromatography. Hamsters showed a slightly low level of activity compared to rats and mice. No significant difference in the enzyme activities between male and female groups of same species was observed. Methylcholanthrene treatment significantly induced lung enzyme in all the groups tested apparently showing no marked sex differences except in hamsters. Relatively more benzo(*a*)pyrene-9,10-diol and 7,8-diol formed in mouse liver and 4,5-diol and phenols in rat liver. Seemingly no marked difference between lung metabolite profiles of rat and mouse was observed. One marked distinction in lung and liver of rodents was in their relative amounts of 9,10-diols and quinones. Methylcholanthrene treatment resulted in or about a 2-fold increase of relative amounts of diols and reduction of quinones in rat liver. Changes observed in mouse liver were significant only in the case of 4,5-diol, 7,8-diol and quinone. About a 4-fold increase in the relative amounts of 9,10-diols and 2-fold increase of other diols and phenols was observed in lung of rat and mice after methylcholanthrene administration.

80-0053. Janoff, A.; White, R.; Carp, H.; Harel, S.; Dearing, R.; Lee, D. **Lung Injury Induced by Leukocytic Proteases.** *American Journal of Pathology* 97(1): 111-129, October 1979.

Human polymorphonuclear neutrophilic leukocytes (PMNs) contain large amounts of neutral proteases that can degrade elastin, collagen, proteoglycan, and basement membrane. The instillation of one of the purified enzymes (elastase) into dog lungs in vivo causes degradation of elastic fibers and other alveolar septal components and results in anatomic changes similar to those of human pulmonary emphysema. Cigarette smoking is a major risk factor associated with pulmonary emphysema in man. One mechanism for this association may be interference with the regulation of PMN elastase activity by alveolar antiproteases. This possibility is supported by the observation that the oxidizing activity of tobacco smoke inactivates α_1-proteinase inhibitor in vitro. Macrophages also secrete an elastolytic protease, albeit at low levels. The short term exposure of cultured mouse macrophages to cigarette smoke augments the rate of elastase secretion by these cells. Mouse macrophage elastase is not inhibited by α_1-proteinase inhibitor or α_2-macroglobulin. This unusual property of macrophage elastase may facilitate its attack upon elastin over prolonged intervals despite very low levels of macrophage elastase production. A unified hypothesis of lung injury in pulmonary emphysema is presented, involving both PMN and macrophage elastases and the actions of cigarette smoke. (Auth. Abs.)

80-0054. Kaden, D. A.; Hites, R. A.; Thilly, W. G. **Mutagenicity of Soot and Associated Polycyclic Aromatic Hydrocarbons to *Salmonella typhimurium*.** *Cancer Research* 39(10): 4152-4159, October 1979.

The mutagenic activity of the polycyclic aromatic hydrocarbon-containing fraction of several soot samples was measured in *Salmonella typhimurium*, using resistance to the purine analog 8-azaguanine as a genetic marker. A postmitochondrial supernatant derived from livers of phenobarbital- and/or Aroclor-pretreated male Sprague-Dawley rats was incorporated into all assays to allow metabolism of promutagens to their active forms. The mutagenic activity of the soot extracts ranged from 10 to 20 times higher than could be accounted for by the amount of benzo(*a*)pyrene present. The possibility that synergism occurs between benzo(*a*)pyrene and some component in the soot extracts was discounted by the finding of a simple additive relationship of mutagenicity of a soot extract and added benzo(*a*)pyrene. To examine the alternative explanation that other components of soot may have undiscovered mutagenic activity, 70 polycyclic aromatic hydrocarbons were quantitatively assayed for their mutagenic potential; 34 of these compounds induced a significant increase in the mutant fraction resistant to 8-azaguanine. Of particular interest are the extreme mutagenicities of perylene, cyclopenta(*cd*)pyrene, and fluoranthene, all of which exhibit greater mutagenicity than does benzo(*a*)pyrene at equimolar concentrations. Using the measured activities of each polycyclic aromatic hydrocarbon constituent in a kerosene soot, the mutagenic activity of the whole polycyclic aromatic hydrocarbon fraction could be accounted for in terms of the additive mutagenicity of its individual components. (Auth. Abs.)

80-0055. Kozak, P. P., Jr. **Factors That Influence Theophylline Metabolism.** *Western Journal of Medicine* 131(4): 319-320, October 1979.

Many allergists in the United States consider theophylline to be the initial bronchodilator of choice. Serum levels of theophylline of 7 to 20 µg/ml are generally accepted as therapeutic. Although symptoms of toxicity may occur when the level of theophylline in the serum is in the therapeutic range, toxicity is generally associated with levels in excess of 20 µg/ml. Theophylline clearance is also affected by diseases involving the cardiorespiratory systems and the liver and by factors such as weight, age, and diet. It is incumbent on the physician using this valuable but potentially toxic drug to appreciate the number of factors that influence its biotransformation and clearance. In cigarette and marihuana smokers, there is a faster plasma theophylline clearance and a shorter half-life than in nonsmokers. An additive increase in theophylline clearance has been noted in those who smoke both substances.

80-0056. Kozjek, F.; Mrhar, A.; Karba, R.; Bremsak, F.; Kopitar, Z. **Theophylline: Repeated Oral Administration to Nonsmokers and Smokers Simulated by Analog Computer.** *Acta Pharmaceutica Jugoslavica* 28(1): 1-6, January-March 1978.

Simulated plasma concentration profiles show that repeated 210 mg doses of theophylline are insufficient to bring the plasma concentration of the drug into its therapeutic range. A higher dosage level (350 mg) appeared to be more satisfactory. A 12-hour dosing interval may suffice for nonsmokers, but a shorter interval is needed for smokers: computer analysis suggests that an 8-hour interval may be adequate. (Auth. Abs.)

80-0057. Lakowicz, J. R.; Englund, F.; Hidmark, A. **Particle-Enhanced Membrane Uptake of a Polynuclear Aromatic Hydrocarbon: A Possible Role in Cocarcinogenesis.** *Journal of the National Cancer Institute* 61(4): 1155-1159, October 1978.

One possible mechanism by which cocarcinogenesis occurs was investigated. The effect of a particulate, silica, upon the rate of membrane uptake of a polynuclear aromatic hydrocarbon, benz(*a*)anthracene (BA), was studied. The fluorescence emission spectrum and quantum yield of BA, when adsorbed to silica, underwent spectral shifts upon addition of phospholipid vesicles. These spectral changes appeared to result from the transfer of BA from the silica surface into the lipid bilayer. These spectral changes were used to measure the rates of membrane uptake of BA from the silica-adsorbed state and from the crystalline and microcrystalline states of BA. Our studies demonstrated that adsorption of BA to silica resulted in an increased rate of uptake of BA into dipalmitoyl-L-α-phosphatidylcholine vesicles compared to the uptake rate into these vesicles from crystalline states. Such particle-enhanced membrane uptake of chemical carcinogens may be of importance in explaining the enhanced carcinogenicity of the polynuclear aromatic hydrocarbons in the presence of particulate matter. (Auth. Abs.)

80-0058. Matulionis, D. H. **Reaction of Macrophages to Cigarette Smoke. II. Immigration of Macrophages to the Lungs.** *Archives of Environmental Health* 34(5): 298-301, September-October 1979.

The present study identifies the source of the elevated pulmonary macrophage population which results when young adult male mice are exposed to cigarette smoke. Light microscopic and autoradiographic analysis of pulmonary tissue from smoke-exposed animals revealed that pulmonary macrophages (free, attached, and septal or interstitial) divide only rarely. Further, during the progressive increase in the labeled macrophage population in the lungs, the number of silver grains over the nuclei of labeled macrophages did not become significantly diluted. Thus, the markedly elevated macrophage population which results when animals are exposed to cigarette smoke appears to be largely due to immigration of cells from bone marrow rather than in situ division of resident macrophages. (Auth. Abs.)

80-0059. Matulionis, D. H. **Reaction of Macrophages to Cigarette Smoke. I. Recruitment of Pulmonary Macrophages.** *Archives of Environmental Health* 34(5): 293-297, September-October 1979.

Recruitment of pulmonary macrophages was studied in adult male mice exposed to cigarette smoke for 42 to 82 days.

Scintillation counting procedures revealed that cigarette smoke induced an increase in DNA activity in pulmonary tissue. No such induction was noted in the liver or spleen—organs which, like the lungs, are involved in clearance of foreign material. Autoradiographic analysis of lung samples taken at intervals up to 40 days following ^3H-thymidine injection revealed an increase in DNA activity reflecting a marked increase in the number of labeled pulmonary macrophages. At times, more than 50 percent of the total pool of labeled cells was identifiable as macrophages. Therefore, cigarette smoke appears to elicit a recruitment of macrophages specifically to the lungs. (Auth. Abs.)

80-0060. Mehta, R.; Cohen, G. M. **Major Differences in the Extent of Conjugation With Glucuronic Acid and Sulphate in Human Peripheral Lung.** *Biochemical Pharmacology* 28(16): 2479-2484, August 15, 1979.

Short-term organ cultures of human peripheral lung metabolize benzo(*a*)pyrene to water-soluble metabolites. Enzymic hydrolysis of these water-soluble metabolites with arylsulfatase (with saccharic acid 1,4-lactone) but not with β glucuronidase released significant amounts of ethyl acetate-soluble radioactivity, which cochromatographed with metabolites of benzo (*a*)pyrene. Similar studies of human peripheral lung showed that 1-naphthol, a model phenolic substrate, was metabolized extensively to its sulfate conjugate with little or no glucuronic acid conjugate being formed. In marked contrast to this, with short-term organ culture of rat lung, 1-naphthol is mainly conjugated with UDP-glucuronic acid. Thus, in human but not in rat lung, phenolic substrates such as monohydroxybenzo(*a*)pyrenes or 1-naphthol are metabolized predominantly to their respective sulfate conjugates with little or no glucuronide conjugates being formed. (Auth. Abs.)

80-0061. Oliver, J. S.; Smith, H.; Williams, D. J. **Nicotine in Post-Mortem Tissue.** *Medicine, Science and the Law* 18(1): 52-55, January 1978.

Techniques for the separation of nicotine from other materials present in human post-mortem tissues are described. Nicotine is commonly found in tissues of smokers. Nicotinamide is usually found in relatively large amounts in tissue or blood, but in urine analysis nicotine is usually the only finding. Thin-layer chromatography can be used to identify either nicotine or nicotinamide. Biological materials were extracted and analyzed by ultraviolet spectrometry, thin-layer chromatography, and gas chromatography. Analysis of 10 random post-mortem urine samples from smokers showed nicotine levels as high as 1.5 mg per 100 ml (average of 0.2 mg per 100 ml). The effect of decay on nicotine was investigated by daily analysis of a blood sample containing 0.03 mg nicotine per 100 ml over a period of 30 days. No change in nicotine level was found, and neither preservatives nor refrigeration had any effect. Even after periodic followups (up to 3 years) no significant change in the nicotine concentration was found.

80-0062. Rana, S.V.S.; Gautam, R. K.; Agrawal, V. P. **A Comparative Histochemical Study of Lipids in the Trachea and Lungs of Squirrel, Funambulus pennanti, Exposed to Three Chief Air-Pollutants.** *Gegenbaurs Morphologisches Jahrbuch* 124(4): 619-626, 1978.

A histochemical study of lipids has been made in the trachea and lungs of common ground squirrels exposed to three chief air pollutants: carbon monoxide (CO), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). Significant changes observed in pulmonary lipids have been correlated with altered mechanical properties of the lung and morphology. Supplementary biochemical findings made on total lipids indicate that CO ranks at the top affecting lipid contents of both trachea and lungs followed by SO_2 and NO_2, respectively. Physiological mechanisms of damage have also been analyzed. (Auth. Abs.)

80-0063. Sershen, H.; Lajtha, A. **The Effect of Nicotine on the Metabolism of Brain Proteins.** *Neuropharmacology* 18(10): 763-766, October 1979.

Nicotine is capable of influencing cerebral protein metabolism. In adult mouse and rat brain slices incubated in vitro with nicotine, valine incorporation into protein was stimulated. The effect was specific for L-nicotine; D-nicotine decreased incorporation, and nicotinamide or nicotinic acid had no effect. In animals treated in vivo with nicotine there was a tendency towards increased incorporation, but the increase was not significant. Protein synthesis in the immature brain was affected differently from that in the adult brain; valine incorporation in the newborn was significantly inhibited by nicotine both in vitro and in vivo. The variability of the results in adult brain tissue suggests individual differences in sensitivity to nicotine. The decrease in protein synthesis in the newborn may support the findings of others of a delayed development in newborns from animals treated with nicotine during pregnancy, but the effect may not be long lasting since no effect is found in adult tissue. The opposite effects of nicotine on growing brain, in contrast to adult brain, indicates that control and pharmacological sensitivity of immature brain protein metabolism differs from that of the adult brain. (Auth. Abs.)

80-0064. Stephens, R. M. **Psychophysiological Variables in Cigarette Smoking and Reinforcing Effects of Nicotine.** *Addictive Behaviors* 2(1): 1-7, 1977.

Studies of psychophysiological changes associated with cigarette smoking are reviewed. Considered variables are: heart rate, peripheral circulation, skin temperature, blood pressure, flicker fusion frequency, and multiple measures. The psychophysiological changes are in the direction of increased metabolic activation and appear to be mediated by nicotine. Smoking behavior may be maintained by reinforcing effects of general autonomic activation following smoking. The importance of identification of those who smoke for physiological vs. social factors is discussed in relation to the development of effective programs to treat smoking behavior. (Auth. Abs.)

80-0065. Terzaghi, M.; Nettesheim, P. **Dynamics of Neoplastic Development in Carcinogen-Exposed Tracheal Mucosa.** *Cancer Research* 39(10): 4003-4010, October 1979.

The qualitative and quantitative cellular changes occurring in tracheal epithelium in vivo during the process of carcinogenesis were investigated by use of an in vitro assay which measures the formation of epithelial foci (EF); this method is called EF assay. The cell or cells giving rise in vitro to an EF which survives and proliferates for at least 1 month after tracheal cells have been seeded into culture dishes was defined as the epithelial focus-forming unit (EFFU). Viable epithelial cells were harvested from heterotopic tracheal grafts immediately and at 2, 4, and 8 months after they had been exposed in vivo for 4 weeks to 165 μg of dimethylbenz(*a*)anthracene. This carcinogen exposure was known to induce a 9 percent incidence of invasive tracheal carcinomas. The cell suspensions obtained from individual tracheas (20 tracheas per time point) were seeded into culture dishes, and the number of EF was scored 1 month later. Normal tracheal cells and cells from tracheas exposed to 100 μg of 12-*O*-tetradecanoylphorbol-13-acetate produced no EF. In contrast, 80 to 90 percent of the cultures of all tracheas exposed to dimethylbenz(*a*)anthracene 8 months prior to the collection of cells, developed one or more EF. During the first 4 months after termination of dimethylbenz(*a*)anthracene exposure, the number of EF per trachea remained constant at 3 to 4. Between 4 and 8 months, the number of EF per trachea increased 3- to 5-fold. Three different types of EF could be distinguished experimentally: EF that could not be subcultured (EF_O); EF that could be subcultured but did not grow in soft agarose ($EF_{s,ag-}$); and EF that could be subcultured and that also grew in soft agarose ($EF_{s,ag+}$). During the 8 months after exposure, EF that can be subcultured (EF_s) increased from 53 to 84 percent, and $EF_{s,ag+}$ increased from 23 to 57 percent. Simultaneously, the frequency of EF_O dropped from 47 to 16 percent. The relative frequency of $EF_{s,ag-}$ remained the same with time. Studies with "mixed-population" cell cultures derived from the same tracheas with which the EF assays were carried out showed similar trends, namely, an increase in subculturability and tumorigenicity in cultures established at increasingly longer time intervals after carcinogen exposure. Findings suggest the existence, in the epithelium of carcinogen-exposed tracheas, of EFFU's endowed with different in vitro growth capacities. The data are consistent with a gradual conversion of EFFU's with limited growth capacity to EFFU's with neoplastic growth capacity as a function of time after carcinogen exposure ($EFFU_O$ to $EFFU_{s,ag-}$ to $EFFU_{s,ag+}$). A tumor induction study, in which tracheal transplants were exposed during a 4-week period to 165 μg dimethylbenz(*a*)anthracene, showed an incidence of 9 percent invasive tracheal carcinomas. In comparison, the EF assay demonstrated that at 8 months 80 percent of such tracheas contain cells with neoplastic potential ($EFFU_{s,ag+}$). This suggests that, left in the host animals, only a fraction of these cells actually succeed in establishing a malignant tumor. (Auth. Abs.)

80-0066. Thompson, P. D.; Jeffery, R. W.; Wing, R. R.; Wood, P. D. **Unexpected Decrease in Plasma High Density Lipoprotein Cholesterol With Weight Loss.** *American Journal of Clinical Nutrition* 32(10): 2016-2021, October 1979.

High density lipoprotein (HDL) cholesterol is inversely related to coronary heart disease prevalence. Despite the fact that obese patients have lower plasma HDL cholesterol concentrations, there are few prospective studies on the effect of weight loss on HDL cholesterol. Consequently, plasma lipoprotein levels were measured in 15 obese females before and after a 10-week weight loss program. Mean weight loss was 8.6±3.9 kg ($p<0.001$). Total plasma cholesterol and low density lipoprotein cholesterol did not change significantly. Plasma triglyceride levels decreased ($p<0.05$) as did HDL cholesterol ($p<0.02$). A subgroup of 11 of the subjects had repeat lipid measurements 8 months after the start of treatment. Mean weight loss at this time was 12.8±9.8 kg ($p<0.01$). No subject had returned to her pretreatment weight but mean weight loss was not significantly different from the 10 week value. At 8 months all lipid values, including HDL cholesterol, had returned to their pretreatment value. By multiple regression analysis HDL cholesterol decreased with increasing relative weight but also decreased with increasing rate of weight loss. Smoking, alcohol consumption, and exercise habits of the subjects were determined by questionnaires before and after treatment and at the 8-month followup visit. Statistical analysis of the subjects' habits showed no significant changes. Results suggest that negative caloric balance produces a decrease in HDL cholesterol that in prospective studies may obscure the inverse relationship between HDL cholesterol and indices of obesity. (Auth. Abs. Mod.)

80-0067. Vahakangas, K.; Nevasaari, K.; Pelkonen, O.; Karki, N. T. **Effects of Various *in Vitro* -Inhibitors of Benzo(*a*)pyrene Metabolism in Isolated Rat Lung Perfusion.** *Acta Pharmacologica et Toxicologica* 45(1): 1-8, July 1979.

Various in vitro inhibitors were added with ^{3}H-benzo(*a*) pyrene (BP) into the perfusion fluids in isolated rat lung perfusions to see whether their effects are dependent on the integrity of tissue. ^{3}H-BP and its metabolites were measured by thin-layer chromatography and radiometry from both samples of perfusion medium and homogenates of lung tissue. The total covalent binding to lung tissue was used as a measure of the formation of reactive metabolites. In methylcholanthrene-induced rat lung, the metabolism of BP was inhibited by α-naphthoflavone, an inhibitor of monooxygenase, and less with diethylmaleate, a depletor of glutathione, with salicylamide, an inhibitor of conjugases, and, astonishingly, with D-saccharo-1,4-lactone, an inhibitor of β-glucuronidase. With trichloropropene oxide, which inhibits epoxide hydratase, the metabolism was either decreased or unchanged. Nicotine had no effect on BP metabolism. Nicotine and diethylmaleate increased statistically significantly and α-naphthoflavone and salicylamide decreased the covalent binding of radioactivity to lung tissue. In most cases, the changes in BP metabolism observed during perfusion can be explained on the basis of effects of modifiers on the enzyme systems. (Auth. Abs.)

80–0068. Wattenberg, L. W.; Jerina, D. M.; Lam, L.K.T.; Yagi, H. **Neoplastic Effects of Oral Administration of (±)-*trans*-7,8— Dihydroxy-7,8-Dihydrobenzo(*a*)pyrene and Their Inhibition by Butylated Hydroxyanisole.** *Journal of the National Cancer Institute* 62(4): 1103-1106, April 1979.

The neoplastic effects of administration of benzo(*a*)pyrene (BP) and (±)-*trans*-7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene (BP 7,8-dihydrodiol) by oral intubation to noninbred female Ha:ICR mice have been determined. Under the experimental conditions, BP-induced papillomas of the forestomach. Benzo(*a*)pyrene- 7,8-dihydrodiol also induced papillomas of the forestomach and was more potent than BP. In addition, administration of BP 7,8-dihydrodiol caused a large number of pulmonary adenomas and lymphomas. Butylated hydroxyanisole (BHA) added to the diet at a concentration of 5 mg/g inhibited BP-induced neoplasia of the forestomach. Neoplasia of the forestomach, lungs, and lymphoid tissues, caused by administration of BP 7,8-dihydrodiol, were also inhibited by BHA. These data suggest that the inhibitory effect of BHA on BP carcinogenesis may entail events that occur subsequent to the formation of BP 7,8-dihydrodiol. (Auth. Abs.)

80–0069. Webster, W. S. **Iron Deficiency and Its Role in Cadmium-Induced Fetal Growth Retardation.** *Journal of Nutrition* 109(9): 1640-1645, September 1979.

The aim of this investigation was to establish the relationship of cadmium-induced fetal growth retardation in the mouse to iron deficiency. Pregnant mice were either fed a low iron diet or given 40 ppm cadmium in their drinking water. The effects of these factors on fetal weight and hematological values of the fetuses and dams were established and compared with each other and with appropriate controls. Both treatments caused maternal and fetal anemia, the fetuses being more severely affected. The anemic fetuses were also severely growth retarded. These changes, when caused by the iron deficient diet, could be completely prevented by either parenterally or orally administered iron supplements. When the changes were caused by cadmium in the drinking water, they were only partially prevented by oral supplements. From these results, it was concluded that iron deficiency in pregnancy causes not only anemia, but also fetal growth retardation. Cadmium exposure in pregnancy also causes anemia and hence fetal growth retardation, presumably by blocking intestinal absorption of iron. This work was initially undertaken to investigate the possible relationship between cadmium inhaled during cigarette smoking, and the well-known association between cigarette smoking and fetal growth retardation. The demonstrated role of anemia in fetal growth retardation in the mouse, and the differences between oral and respiratory exposure, complicate any possible relationship. (Auth. Abs. Mod.)

80–0070. Weiser, P. C.; Morrill, C. G.; Dickey, D. W.; Kurt, T. L.; Cropp, G.J.A. **Effects of Low-Level Carbon Monoxide Exposure on the Adaptation of Healthy Young Men to Aerobic Work at an Altitude of 1,610 Meters.** In: Folinsbee, L. J.; Wagner, J. A.; Borgia, J. F.; Drinkwater, B. L.; Gliner, J. A.; Bedi, J. F. (Editors). *Environmental Stress.* New York, Academic Press, 1978, pp. 101-110.

Low-level carbon monoxide (CO) exposure resulting in 5.1 percent carboxyhemoglobin (HbCO) at an altitude of 1,610 m impaired the work performance of nine healthy male volunteers (all of them nonsmokers) to the same extent as that reported for sea level studies. Small but significant submaximal exercise changes in cardiorespiratory function were found during CO exposure. Work heart rate was elevated, and post-exercise left ventricular ejection time did not shorten to the same degree as with filtered air exposure. CO exposure resulted in a lower anaerobic threshold, and a greater minute ventilation at work rates heavier than the anaerobic threshold due to an increased blood lactate level. (Auth. Abs.)

80–0071. Wright, G.; Shephard, R.J. **Blood Carboxyhaemoglobin Levels in Police Officers.** *Canadian Journal of Public Health* 69: 393-398, September-October 1978.

A rebreathing technique has been used to estimate blood carboxyhemoglobin levels (COHb) in 24 police officers (14 smokers, 10 nonsmokers) before, during, and after completion of patrols in downtown Toronto. The major determinant of COHb readings was the smoking habit of the individual. In smoking officers gains of COHb were positively correlated with cigarette consumption ($p<0.01$). The regression equation indicated that if no cigarettes were consumed, COHb would drop by 0.4±1.2 percent in 4 hours, compared with a predicted clearance of 1.5 percent for an unpolluted atmosphere. Initial readings were quite high in nonsmoking officers (average 1.9±0.2 percent); probable contributing factors include commuting substantial distances through heavy morning traffic, and passive exposure to accumulations of cigarette smoke in car pools and in the lunch room. On the average, COHb concentrations in the nonsmokers remained unchanged over the 4 hours to the lunch break, but rose by a further 0.2 percent during the afternoon patrol. The final figure of 2.0 percent implies 4 hours' exposure to <30 ppm carbon monoxide (CO), or 8 hours' exposure to 10-15 ppm. Individuals patrolling polluted areas had increments as large as 3.8 percent (to 5.3 percent COHb). In general, final COHb levels were positively correlated with vehicle patrol hours, but were negatively correlated with foot patrol hours. This reflects the local nature of CO accumulation, with use of the vehicles on major arterial routes and foot patrols on broad sidewalks or back streets. (Auth. Abs.)

80-0072. Yamatake, Y.; Sasagawa, S.; Yanaura, S. **Drug Responses of Canine Trachea, Bronchus and Bronchiole.** *Chemical and Pharmaceutical Bulletin* 26(1): 318-321, January 1978.

Effects of acetylcholine, pilocarpine, histamine, 5-hydroxytryptamine, and nicotine on the isolated canine trachea, bronchus, and bronchiole were investigated. In addition, Schultz-Dale test to *Ascaris suum* antigen on these airway smooth muscle preparations was attempted. Acetylcholine had a strong contractile effect on the trachea, bronchus, and bronchiole, whereas large concentrations of histamine and nicotine were necessary to contract these preparations. Histamine was suggested to produce a stronger constriction of the peripheral airways like bronchioles. Pilocarpine showed a selective and remarkable contraction of bronchus. Schultz-Dale phenomenon to *Ascaris* antigen failed to be demonstrated on the airway preparations. (Auth. Abs.)

See also, **80-0014, 80-0024**

MORTALITY AND MORBIDITY

80-0073. Armstrong, B. K.; McNulty, J. C.; Levitt, L. J.; Williams, K. A.; Hobbs, M.S.T. **Mortality in Gold and Coal Miners in Western Australia With Special Reference to Lung Cancer.** *British Journal of Industrial Medicine* 36(3): 199-205, August 1979.

Cohorts of 1,974 gold miners and 213 coal miners in Western Australia surveyed for respiratory symptoms, smoking habits, occupational history, and radiographic evidence of pneumoconiosis have been followed up for 13 to 14 years. Overall, neither group had a significantly higher mortality than expected from the experience of Western Australian men in general. Lung cancer mortality was relatively high in the gold miners (59 deaths observed, 40.8 expected) but weakly and inconclusively related to the extent of their underground mining experience. Cigarette smoking may explain the excess of lung cancer in the gold miners because the prevalence of the habit in the latter (66.3 percent) was higher than in the coal miners (58.7 percent) or in other men in Western Australia (53.2 percent); 15.7 percent of the gold miners compared to 20.7 percent of the coal miners had never smoked. Radiographic evidence of silicosis was present in 21.7 percent of the gold miners but did not appear to have contributed substantially to their mortality. The coal miners showed a lower than expected rate of lung cancer but an excess of deaths from all other forms of cancer (11 observed, 5.6 expected). This excess was not attributable to any one cancer site and cannot be explained readily. (Auth. Abs.)

80-0074. Dales, L. G.; Friedman, G. D.; Collen, M. F. **Evaluating Periodic Multiphasic Health Checkups: A Controlled Trial.** *Journal of Chronic Diseases* 32(5): 385-404, 1979.

In 1964, a controlled trial was initiated to evaluate the effectiveness of a program which urged periodic multiphasic health checkups to prevent or postpone morbidity, disability, and mortality. Over 5,000 Kaiser Foundation Health Plan members, aged 35-54 at study entry, were urged to take annual multiphasic health checkups. Over a period of 11 years they were compared with a control group of members who did not have the checkups. No differences were found between the overall groups in self-reported disabilities. But self-reported disabilities were significantly less common in the older study group men (aged 45-54 at entry) due to less disability from hypertension complications, ischemic heart disease, and back conditions. Although the study and control groups were similar in age and sex distributions, it was possible that differences in baseline health status might account for some of the differences in disability outcome. The clinical records of all subjects were reviewed for the period between 1962-1964 to determine the initial prevalence of a wide variety of conditions. Twenty risk factors were derived which had a potential for confounding the 1973 survey results. The prevalence of some risk factor conditions and characteristics were very low (e.g., 17.3 and 17.8 percent of the study-control groups, respectively, were known to smoke cigarettes). Mortality from conditions hypothesized to be most postponable or preventable through periodic medical health checkups was significantly lower in the study group. This was due to fewer deaths from hypertension complications and colorectal cancer. There was more identification and treatment of hypertension, hyperlipidemia, cigarette smoking, obesity, and diabetes in the study group, but the differences were not large. When the economic impact of all major health-related events was estimated for the older men, there was a net difference of $2100 per man in favor of the study group.

80-0075. Eyer, J. **Does Unemployment Cause the Death Rate Peak in Each Business Cycle? A Multifactor Model of Death Rate Change.** *International Journal of Health Services* 7(4): 625-662, 1977.

A combination of natural time series and prospective studies was evaluated to determine the contribution of selected causal factors to variations in the death rate during business cycles. The most probable quantitative estimate of the contribution of unemployment to business cycle variations in the death rate was found to be somewhere between 1 and 5 percent. The contribution of the following factors was also considered: nutrition, medical care, housing, alcohol consumption, and tobacco consumption. These factors were found to account for 24.4 percent of the business cycle variation in the death rate, with an additional 3.4 percent due to medicine, and 16.5 percent due to nutrition. Although many analyses have implicated smoking as a fundamental cause of excess mortality of urban dwellers compared with rural dwellers, and of blue collar workers compared with higher classes, the contribution of the variation of smoking to the business cycle variation in the death rate is smaller, because (1) the variation of tobacco consumption with the business cycle is only slightly significant (2 percent); and (2) not all of the excess mortality among smokers compared to nonsmokers can be considered as a short-term effect, developing over times as short as 3-4 years. Tobacco smoke carcinogens, over long periods of time, contribute to lung cancer, and the carbon monoxide in tobacco smoke accelerates the atherosclerotic process. Over shorter periods, but still longer than those of the business cycle, smoking produces a chronic irritation of the upper respiratory mucuous membranes, contributing to colds, chronic bronchitis, and eventually emphysema. Several large prospective studies showed that the total excess mortality ratio of smokers to nonsmokers is about 1.6. More males than females were found to be heavy alcohol and tobacco consumers. Different causes of death account for the overall variation in mortality for each age and sex. Social relationship changes may account for the greatest part of the death rate variation during the business cycle. When the causal relations examined in this paper were summarized (data for 1949-1975), 97 percent of the variation in the death rate through the business cycle is accounted for. Social relationships directly account for the greatest part of the variation, but social stress indirectly produces a further variation by increased consumption of alcohol and tobacco.

80-0076. Guberan, E. **Surprising Decline of Cardiovascular Mortality in Switzerland: 1951-1976.** *Journal of Epidemiology and Community Health* 33(2): 114-120, June 1979.

In Switzerland, the age-standardized death rate for all diseases of the circulatory system decreased by 22 percent in males, and by 43 percent in females, between 1951 and 1976, which represented a third and nearly a half respectively of the decrease in all deaths. Mortality was reduced by 13 percent in males and by 40 percent in females for nonrheumatic heart disease and hypertension, and by 36 percent and 47 percent, respectively, for cerebrovascular disease. These reductions were on the whole greater than those observed in the 13 other developed countries studied. From 1951 to 1976, consumption of animal fats per capita increased by 20 percent in Switzerland, in spite of a 46 percent reduction in milk intake. The literature on smoking prevalence in Switzerland during this same period (1951-1976) is reviewed. The annual sales of cigarettes per population aged 15 years and over rose dramatically from 1,580 in 1951 to 3,370 in 1973. Data from the only study of smoking habits based on a representative sample of the population show comparatively small differences in males between the age groups, except for the youngest age group (15-24) where the highest proportion of those who never smoked (55 percent) was observed. Young women smoked much more than older ones, and the proportion of those who never smoked was higher in the oldest age group (76 percent) than in the youngest (39 percent). Therefore, as in other countries of Western Europe, it is likely that smoking increased during the last two decades among women and remained unchanged or decreased slightly among men. During the last decade, a threefold rise in the sales of antihypertensive drugs was observed. Oral contraceptives seem to have been widely used by Swiss women since the 1960s. There were also increases in the proportion of women in professional occupations, and in urbanization. The advent of the economic recession was associated with a sudden rise in mortality from nonrheumatic heart disease and hypertension in 1974-76. (Auth. Abs. Mod.)

80-0077. Swann, P. G. **Smoking or Health—A Report of the Royal College of Physicians. Review of the Publication.** *Medical Bulletin* 38(1): 40-65, Spring 1978.

The Royal College of Physicians' Third Report on Smoking, entitled *Smoking or Health,* two sections of which are reprinted in this review, draws attention to the association of smoking with illness, and the shortening of life. From prospective studies of premature death rates in smokers, it is possible to estimate the number of years of life a smoker is likely to lose according to the number of cigarettes smoked per day, and to estimate the decrease in life expectancy for each cigarette smoked. A long-term British study showed that at age 35 and over more than twice as many heavy smokers as nonsmokers (40 percent vs. 15 percent) died before age 65; the smoker shortens his lifespan by about 5 1/2 minutes per cigarette smoked. Other prospective studies agree with the British study that this extra risk of death in smokers, compared with nonsmokers, declines after cessation. A comparison of total death rates in British physicians with those of all men between 1954 and 1971 clearly shows that smoking cessation improves the smoker's life expectancy. Compared with nonsmokers, the cigarette smoker's life is, on the average, not only shorter but also less healthy. Although some of this ill health is due to other habits often associated with heavy smoking (i.e., heavy drinking), a review of the evidence indicates that most of the harm is the direct consequence of smoking. Cigarette smokers are more susceptible than nonsmokers to acute bronchitis and other chest illnesses, lose more time from work and make greater use of medical services than nonsmokers. Although there is a strong statistical association between smoking and coronary disease, it has been suggested that this association is not one of cause and effect, but is due to an inherited predisposition, both to heart disease and to smoking cigarettes. However, it is argued that the association between smoking and heart disease is largely one of cause and effect, because of the strength and consistency of the association, its independence of other risk factors, its enhancement in smokers who inhale, the progressive lessening of risks in smokers who stop, and the known injurious effects of nicotine and carbon monoxide on the cardiovascular system in animals. Coronary heart disease is responsible for about one-half of the total excess deaths among cigarette smokers which are greater in number than excess deaths from either lung cancer or chronic bronchitis.

See also, **80–0078, 80–0083, 80–0119, 80–0122**

NEOPLASTIC DISEASES

80–0078. American Cancer Society. **1980 Cancer Facts and Figures.** American Cancer Society, New York, 1979, 31 pp.

General information concerning survival rates, mortality rates, risk factors, warning signals, and trends in diagnosis and treatment of cancer is given. Smoking is reported as being more dangerous than was supposed in 1964 when the first Surgeon General's Report linking smoking and lung cancer was published. Smoking is responsible for approximately 83 percent of male lung cancer cases and for 43 percent of female lung cancer cases, and is implicated in cancer of the mouth, pharynx, larynx, esophagus, pancreas, and bladder. However, the number of smokers is declining. From 1964 to 1975, the proportion of adult smokers dropped from 52 to 39 percent for men, and from 34 to 29 percent for women. Teenage smoking declined 25 percent between 1974 and 1979. Per capita cigarette consumption went from 4,345 in 1963, to 3,965 in 1978, the lowest in 20 years. Approximately 54 million Americans are now smoking more than 615 billion cigarettes each year. Some 30 million Americans are ex-smokers. Among smoking hazards, maternal smoking is linked with fetal growth retardation and increased mortality risk either prior to or shortly after birth. Further, children of smoking mothers may have deficiencies in physical, intellectual, and/or emotional development. Also, cigarette smoking and exposure to toxic substances or to radioactivity combine to make industrial workers especially susceptible to lung diseases. Tobacco companies have manufactured lower tar and nicotine cigarettes. Average tar contents fell from 36 mg in 1954, to 23 mg in 1964, to 17 mg in 1977. Nicotine levels declined from 2 mg to 1.3 mg to 1.1 mg. More than 40 percent of today's cigarette brands and 35 percent of all sales are in the low tar and nicotine category. Other cancers (e.g., breast, uterine, colon-rectum, skin, oral, leukemia) are also discussed. Public and professional education programs and service and rehabilitation programs are described.

80–0079. Auerbach, O.; Saccomanno, G.; Kuschner, M.; Brown, R. D.; Garfinkel, L. **Histologic Findings in the Tracheobronchial Tree of Uranium Miners and Non-Miners With Lung Cancer.** *Cancer* 42(2): 483-489, August 1978.

The remaining tissue of the tracheobronchial tree from 210 men who died from lung cancer was studied to compare the histologic alterations leading to further sites of primary cancer. These men were uranium miners matched with nonminers for age and smoking habits. A total of 105 matched pairs was selected. Up to the onset of their fatal illness, 3 pairs (2.9 percent) smoked $<1/2$ pack per day, 17 pairs (16.2 percent) smoked between 1/2 and 1 pack per day, 70 pairs (66.7 percent) smoked between 1 and 2 packs per day, and 15 pairs (14.3 percent) smoked 2 or more packs per day. In the examination of a total of 28,928 cross-sections, carcinoma in situ was found in 96 percent of the miners and in 92 percent of the nonminers. The number of slides from miners showing degree 2 or 3 atypia in areas of carcinoma in situ was about double the number found from the nonminers. Although the difference was not statistically significant, 32 percent of the miners had at least one section showing early primary invasive carcinoma compared with 22 percent of the nonminers. The data indicate that the synergistic effect of the exposure to uranium dust along with cigarette smoking increases the risk of lung cancer, and in addition to a main tumor mass, other sites of tissue alternations leading to tumor development are frequently already present in the lung. (Auth. Abs. Mod.)

80–0080. Burch, P.R.J. **Smoking and Lung Cancer: The Problem of Inferring Cause.** *Journal of the Royal Statistical Society, Series A* 141(Part 4): 437-477, 1978.

Methodological problems that arise in epidemiology are briefly reviewed. Associations between smoking and lung cancer, and their connection with sex and country, are described. Secular trends in recorded mortality from lung cancer in England and Wales, 1901 to 1970, are derived from the Registrar General's statistics. Estimates are made of the expected trends assuming that the associations found between the various types of smoking and lung cancer reflect causation. Observed and expected trends conflict. The secular trends in mortality from lung cancer in British male doctors are also examined. From the foregoing and other types of evidence, it is argued that no definitive conclusions can yet be reached as to the extent of any causal link between smoking and lung cancer. It is very doubtful, however, whether the entire association observed between smoking and lung cancer in occidental males should be interpreted in causal terms. (Auth. Abs.)

80-0081. Chovil, A. C. **Occupational Lung Cancer and Smoking: A Review in the Light of Current Theories of Carcinogenesis.** *Canadian Medical Association Journal* 121(5): 548-555, September 8, 1979.

This paper considers modern theories of carcinogenesis as they apply to the induction of lung cancer by tobacco smoking and occupational exposure to carcinogens. Some of the known and postulated factors affecting carcinogenesis are discussed, with particular reference to cocarcinogenesis and thresholds. Factors affecting the intensity of smoking exposure are reviewed, and the generally accepted occupational lung carcinogens are listed. Relative risks for the various carcinogens according to smoking status (where known) are presented. The carcinogens are considered individually, and known or postulated interactions with smoking are discussed. It is concluded that the effects of lung carcinogens can be explained on the basis of current theories that support a rational definition of priorities for the prevention of occupational lung cancer. (Auth. Abs.)

80-0082. Decoufle, P.; Stanislawczyk, K.; Houten, L.; Bross, I.D.J.; Viadana, E. **A Retrospective Survey of Cancer in Relation to Occupation.** U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW Publication No. (NIOSH) 77-178, 1977, 215 pp.

Lifetime occupational histories of 13,949 patients (6,434 white males and 7,515 white females) were analyzed for specific forms of cancer and compared with those of patients diagnosed with non-neoplastic conditions. Relative risk estimates for different cancers within a variety of occupational groups were computed according to classic case—control methodology to indicate the degree of association between an occupation and cancer. The results are presented separately for men and women and refer to persons ever employed in an occupation as well as those employed for five or more years. Risk estimates for certain cancers were adjusted for differences in smoking habits between cancer cases and controls. Only cancer sites which have previously been shown to be strongly associated with smoking have been analyzed, namely: buccal cavity and pharynx, larynx, lung, and bladder. Both past and current smoking habits were taken into consideration in this analysis. Smoking habits were classified differently for men than for women in the analysis. Since there were so few nonsmokers among male patients, they were grouped with smokers of less than one pack of cigarettes per day to form a "light" smoking category. Male smokers of more than one pack of cigarettes per day comprised a "heavy" smoking category. However, definitions of light and heavy smoking differed slightly for each of the four sites studied. Female patients were classified simply as "smokers" or "nonsmokers." Among the many comparisons shown in the report, selected possible high-risk occupations are pointed out as examples of findings which are consistent with prior observations and others that warrant further followup. Among these are bladder cancer in leather workers and dairy farmers, lung cancer in brickmasons and smeltermen, and oral cancer in printing workers. Instances of unusual cancer experience in some occupational groups where the numbers of cases are too small for definitive conclusions are pointed out for future reference. (Auth. Abs. Mod.)

80-0083. Gibbs, G. W.; Horowitz, I. **Lung Cancer Mortality in Aluminum Reduction Plant Workers.** *Journal of Occupational Medicine* 21(5): 347-353, May 1979.

The lung cancer mortality of 5,406 men (cohort 1) employed at one aluminum smelter on January 1, 1950, and of 485 men employed at a second plant (cohort 2) on January 1, 1951, is reported. For each man, the total number of years of exposure to tars, number of years since first exposure to tars, and an index of exposure to tars expressed in tar-years were calculated. More than 99 percent of the men in the first cohort and just under 98 percent of the men in the second cohort were traced. As of December 31, 1973, 1,070 men in cohort 1 had died and death certificates were obtained for 990 men (92.5 pecent). As of December 31, 1973, 64 men in cohort 2 had died and death certificates were obtained for 58 (91 percent). There were 84 deaths from lung cancer in cohort 1 and 11 in cohort 2. The results showed that the mortality from lung cancer of that portion of the cohorts 1 and 2 combined who had ever been exposed to tars was similar to that of workers never exposed to tars. The mortality from lung cancer of men in cohort 1 was greater than that expected at Quebec provincial rates, but this was probably due to slightly increased lung cancer mortality in the communities serving the industries. Although the total number of cases in cohort 2 was small, the lung cancer mortality was well in excess of that expected at Quebec rates and could not be explained by community experience. There was a definite dose—response relationship between lung cancer mortality and tar-years and years of exposure. The standardized mortality ratio for persons exposed for more than 21 years to the higher levels of tars was 2.3 times that of persons not exposed to tars. In this study, information on smoking habits of the workers was not available. Although smoking may still be a factor, the evidence suggests that the increased risk of lung cancer is related to employment in definite tar-exposed occupations. (Auth. Abs.)

80-0084. Henderson, B. E.; Louie, E. **Discussion of Risk Factors for Nasopharyngeal Carcinoma.** In: de-The, G.; Ito, Y. (Editors). *Nasopharyngeal Carcinoma: Etiology and Control.* IARC Scientific Publications No. 20, Lyon, International Agency for Research on Cancer, 1978, pp. 251-260.

Two basic hypotheses have been advanced for the etiology of nasopharyngeal carcinoma (NPC). Although inhalation of smoke (as in certain occupations) may be one explanation for the occasional NPC case seen in industrialized countries, solid evidence to support this mechanism for the risk for NPC in southern Chinese is lacking. Use of cigarettes has generally not been associated with risk for NPC, although excessive cigarette smoking (more than 1 pack/day) carried an increased risk in some studies. The high risk (relative risk = 3.1) for heavy smokers found in one study is not consistent with results of other studies and may be due to methodological bias. Cigarette smoking has not been widespread among rural Chinese until relatively recently. Exposure, by inhalation or ingestion, to *N*-nitrosamines or allied compounds in Cantonese salted fish has gained considerable support as a causative factor from recent epidemiological and experimental studies. Further, careful study of dietary patterns, especially during childhood, of southern Chinese are needed. (Auth. Abs. Mod.)

80-0085. Heyden, S.; Heyden, F.; Heiss, G.; Hames, C. G. **Smoking and Coffee Consumption in Three Groups: Cancer Deaths, Cardiovascular Deaths, and Living Controls. A Prospective Study in Evans County, Georgia.** *Journal of Chronic Diseases* 32(9/10): 673-677, 1979.

Two closely associated habits, cigarette smoking and coffee drinking, were investigated during a 10-year followup in a community-based study. The purpose was to examine the potential relationship between heavy coffee consumption and carcinogenesis. In 1967-1969 the Evans County, Georgia, cohort of 2,530 adults was questioned about its habits, and the mortality followup until 1978 is reported here. During this time 74 patients died of cancer. These patients were matched with 74 patients who died of cardiovascular diseases and 74 presumably healthy survivors. Cigarette smoking was related to mortality from cancer. The 74 cancer patients died an average of 4.4 years after the examinations; 33 (45 percent) were regular cigarette smokers in 1967-1969. In the cardiovascular group there was a slightly smaller number of smokers, 27 (36 percent), while the living group contained only 22 (30 percent) smokers. The difference in smoking habits between the white men of the cancer group, which had more smokers than any other group, and those of the living group was statistically significant ($p<0.05$). Similarly, there was a significant difference in smoking habits between the white males combined with the black males of the cancer group as opposed to the white males and black males in the living group. In contrast, the difference in smoking habits of the cardiovascular group as opposed to the living group is less evident. It should be noted, however, that in the total study population as well as in all race and sex categories, the number of smokers in the cardiovascular group exceeded that of the smokers in the living group. Of the 33 smokers, 15 developed lung cancer and 6 had cancer of the mouth, esophagus, pancreas, and kidney-bladder. Thus, 64 percent of the cancers among the smokers are, at least in part, etiologically related to smoking. No evidence was found to associate heavy coffee drinking with cancer death in this population sample from Evans County, Georgia. While the results do not prove conclusively that heavy coffee consumption is unrelated to carcinogenesis, the data support a cocarcinogenic effect of cigarette smoking in certain organ systems. (Auth. Abs. Mod.)

80-0086. Hinds, M. W.; Thomas, D. B.; O'Reilly, H. P. **Asbestos, Dental X-Rays, Tobacco, and Alcohol in the Epidemiology of Laryngeal Cancer.** *Cancer* 44(3): 1114-1120, September 1979.

A case—control study of 47 laryngeal cancers in males of three counties of Washington State was conducted. Personal interview was used to obtain information on smoking, alcohol use, exposure to asbestos and other substances, and head and neck area X-ray histories. Ex-smokers were combined with current smokers for all analyses. One of the cases and eight of the controls were nonsmokers. Smoking and alcohol consumption were found to increase risk of laryngeal cancer independently, with a clear dose—response relationship. The smoking association was apparent whether one examined the usual level of cigarette consumption or the total pack-years consumption calculated from the year of starting smoking to the year of diagnosis for the case. Neither asbestos exposure nor exposure to other substances was found to significantly increase the risk of laryngeal cancer, although the relative risk with asbestos exposure was 1.75. Lifetime history of exposure to dental X-rays on five or more occasions was associated with significantly increased risk of laryngeal cancer among heavy smokers (more than 20 cigarettes per day) but not among light smokers. When a history of 20 or more dental X-rays was reported, a significant relative risk of 7.5 was found among heavy smokers ($p<.02$). The importance of tobacco and alcohol in the epidemiology of laryngeal cancer was reaffirmed, the importance of asbestos exposure was brought into question, and a possible relationship of laryngeal cancer with exposure to dental X-rays among heavy smokers was demonstrated. (Auth. Abs. Mod.)

80-0087. Hirayama, T. **Descriptive and Analytical Epidemiology of Nasopharyngeal Cancer.** In: de-The, G.; Ito, Y. (Editors). *Nasopharyngeal Carcinoma: Etiology and Control.* IARC Scientific Publications No. 20, Lyon, International Agency for Research on Cancer, 1978, pp. 167-189.

Information concerning the descriptive and analytical epidemiology of nasopharyngeal cancer (NPC) that has been reported mainly since the first international symposium on the subject in Singapore in 1964 is reviewed. NPC is rare in most countries in the world, with an age-adjusted incidence rate of less than 1 per 100,000, and the incidence rate is twice as high in males as in females. Chinese of southern origin have a uniquely high risk, the incidence rates per 100,000 being 10-20 in males and 5-10 in females. The greater the admixture of southern Chinese blood in a given ethnic group, the more likely it is that the NPC incidence rate in that group will be raised. The incidence in both sexes begins to rise after the ages of 20-24 and reaches a plateau at between 45-54. When the logarithm of mortality and morbidity is plotted against the logarithm of the age, the power of the age that provides the best fit to a straight line on a log-log graph is approximately two to four. These figures are lower than for other cancers. Seroepidemiological case-control studies indicate that both different birthplace and abnormal response to Epstein-Barr virus (EBV) antigen significantly enhance the risk for NPC; when these two factors are combined, the relative risk appears to rise further. The effect of other environmental chemicals, such as from cigarette smoking, shown to be significant in several retrospective studies, could explain in part epidemiological phenomena such as sex difference in incidence. In Taiwan, the nasopharyngeal incidence rate at an antibody titer of 640X was 107.0 per 100,000 in heavy smoking (>20 cigarettes) males, 49.9 per 100,000 in light smoking or nonsmoking males, and 32.0 per 100,000 in females. At a titer of 160X, the corresponding incidence rates were 15.3, 5.7, and 2.9. The definitive reason for the uniquely high risk in southern Chinese should be further investigated by taking into account the interactions of host factors such as birthplace and histocompatibility antigen (HLA); and environmental factors such as EBV, chemical carcinogens including nitrosamines, excessive intake of salted fish, and nutritional deficiencies. (Auth. Abs.)

80-0088. Mettlin, C.; Graham, S. **Dietary Risk Factors in Human Bladder Cancer.** *American Journal of Epidemiology* 110(3): 255-263, September 1979.

Retrospective data on dietary habits, employment history, and tobacco use were obtained from 569 bladder cancer patients and 1,025 age-matched controls. Sex-adjusted relative risks revealed increases in risk for lower levels of an index of vitamin A intake. A similar pattern of risk elevation was associated with infrequent milk and carrot intake. The possibility that these relationships were the artifact of an association of dietary habits with other risk factors for bladder cancer (cigarette use, coffee drinking, and high-risk occupations) was considered. Some elevation of risk was found for heavy coffee drinking but the apparent protective effect for milk consumption was not found to be a spurious result of lower coffee intake. Neither was the role of vitamin A explained by its relationsip with smoking or employment in high-risk occupations. Among smokers of more and less than half a pack per day, lower risk was associated with higher ingestion of vitamin A. There were no tendencies for smokers of greater than one-half pack of cigarettes a day to report less frequent consumption of dietary variables. Some association of bladder cancer with infrequent consumption of cruciferous vegetables was also observed. The findings of this investigation are consistent with tumor inhibition by retinoids in animal studies and the low risk associated with vitamin A in epidemiologic studies of lung cancer. (Auth. Abs. Mod.)

80-0089. Mettlin, C.; Graham, S.; Swanson, M. **Vitamin A and Lung Cancer.** *Journal of the National Cancer Institute* 62(6): 1435-1438, June 1979.

Retrospective dietary and smoking data were gathered by interview of 292 white male patients with lung cancer and 801 control patients with nonrespiratory, non-neoplastic diseases. Only eight lung cancer patients had never smoked at all; 176 smoked one pack of cigarettes or less per day, and 108 were considered heavy smokers (more than one pack per day). Since the number of nonsmokers was so small, they were assigned to the group of light smokers (one pack or less a day). A computed index of vitamin A intake was used to differentiate lung cancer patients from controls. Lung cancer patients had lower values than did controls. The reduced relative risk (RR) of lung cancer associated with vitamin A was most evident among men who smoked heavily. For them, a dose-response relationship increasing to an RR of 2.4 for low values of the index was observed. Frequency of daily milk drinking was lower among patients with lung cancer. Lower RR was found among the men who smoked heavily and frequently consumed carrots. These findings are consistent with evidence from animal studies on inhibition of tumor incidence by retinoids and with previous findings in prospective and retrospective epidemiologic studies. (Auth. Abs. Mod.)

80-0090. Vutuc, C.; Kunze, M. **Zur Epidemiologie des Pankreas-Karzinoms: Demographie und sekundar-statistische Analyse moglicher exogener Faktoren. [Epidemiology of Pancreatic Carcinoma: Demography and Secondary Statistical Analysis of Possible Exogenous Factors.]** *Oesterreichische Monatsschrift fur Arztliche Fortbildung* 19(11/12): 286-292, 1978, German.

The pancreatic cancer mortality rate in Austria increased from 6.6 to 11.8 per 100,000 between 1955 and 1975. In 1975, the male mortality rate was higher than that for females in all but the >80 year age bracket. Tobacco consumption and nutritional factors appear to be responsible for this trend. Statistical analyses based on secondary data showed that from 1953 to 1975 cigarette consumption increased from 1,320 to 2,479 cigarettes per adult. There was a significant correlation between cigarette consumption and the increase in pancreatic cancer mortality. Intensified research on exogenous factors and development of preventive measures for limiting the risk of pancreatic cancer are urged.

80-0091. Weaver, A.; Fleming, S. M.; Smith, D. B. **Mouthwash and Oral Cancer: Carcinogen or Coincidence?** *Journal of Oral Surgery* 37(4): 250-253, April 1979.

Two groups, each composed of 100 patients with head and neck cancer were studied. Fifty general surgery patients without head and neck cancer were selected as controls. For analysis, the initial group of patients with head and neck cancer was divided into two subgroups: those with oral or pharyngeal cancer, and those with cancer of the larynx. Patients with oral and pharyngeal cancer (n = 36) consumed significantly greater quantities of alcoholic beverages than the controls ($p < .01$). The same was true of the patients with laryngeal cancer (n = 64), although the level of significance was less ($p < .05$). When the quantity of tobacco consumed was tabulated using a quantity x years index, there was again a significant difference between both subgroups when compared with the controls. There were six patients in the first group, and five patients in the second group who did not use alcohol or tobacco products. Among those patients, a t test showed greater use of mouthwash (quantity x years index) than that of control patients ($p < .001$).

See also, **80-0073, 80-0110**

NON—NEOPLASTIC RESPIRATORY DISEASES

80-0092. Aubry, F.; Gibbs, G. W.; Becklake, M. R. **Air Pollution and Health in Three Urban Communities.** *Archives of Environmental Health* 34(5): 360-368, September-October 1979.

A health study was conducted in three communities in the greater Montreal region; the first two were characterized by relatively high particulate and sulfur dioxide levels, respectively, and the third community had no major industrial pollution. In each community, 300 men and women 45 to 64 years of age were studied. Residents of the two more polluted communities exhibited a higher prevalence of respiratory symptoms and mean lung function was lower than in residents of the less polluted community. Lung function tests included the forced expiratory volume test and the single breath oxygen test. Other variables considered were cough and phlegm (graded 0-5), breathlessness (graded 0-2), and smoking habits (graded 0-2). Lifetime cigarette exposure was expressed as the total number of cigarette packs smoked during life. Lifetime pipe and cigar exposure were expressed in ounces of pipe tobacco smoked during life (5 cigars = 1 ounce pipe tobacco). When intercity differences in age and smoking were accounted for, no statistically significant intercity differences in health status could be shown. (Auth. Abs. Mod.)

80-0093. Baker, M. D.; Irwig, L. M.; Johnston, J. R.; Turner, D. M.; Bezuidenhout, B. N. **Lung Function in Sisal Ropemakers.** *British Journal of Industrial Medicine* 36(3): 216-219, August 1979.

Lung function was measured by spirometry in 66 black male workers in a sisal ropemaking factory, and in their matched controls. Comparison of the sisal-exposed and control groups showed no significant differences ($p > 0.05$) between the means or the standard deviations of the matching variables of age, height, and smoking habit. The major atmospheric contaminant was the lubricant (or a component part thereof) used to soften the fiber. The concentration of airborne matter was generally less than 1 $\mu g\ m^{-3}$. There was no difference in lung function between the two groups before the start of the working shift, that is, the mixture of softening lubricant and sisal caused no long-term effects. Although there was no change in lung function over the working shift in the group making sisal rope, the control group did show a significant improvement in lung function over the same period. This suggests that an effect attributable to the lubricant and sisal dust did exist. To determine whether smoking exerted an effect in conjunction with sisal and the lubricant, the changes in lung function over the work shift of the sisal-exposed versus control groups were analyzed separately, for smokers and nonsmokers. The only significant difference ($p < 0.05$) was that for change in the forced expiratory flow in the middle half of expiration. The mean difference in improvement of lung function between the control and sisal-exposed workers was 80 ml s^{-1} for the smokers and 730 ml s^{-1} for the nonsmokers. In previous studies little mention has been made of the softeners used in the processing of sisal fiber. These additives may exert a significant effect on ventilatory capacity and may act in conjunction with sisal dust. (Auth. Abs. Mod.)

80-0094. Buist, A. S.; Nagy, J. M.; Sexton, G. J. **The Effect of Smoking Cessation on Pulmonary Function: A 30-Month Follow-up of Two Smoking Cessation Clinics.** *American Review of Respiratory Disease* 120(4): 953-957, October 1979.

Eighty persons who attended two smoking cessation clinics were followed up for 30 months in order to evaluate the effects of smoking cessation on pulmonary function. Testing consisted of a respiratory symptom questionnaire, spirometry, and the single-breath N_2 test. Results are presented for 15 persons who managed to stop smoking for the entire 30 months and for 42 who did not stop smoking for any appreciable length of time. Persons who quit smoking showed a significant improvement ($p < 0.05$) in all the respiratory functions tested: vital capacity (VC); forced expiratory volume in one second (FEV_1); mean maximal mid-expiratory flow (MMEF); ratio of closing volume to vital capacity (CV/VC), and of closing capacity to total lung capacity (CC/TLC); and the single-breath N_2 test. This improvement continued for 6 to 8 months and then remained stable. The subjects with the greatest initial impairment showed the greatest improvement. Subjects who continued to smoke showed initial improvement in some function tests, possibly due to a decrease in cigarette consumption, but showed no significant improvement during the whole period. There was no apparent difference between men and women in lung function after smoking cessation.

80-0095. Buist, A. S.; Sexton, G. J.; Azzam, A.M.H.; Adams, B. E. **Pulmonary Function in Heterozygotes for Alpha$_1$-Antitrypsin Deficiency: A Case-Control Study.** *American Review of Respiratory Disease* 120(4): 759-766, October 1979.

The initial cross-sectional data from a prospective study of lung aging in 37 heterozygotes for α_1-antitrypsin deficiency is presented. Using a case-control design, cases included heterozygotes for α_1-antitrypsin deficiency, and protease inhibitor phenotypes MZ and MS selected because they were parents of children identified in a 1971-1974 statewide newborn screening program as having homozygous α_1-antitrypsin deficiency. All of the heterozygotes were less than 40 years of age. Control subjects were selected from a random working population sample participating in a longitudinal study of lung aging. A 2:1 match of control subjects to cases was used, matching age, sex, ethnic origin, and smoking history. A respiratory symptom questionnaire, spirometry, and the single-breath N_2 test, showed no significant differences between heterozygotes and control subjects in terms of respiratory symptoms or pulmonary function. Within each group, however, overall respiratory symptoms were more prevalent among smokers than nonsmokers. Bronchitis and asthma occurred significantly more often ($p<0.05$) in heterozygotes who smoked than in the appropriate control group. Smokers also had lower spirometric variables and higher single-breath N_2 variables. Up to 40 years of age, the heterozygous phenotype Pi MZ and MS was not found to be a risk factor for impairment of lung function. (Auth. Abs. Mod.)

80-0096. Burge, P. S.; Perks, W. H.; O'Brien, I. M.; Burge, A.; Hawkins, R.; Brown, D.; Green, M. **Occupational Asthma in an Electronics Factory: A Case Control Study to Evaluate Aetiological Factors.** *Thorax* 34(3): 300-307, June 1979.

This is the final part of a study carried out to investigate occupational asthma due to sensitivity to colophony fumes (a component of soldering flux) in an electronics factory. Fifty-eight workers with occupational asthma employed on the main shop floor were investigated. The interval between first exposure and sensitization varied widely, with 1 group becoming sensitive within 1 to 2 years of first exposure, and another group whose sensitization was delayed for 3 to 23 years. Once sensitized, the interval between arriving at work and the onset of daily symptoms seemed to be bimodally distributed, resembling the immediate and late asthmatic symptoms seen on provocation testing. Twenty-three out of 58 had no definite daily deterioration at work but improved on weekends. Wheeze and breathlessness occurred in the evenings at home in most, and one-third were woken by breathlessness at least on some nights. These 58 cases were compared with 48 controls without occupational asthma who had worked on the same shop floor for at least 4 years. Overall, 64 percent of the cases were current smokers or ex-smokers, compared with 46 percent of the controls. Mean levels of forced expiratory volume in 1 second (FEV_1) were significantly worse in the cases before exposure on Monday morning. The cases also had more than twice as much sickness absence as controls. FEV_1 fell by more than 10 percent over a working shift in 33 percent of cases and 5 percent of controls. Atopy (a positive skin prick test to one or more common allergens) and a past history of allergic disease were weakly, but significantly associated with being a case. The effects of smoking and a family history of allergic disease were trivial. Prick testing with an antigen derived from the colophony in the solder flux was completely negative, but cases and controls had significantly raised levels of total IgM compared with blood bank controls, perhaps suggesting some previously unrecognized immunological process. (Auth. Abs. Mod.)

80-0097. Demedts, M.; Cosemans, J.; De Roo, M.; Billiet, L.; van de Woestijne, K. P. **Emphysema With Minor Airway Obstruction and Abnormal Tests of Small Airway Disease.** *Respiration* 35(3): 148-157, 1978.

Heavy smoking, prolonged exposure to silicosis, or other occupational hazards were characteristics common in 9 patients complaining primarily of exertional dyspnea. All patients smoked in excess of 10 cigarettes/day. The diagnosis of early emphysema was made on the basis of a decrease of the elastic recoil of the lung and of the single breath diffusing capacity, in the presence of only minor airway obstruction as estimated from routine pulmonary function tests. Closing volume, the alveolar plateau, dynamic compliance and maximal flow at 50 percent of the vital capacity were markedly abnormal; the maximal flow-static recoil pressure relationships suggest, however, that none of the patients suffered from small airway disease. It is concluded that early emphysema should be considered in the differential diagnosis of small airway disease. (Auth. Abs.)

80-0098. Ferris, B. G., Jr.; Speizer, F. E.; Spengler, J. D.; Dockery, D.; Bishop, Y.M.M.; Wolfson, M.; Humble, C. **Effects of Sulfur Oxides and Respirable Particles on Human Health. Methodology and Demography of Populations in Study.** *American Review of Respiratory Disease* 120(4): 767-779, October 1979.

As part of a study of health effects of sulfur dioxide and particulate matter, a cohort of adults 25 to 74 years of age was established in 6 communities, who will be followed prospectively. Historical data for levels of sulfur dioxide and suspended particulate matter in each of the communities were gathered. At the conclusion of the first cycle of measuring the health of adults in 6 sites, it was found that samples were close to the distribution shown in the U.S. Census for age, sex, and occupation, with the possible exception of one city. Analysis of the cross-sectional data indicated that for both age- and height-adjusted values for forced expiratory volume in 1 second (FEV_1) and for selected rates of various respiratory symptoms standardized for age, differences among smoking groups were apparent. An estimate of lifetime cigarette smoking was obtained by asking respondents the number of packs of cigarettes they currently smoked each week and how this had varied in previous years. The lifetime totals of cigarettes smoked were divided into the following 5 categories: "never", <3,000 packs, 3,000 to 8,999 packs, 9,000 to 17,999 packs, and >18,000 packs. For both sexes the effect of these smoking categories was a monotonic decrease in lung function. The decrease was more marked for FEV_1 than for forced vital capacity for each sex, and was more pronounced for men (0.25 l for nonsmokers to -0.45 l for >18,000 pack smokers) than for women (0.06 l for nonsmokers to -0.28 l for >18,000 pack smokers). Differences in these parameters between sites suggest trends that were associated with levels of pollution. (Auth. Abs. Mod.)

80-0099. Fletcher, C. M. **The Prevention of Chronic Bronchitis.** *British Journal of Diseases of the Chest* 73(1): 48-50, January 1979.

Cigarette smoking has been shown to be a major cause of chronic cough and expectoration, and to be heavily implicated in lung function deterioration and bronchitis. The low risk of ex-smokers has shown the value of stopping smoking; however, the only big reduction in the percentage of smokers has been among upper class male professionals. Simple methods of spirometry such as forced expiratory volume in one second or the Wright peak flow meter could be used in conjunction with tests like the expiratory nitrogen slope or the maximum flow rate at 25 percent vital capacity to detect moderate smokers (<10 percent) and heavy smokers (20 percent) who are going to develop disabling airflow obstruction. Perhaps vigorous persuasion to stop or to adapt less hazardous forms of smoking might greatly reduce the prevalence and deadly effects of severe disease.

80-0100. Haack, D. G.; Galdston, M.; Melnick, E. L. **Interactions of Neutrophil Elastase, Serum Trypsin Inhibitory Activity, and Smoking History as Risk Factors for Chronic Obstructive Pulmonary Disease in Patients with MM, MZ, and ZZ Phenotypes for Alpha$_1$-Antitrypsin. (Letters).** *American Review of Respiratory Disease* 117(4): 812-813, April 1978.

A response to an article on the causes of obstructive pulmonary disease (*American Review of Respiratory Disease* 116(5): 837-846, November 1977) considers smoking as an environmental factor effecting chronic obstructive pulmonary disease (COPD) and questions the use of pack-years with control subjects unmatched by age as a relevant indicator of smoke exposure among groups of subjects. The authors of the original article respond that age-matched control and COPD groups were not germane to their study, and that the relationship between pack-years and pulmonary function status was investigated within the two categories of COPD subjects (MM + COPD and MZ + ZZ + COPD phenotypes for alpha$_1$-antitrypsin) not between control and COPD subject groups. See also 78-0480.

80-0101. Hawley, R. J.; Beaman, B. L.; Williams, M. C.; Yeager, H., Jr. **The Ultrastructure of Bronchial Macrophages and Lymphocytes in Sarcoidosis.** *Human Pathology* 10(2): 155-163, March 1979.

A physical interaction between macrophages and lymphocytes was observed more frequently in bronchial lavage fluid obtained from patients with sarcoidosis than from normal volunteers, irrespective of their smoking histories. In this spontaneous interaction, more than two lymphocytes were commonly seen adhering to a macrophage without evidence of cytoplasmic bridging or membrane fusion. To a greater extent than in normal volunteers, macrophages from patients with sarcoidosis were characterized by the appearance of a more highly irregular cell surface, more membrane bound inclusions, and fewer lysosomes and phagolysosomes. The number of surface projections (microvilli) of macrophages from smokers appeared to be reduced. Significant differences were not apparent in the nuclear or cellular diameters of macrophages from sarcoid and normal individuals. (Auth. Abs. Mod.)

80-0102. Higenbottam, T.; Hamilton, D.; Feyerband, C.; Clark, T.J.H. **Bronchial Response to Inhaled Cigarette Smoke in Relation to Respiratory Illness and the Bronchial Threshold to Inhaled Histamine.** *Thorax* 33(5): 679-680, October 1978.

In order to determine what factors were responsible for the wide variation among individual smokers and their acute bronchial responses, 40 individuals were divided, according to smoking habit and symptoms, into 5 groups, and bronchial responses to smoking ordinary low-tar filter cigarettes and comparable tar and nicotine New Smoking Material cigarettes were studied by measuring maximal forced expiratory flow volume curves. Changes in venous blood nicotine and carboxyhemoglobin levels were measured as indicators of the amount of smoke inhaled. The bronchial threshold of each individual to inhaled histamine was also determined. The results indicate that despite heightened bronchial reactivity, smokers tolerate the acute irritant effects of cigarette smoke inhalation that may produce acute airway narrowing, whereas asthmatic and nonsmoking individuals remain sensitive to these effects. The results further suggest that the mechanism of chronic intermittent acute airway narrowing following smoking is not the major mechanism responsible for the development of airflow obstruction in nonasthmatic smokers.

80-0103. Hillerdal, G. **Pleural Plaques in a Health Survey Material: Frequency, Development and Exposure to Asbestos.**

Scandinavian Journal of Respiratory Diseases 59: 257-263, 1978.

All cases of pleural plaques discovered at mass X-ray screening in Uppsala County, Sweden, during 1970-1976 were collected. Case histories, including the history of exposure to asbestos and smoking habits, were taken. A total of 508 cases, 492 males and 16 females, were found. Of 354 interviewed men, 78.9 percent were current smokers or had been smokers at the time of exposure to asbestos. From an investigation made of smoking habits in Sweden, the expected percentage of smokers in these occupations and age groups should be approximately 55. Thus, there were more smokers than expected in the group with pleural plaques. Two-fifths of the "smokers" were ex-smokers, most of them having stopped smoking in the 1970's. The amount and type of tobacco consumed in the pleural plaque group were no different from those expected in a normal smoking population. Exposure to asbestos was confirmed by 80 percent of the population. The frequency of pleural plaques was found to increase steadily with the increase in the use of asbestos which had started 30 years earlier. The pleural plaques showed gradual development and many finally calcified. (Auth. Abs. Mod.)

80–0104. Jackson, H. **Early Chronic Obstructive Pulmonary Disease: A Clinical Diagnosis.** *Journal of the Louisiana State Medical Society* 131(5): 141-145, May 1979.

Chronic obstructive pulmonary disease is a common disorder and a frequent cause of severe dyspnea. Its earliest manifestation is chronic bronchitis, which is diagnosed solely on the basis of sputum production. In general, chronic bronchitis is present for years before significant respiratory symptoms develop. It is more common in men, after the age of 40, and particularly in heavy cigarette smokers (more than 20 cigarettes a day), where it is 4 to 10 times more common than in nonsmokers. When patients with chronic bronchitis become dyspneic, they usually have developed emphysema and the disease is largely irreversible. Both the physical examination and chest X-ray are not helpful in early diagnosis. Simple spirometry can be used to predict those patients who are prone to develop severe airway obstruction. Since cigarette smoking is by far the most important variable in producing chronic obstructive pulmonary disease, reduction of smoking will usually modify the course of the disease. It is suggested that all smokers have simple spirometry yearly.

80–0105. Jedrychowski, W.; Bucholc, B. **Zwiazek Miedzy Poziomem Alfa$_1$-Antytrypsyny w Surowicy Krwi, Paleniem Tytoniu i Przewleklym Niezytem Oskrzeli. [Serum Alpha$_1$-Antitrypsin Levels in Smokers with Chronic Bronchitis.]** *Pneumonologia Polska* 46(7): 505-510, July 1978, Polish.

The purpose of the study was to learn whether symptoms of chronic bronchitis (CB) are related to the serum levels of alpha$_1$-antitrypsin. The study group consisted of 161 industrial workers. The findings showed that over a 5-year period, the probability of persistent CB symptoms in smokers and nonsmokers with alpha$_1$-antitrypsin deficiency was greater, although not statistically significant. In smokers, the difference increased with the mean number of cigarettes smoked daily. The higher level of alpha$_1$-antitrypsin in smokers is probably due to active disintegration of pulmonary macrophages by components of tobacco smoke. (Auth. Abs. Mod.)

80–0106. Kubik, A. **Studies on Smoking, Chronic Nonspecific Respiratory Diseases, and Lung Cancer in Selected Territories of Czechoslovakia.** *Zeitschrift fur Erkrankungen der Atmungsorgane* 152(2): 180-185, 1979.

Results of epidemiological studies in selected territories of Czechoslovakia confirmed the important role of cigarette smoking as a risk factor for chronic bronchitis and lung cancer. Surveys in the Kolin district, Prague, and Zatec revealed that the prevalence of chronic bronchitis for adult male smokers was three to four times higher than that for nonsmokers. The prevalence rate for adult female smokers was two times higher than that for nonsmokers. Chronic bronchitis was more frequent in male cigarette smokers more than 25 years of age in Zatec, an industrial and mining area, than in the corresponding Prague subgroup. The risk of developing lung cancer in male smokers 40-64 years of age who had smoked a total amount of 200,000 cigarettes and over was approximately 50 times higher than for nonsmokers. (Auth. Abs. Mod.)

80-0107. McMillan, G.H.G.; Sheers, G.; Pethybridge, R. J. **A Radiological Follow-up Study of the Effect of Asbestos in Dockyard Workers in Portsmouth.** *Journal of the Royal Naval Medical Service* 65(2): 63-74, Summer 1979.

In 1968 a 10 percent random sample of men variously exposed to asbestos at work in a dockyard in Portsmouth, Great Britain, was examined to determine the prevalence of asbestos-related lesions. Those who responded at that time have been studied after an interval of 9 years to determine attack rates. Among those who had no lesion in 1968 and were reexamined, 10.6 percent had developed some asbestos-related lesion, but only 0.6 percent had pulmonary parenchymal fibrosis, whereas 3.7 percent of those with a pleural lesion at the initial survey were found to have developed parenchymal fibrosis. Dose-response relationships were found in relation to occupations (potential risk of exposure), time in that occupation, and time elapsed since first employment in that occupation. The prevalence of lesions among smokers and ex-smokers was almost double that in nonsmokers. Of 719 men examined, 350 were smokers, 206 ex-smokers, and 163 nonsmokers; the prevalence rates of asbestos-related disorders were 13.1 percent, 17.0 percent, and 8.0 percent, respectively. All those who developed parenchymal fibrosis had a positive smoking history. (Auth. Abs. Mod.)

80–0108. Morgan, W.K.C. **Industrial Bronchitis.** *British Journal of Industrial Medicine* 35(4): 285-291, November 1978.

Epidemiological studies have shown that the prolonged inhalation of dust leads to an increase in prevalence of cough and sputum. New physiological techniques have demonstrated a slight decrement in ventilatory capacity as a result of industrial bronchitis which is related to lifetime dust exposure. Nonetheless, cigarette smoking has about a five to ten times greater effect on ventilatory capacity, as measured by the forced expiratory volume in 1 second (FEV_1), than does dust. Unlike bronchitis induced by cigarette smoke, the predominant effect of industrial bronchitis is on large rather than small airways and the condition is not accompanied by emphysema. While cigarette smoking affects flows at all lung volumes, industrial bronchitis predominantly affects peak flow and flow at high lung volumes. This is especially evident when the flows are expressed as a percentage of total lung capacity (TLC) or as absolute lung volumes. Although industrial bronchitis leads to a moderate increase in the residual volume (RV), thereby suggesting that small airways are not entirely spared, it does not increase the TLC. The evidence, therefore, indicates that in a nonsmoking subject with long continued dust exposure who has cough and sputum plus a minor degree of large airways obstruction, both the symptoms and the pulmonary impairment can be assumed to be a consequence of occupational factors. In contrast, cigarette smoking, whether associated with bronchitis or not, leads to an increase in TLC. The inference to be drawn is that cigarette smoking leads to a loss of retractive forces in the lungs, and that this parenchymal change often occurs before the subject has symptoms. The presence of such structural changes in smokers does much to explain previously reported differences in morbidity and mortality in cigarette- and dust-induced bronchitis. (Auth. Abs. Mod.)

80–0109. Shelley, E.; Dean, G.; Collins, D.; Dinah, F.; Evans, J.; McHardy, J. **Farmer's Lung: A Study in North-West Ireland.** *Irish Medical Journal* 72(6): 261-264, June 30, 1979.

A random sample of farms in three counties in North-West Ireland were visited in an effort to determine the prevalence of farmer's lung. A total of 343 persons (276 men and 67 women), ranging in age from 14 to 86 years, were interviewed. Many of the farm workers had risk factors for respiratory disease: 47 percent were smokers, 18 percent were ex-smokers, a past history of chest illness was reported by 37 percent of the subjects, and 18.5 percent of the men had previously worked in a dirty occupation. Among the 343 farm workers, there were three definite and six probable cases of farmer's lung (2.6 percent of those interviewed). Ten other men were classified as "possible farmer's lung" cases in view of their respiratory tract symptoms 4-12 hours after exposure to moldy hay. Many of these farm workers were heavy smokers, or had a past history of pulmonary disease.

80–0110. Torjussen, W. **Rhinoscopical Findings in Nickel Workers, With Special Emphasis on the Influence of Nickel Exposure and Smoking Habits.** *Acta Oto-Laryngologica* 88(3/4): 279-288, September-October 1979.

Rhinoscopy and X-ray examination were performed on 318 nickel workers and 57 controls, to study the significance of these methods in detecting cancerous and precancerous mucosal changes. The clinical and radiological findings were compared with histopathological data and mucosal nickel concentrations determined in nasal biopsy material from the middle turbinate, with duration of nickel exposure, and with tobacco smoking habits. Although nickel workers smoked larger amounts of tobacco than the controls, the difference in tobacco consumption was not statistically significant. The nickel-exposed subjects had statistically significantly more pathological changes (43 percent) than the controls (26 percent), ($0.01<p<0.02$), mainly due to differences in the frequency of hyperplastic rhinitis. Thirteen nickel workers (4 percent) had nasal polyps. Two of these cases, both employed at the nickel refinery for 28 years, appeared to have nasal carcinoma, according to histological examination. No distinct association was established between rhinoscopical findings and epithelial dysplasia found by histological examination. The explanatory values for the rhinoscopical findings of different factors, such as working category, age, duration of nickel exposure, grams tobacco smoked per week and nickel content of nasal mucosa, were evaluated by applying a stepwise multiple regression analysis. Number of years from first employment at the nickel refinery and tobacco consumption ($p<0.05$) were the only explanatory factors that showed a statistically significant correlation to the rhinoscopical findings. The radiological examination revealed few characteristic findings. Chemical analysis of cigarettes handrolled by nickel workers showed high nickel concentrations ($p<0.01$) compared with commercial brands of U.S. and Norwegian cigarettes. (Auth. Abs. Mod.)

See also, **80–0141, 80–0161**

CARDIOVASCULAR DISEASES

80-0111. Bianchi, G.; Fergusson, D.; Walshe, J. **Psychiatric Antecedents of Myocardial Infarction.** *Medical Journal of Australia* 1(6): 297-301, March 25, 1978.

This study examined the extent to which stress factors, personality or trait characteristics, and short-term mental state variables, such as anxiety and depression, are related to myocardial infarction (MI) risk. Interviews with 40 MI patients and 40 controls provided the following information: demographic and medical data, medical and psychiatric history, a Holmes and Rahe scale of life-events compilation, measures of the subject's mental status during the preceding 6 months, and measures of the usual personality of the patient. Three of the four measures of short-term mental state (State Anxiety Scale, Wakefield Depression Inventory Score, and the Cornell Medical Index Score) appeared to strongly discriminate between the study and control patients. These tests were highly correlated and appeared to be measuring some underlying common trait, probably anxiety-depression. MI patients were more upset, anxious, and worried (State Anxiety Scale items); they were also more restless, tired, irritable, and insomniac, and lost interest in things (Wakefield Depression Inventory items). The pre-MI period was characterized by elevated levels of anxiety and depression, which were often coupled with many adverse life events. The findings also confirmed the well-known associations between MI and smoking, family history of heart disease, and coffee intake. Comparison of the MI patients with the controls showed that 20 vs. 27.5 percent were nonsmokers, 22.5 vs. 27.5 percent were ex-smokers, and 57.5 vs. 45 percent were regular smokers. Although some well-known associations between myocardial infarction and physical factors were confirmed, the size of the effect of the physical variables was substantially smaller than that of short-term mental state or life-event variables. These results suggest that an examination of patients with a theoretical risk of myocardial infarction should be accompanied by a brief check on recent life history and current mental state. This may alert the doctor and the patient to extra factors which could influence the patient's well-being and future chances of survival.

80-0112. Criqui, M. H.; Austin, M.; Barrett-Connor, E. **The Effect of Non-Response on Risk Ratios in a Cardiovascular Disease Study.** *Journal of Chronic Diseases* 32(9/10): 633-638, 1979.

In a population-based cardiovascular disease study with a response rate of 82.1 percent, information on cardiovascular risk factors and diseases was obtained in both respondents and nonrespondents. Nonrespondents were more likely to be smokers, a risk factor which differs from the others studied (hyperlipidemia, hypertension, stroke, and diabetes) in that it is a self-selected variable not expected to fit the "worried well" concept, i.e., those with risk factors but without disease. In general, participants were a somewhat biased subset of the target population in that they tended to overrepresent the "worried well." This response bias represents independent bias in both risk factor and disease distribution. This "combined bias" produced minor to moderate errors in risk ratio calculations in our study. However, greater error in risk ratios could occur in a study with a similarly biased respondent sample and a lower response rate. (Auth. Abs. Mod.)

80-0113. Dyer, A. R.; Stamler, J.; Ubell, E.; Paul, O.; Lepper, M. H.; Berkson, D. M.; Lindberg, H. A. **A Self-Scoring Five-Question Risk Test for Coronary Heart Disease.** *Circulation* 60(4): 914-920, October 1979.

A simple, five question risk test for coronary heart disease (CHD) is presented. The test allows a person to estimate his or her relative risk of CHD, based on answers to questions on smoking habits, history of diagnosis, and treatment for high blood pressure, weight, pulse rate, and family history of heart disease. For smoking, possible scores ranged from 0 to 9, depending on the number of cigarettes smoked per day: none—0; half-pack—4; one pack—8; and >one pack—9. The constant terms were designed to provide a mean score close to 10 for most age-sex-race groups. A combined risk score of 15 for all risk factors would indicate that the individual's risk for coronary disease is about 50 percent higher than average for his age-race-sex group, while a score of 6 indicates a risk that is about 40 percent below average. The scoring system, developed from the 17-year CHD mortality study of 1,899 white males from 40 to 55 years of age in the Chicago Western Electric Company study, is applied to the men of that study and predictively to 1,158 white men from 40 to 59 years of age followed for 15 years in the Chicago Peoples Gas Company study. In these two studies, the men with the highest quintile of risk scores had more than four times the observed numbers of CHD deaths as the men in the lowest quintile of risk scores. In addition, a test that uses actual blood pressure instead of treatment status and includes serum cholesterol level, is also presented for use in cases where the values of these variables are available. Although the scoring systems for these tests are based on the mortality experience of middle-aged white males, the tests should have approximate validity for all age-sex-race groups because the scores are a measure of relative risk. (Auth. Abs. Mod.)

80-0114. Epstein, F. H. **Predicting, Explaining, and Preventing Coronary Heart Disease. An Epidemiological View.** *Modern Concepts of Cardiovascular Disease* 48(2): 7-12, February 1979.

An epidemiological review of coronary heart disease (CHD) is presented, with emphasis on predictive and preventive measures. The three most firmly established risk factors for CHD are elevated levels of serum cholesterol, blood pressure, and smoking. Approximately half of the new events of myocardial infarction and sudden death will occur among one-fifth of the population of middle-aged men at highest risk from these three factors, either singly or in combination, in terms of a quantitative risk index and the multiple logistic function. Data from the Pooling Project were used to predict CHD. Calculating for 1,000 middle-aged men the relative risk for CHD events, excluding angina pectoris, within the next observation period (8 1/2 years) was as follows: 15.9, smokers (S) only; 9.5, cholesterol (C) or diastolic blood pressure (H); 25.1, S + (C or H); 4.8, C + H; and 15.5, S + C + H.

80-0115. Gillum, R. F.; Paffenbarger, R. S., Jr. **Chronic Disease in Former College Students. XVII. Sociocultural Mobility as a Precursor of Coronary Heart Disease and Hypertension.** *American Journal of Epidemiology* 108(4): 289-298, October 1978.

The relationship between sociocultural mobility and subsequent coronary heart disease (CHD) and hypertension (HT) was studied in a cohort of 13,728 male former Harvard University students examined in 1939-1950. All 13,728 were followed for CHD mortality, while 8,852 returned self-administered mail questionnaires in 1962 or 1966 and in 1972 which queried for doctor-diagnosed myocardial infarction (MI), angina pectoris (AP) and HT. For each of 98 cases of fatal CHD, 78 cases of MI and 48 cases of AP, four controls were randomly selected. One control was selected for each of 319 HT cases. Significant negative associations between father's occupational status and risk of combined fatal CHD and MI, between geographic mobility and risk of HT, and between smoking history and fatal CHD, MI, and AP were noted in univariate analysis. Smoking was strongly associated with MI ($p<0.005$), CHD ($p<0.005$) and for fatal CHD and MI ($p<0.001$). These associations persisted with stratification by individual confounding factors (relative risk for smoking was 1.75) and by a multivariate confounder-summarizing score. Intergenerational mobility, as indicated by occupational status of the father, was associated with a 1.5 times increased risk of fatal CHD and MI. Intragenerational geographic mobility was associated with a slightly reduced risk of HT. (Auth. Abs. Mod.)

80-0116. Hasin, Y.; Eisenberg, S.; Friedlander, J.; Lewis, B. S.; Gotsman, M. S. **Relationship Between Extent of Coronary Artery Disease and Correlative Risk Factors.** *American Heart Journal* 98(5): 555-561, November 1979.

An analysis was made of correlative factors which might be related to the angiographically measured extent of coronary artery disease in 140 Israeli patients. Seventy-six were smokers. All patients had clinically important chest pain. Thirty-three had a normal coronary arteriogram. The extent of the atheromatous process was measured precisely at angiography by three different techniques. A coronary score, based on the percentage of luminal narrowing, was found to be best suited for the analysis. The most important contributory factors to the severity of atherosclerosis were duration of clinical history, number of previous myocardial infarctions, and male sex, but more specifically, elevation of serum cholesterol and diabetes mellitus. Cigarette smoking, obesity, hypertension, a family history of atherosclerosis, and elevated serum triglycerides had a positive influence, but this was not statistically significant. (Auth. Abs. Mod.)

80-0117. Haynes, S. G.; Feinleib, M.; Levine, S.; Scotch, N.; Kannel, W. B. **The Relationship of Psychosocial Factors to Coronary Heart Disease in the Framingham Study. II. Prevalence of Coronary Heart Disease.** *American Journal of Epidemiology* 107(5): 384-402, May 1978.

In a cross-sectional study of 1,822 persons (from 45 to 77 years of age) the association of 20 psychosocial scales with coronary heart disease (CHD) prevalence was assessed. Women (aged 45 to 64 years) with coronary disease scored significantly higher on the Framingham Type A behavior, emotional lability, aging worries, tension, and anger symptoms scales than women free of CHD. The prevalence of CHD was significantly higher among working women and housewives classified as having Type A rather than Type B behavior. Among men under 65 years, Framingham Type A behavior, aging worries, daily stress, and tension were associated with the prevalence of myocardial infarction (MI). For men and women over 65 years, marital dissatisfactions or disagreements were significantly related to the prevalence of CHD. In a multivariate analysis, the above associations were controlled for age, blood pressure, cholesterol, smoking, and other psychosocial scales. The number of cigarettes smoked per day was not an independent discriminator of CHD in either men or women. Among women under 65 years of age, Framingham Type A behavior and emotional lability remained significant discriminators of CHD prevalence. Aging worries significantly differentiated men under 65 with and without MI. Issues related to the interpretation of results from cross-sectional studies are discussed, and four hypotheses are suggested for future prospective work in this area. (Auth. Abs.)

80-0118. Hertzer, N. R.; Young, J. R.; Kramer, J. R.; Phillips, D. F.; deWolfe, V. G.; Ruschhaupt, W. F., III.; Beven, E. G. **Routine Coronary Angiography Prior to Elective Aortic Reconstruction. Results of Selective Myocardial Revascularization in Patients With Peripheral Vascular Disease.** *Archives of Surgery* 114(11): 1336-1344, November 1979.

Routine coronary angiography to determine the prevalence of severe coronary artery disease (CAD) has been recommended to all patients under consideration for elective peripheral vascular reconstruction at the Cleveland (Ohio) Clinic since April 1978. Forty-one of the 68 patients with abdominal aortic aneurysms (AAA) and 26 of the 71 patients with aortoiliac occlusive arterial disease (AI) had clinical evidence of CAD; coronary angiography demonstrated severe, correctable CAD in 23 patients with AAA and in 14 patients with AI. Twenty-seven patients with AAA and 45 patients with AI had no clinical evidence of CAD; severe, correctable CAD was found in six patients with AAA and in six patients with AI. Forty-six percent of AAA patients and 72 percent of AI patients were smokers. Significant coronary artery lesions (>70 percent stenosis of one or more coronary arteries) were documented by angiography in 75 percent of 32 patients in the AAA group who smoked, and significant coronary artery stenosis was identified in 53 percent of 51 patients in the AI group who smoked. Those who had stopped smoking within the past 5 years were included as smokers. (Auth. Abs. Mod.)

80-0119. Lyon, J. L.; Wetzler, H. P.; Gardner, J. W.; Klauber, M. R.; Williams, R. R. **Cardiovascular Mortality in Mormons and Non-Mormons in Utah, 1969-1971.** *American Journal of Epidemiology* 108(5): 357-366, November 1978.

Cardiovascular mortality in the State of Utah is among the lowest in the U.S. The religion of 72 percent of the State residents (Mormon) proscribes the use of tobacco and alcohol; a large number of Mormons adhere to this proscription. This study analyzed the 6,108 deaths from cardiovascular diseases among members of the Mormon Church in Utah between 1969 and 1971. For both sexes, Mormons had 35 percent less mortality than expected from U.S. rates for ischemic heart disease, while non-Mormons were not significantly different from U.S. whites. These results support the relationship between cigarette smoking and mortality from cardiovascular disease. Mormon men also had lower mortality from hypertensive heart disease and Mormon women from rheumatic heart disease than non-Mormons in Utah. (Auth. Abs.)

80-0120. Maccoby, N.; Farquhar, J. W.; Wood, P. D.; Alexander, J. **Reducing the Risk of Cardiovascular Disease: Effects of a Community-Based Campaign on Knowledge and Behavior.** *Journal of Community Health* 3(2): 100-114, Winter 1977.

In 1972, the Stanford Heart Disease Prevention Program launched a three-community field study. A multimedia campaign was conducted for 2 years in two California communities (Watsonville and Gilroy). In Watsonville, it was supplemented by an intensive-instruction program with high-risk subjects. A third community (Tracy) was used as a control. The campaigns were designed to increase participants' knowledge of the risk factors for cardiovascular disease, to change such risk-producing behavior as cigarette smoking, and to decrease the participants' dietary intake of calories, salt, sugar, saturated fat, and cholesterol. Results of a sample survey indicate that substantial gains in knowledge, in behavioral modification and in the estimated risk of cardiovascular disease can be produced by both methods of intervention. The intensive-instruction program, when combined with the mass media campaign, emerged as the most effective for those participants who were initially evaluated to be at high risk. Compared with the nonintervention town (T), only when intensive instruction was included in the total community and high risk samples was there a substantial reduction in percent change from baseline in the number of cigarettes smoked per day. One year values for total community vs. T were -18.9 vs. -1.1, $p<0.05$; and two year values were -24.1 vs. -2.5, $p<0.05$. One year values for high risk vs. T were -36.3 vs. -8.5, $p<0.05$; and two year values were -42.3 vs. -17.2, $p<0.05$. Similar results are reported for effects on knowledge and behavioral change for the total participant samples and for the high-risk subsamples in each of the three communities. (Auth. Abs. Mod.)

80-0121. Mahendru, R. K.; Alam, S. M.; Sikka, K. K. **Smoking Habits and Emotional Factors in Coronary Heart Disease.** *Journal of the Association of Physicians of India* 26(10): 909-914, October 1978.

The relationship of emotional factors to smoking habits in the development of coronary heart disease was studied in 76 patients (75 males) with myocardial infarct, angina pectoris, and coronary ischemia. Each patient underwent a complete psychiatric assessment, including a clinical interview and administration of the Maudsley Personality Inventory (MPI). Smoking habits and any emotional stress the patient experienced prior to the onset of coronary disease were also recorded. Over two-thirds of the patients (71.1 percent) were smokers. The percentage of schizoid, obsessive, and emotionally unstable personalities was greater in smokers than nonsmokers (77.8 vs. 63.6 percent). Neuroticism scores were significantly higher ($p<.05$) and extroversion scores significantly lower ($p<.05$) when smokers were compared to nonsmokers. Patients who had experienced stress prior to the onset of coronary disease had longer smoking histories and smoked more heavily than those without stress.

80-0122. Milvy, P. **Statistical Analysis of Deaths From Coronary Heart Disease Anticipated in A Cohort of Marathon Runners.** *Annals of the New York Academy of Sciences* 301: 620-626, 1977.

A prospective study to evaluate the influence of marathoning on the incidence of mortality from heart disease was carried out in 9,958 male marathoners. Controls were 9,958 white American males whose age distribution was the same as the marathoners, but whose relative weight and smoking habits were the same as the general American male population. Marathoners are characterized by the virtual absence of these two risk factors for coronary heart disease (CHD). Deaths per year expected from a cohort equivalent in age distribution, smoking habit, and weight to the marathoners, taken from data on mortality for ischemic and related heart disease in 1973 for metropolitan policy holders, is only one or two per year. For the general insured American male population, 5.37 deaths per year would be expected. If marathoning conferred absolute protection from death from ischemic and related heart disease, no CHD deaths per year from the 10,000 cohort of male marathoners would be expected. If it provided 50 percent protection, about one runner per year would be expected to die from this disease, while if it provided no protection, an average of one or two runners per year would be expected to die. In addition to the formidable problems of discovering these deaths, were death to occur at all, it is clear that the statistics are unable to assess meaningful differences in risk factors in the two cohorts, let alone provide evidence that marathoning prevents CHD.

80-0123. Moss, A. J.; Davis, H. T.; DeCamilla, J.; Bayer, L. W. **Ventricular Ectopic Beats and Their Relation to Sudden and Nonsudden Cardiac Death After Myocardial Infarction.** *Circulation* 60(5): 998-1003, November 1979.

The role of ventricular ectopic beats (VEBs) in identifying patients who die of cardiac cause in the posthospital phase of myocardial infarction was evaluated in 940 patients who survived an acute coronary event. Six-hour Holter ECG recordings were obtained before hospital discharge, and VEBs were classified as complex (bigeminal, multiform, repetitive or R on T), simple (one or more VEBs that did not have complex patterns), or not present. Patients were followed 1-60 months (average 36 months) and cardiac mortality was categorized as sudden (<1 hour) or nonsudden (>1 hour) among 98 witnessed cardiac deaths. Complex VEBs were associated with a significantly increased cardiac death rate, but did not discriminate between sudden and nonsudden death. Simple VEBs were associated with a 3-year cardiac mortality rate intermediate between those with complex and those with no VEBs. The relationship between complex VEBs and cardiac mortality was independent of 10 relevant clinical variables, including myocardial infarction and cigarette smoking histories. Smokers accounted for 60 percent of the total population, 64 percent of the cardiac death group, 59 percent of the sudden death group, and 70 percent of the nonsudden death group. (Auth. Abs. Mod.)

80-0124. Naik, R. S.; Tripathi, B. P.; Vaidya, R. C.; Sao, V. K. **Buerger's Disease *versus* Adrenalectomy and Sympathectomy.** *Journal of the Indian Medical Association* 71(8): 199-202, October 16, 1978.

The natural history, clinical diagnostic criteria, and efficacy of unilateral adrenalectomy and sympathectomy were evaluated in a group of 14 male patients (from 20 to 45 years) who were followed up for 1-1/2 years. All of the subjects were bidi smokers, considered a possible cause of Buerger's disease. Adrenalectomy combined with sympathectomy appeared to be definitely superior to sympathectomy alone and resulted in an increased time span between the onset of the disease and amputation of the limb.

80-0125. Regan, T. J. **Of Beverages, Cigarettes, and Cardiac Arrhythmias.** *New England Journal of Medicine* 301(19): 1060-1062, November 8, 1979.

Alcohol, cigarettes, and coffee do not appear to precipitate cardiac arrhythmias in healthy persons who have no underlying cardiac risk factors. There is no clear evidence that their combined use, in moderation, disturbs cardiac rhythm in normal subjects. However, excessive fatigue or physical effort may increase susceptibility to the effects of these agents on the heart. In patients with unexplained heart irritability, or those who have arrhythmias in association with mitral-valve prolapse or hypertrophic subaortic stenosis, the response to the use of these agents should be carefully studied by electrocardiographic monitoring at rest and during exercise.

80-0126. Rissanen, A. M. **Familial Occurrence of Coronary Heart Disease: Effect of Age at Diagnosis.** *American Journal of Cardiology* 44: 60-66, July 1979.

The occurrence of coronary heart disease and its main risk factors were assessed among the first degree relatives of 309 men from South and East Finland, including 203 men with fatal or nonfatal myocardial infarction and 106 healthy reference men under age 56 years. The younger the patient at the diagnosis of a first myocardial infarction, the more common was coronary heart disease in his parents and siblings. The risk of having coronary heart disease by age 55 was, respectively, 11.4, 8.3, and 1.3 times greater in the South and 6.7, 3.6, and 1.8 times greater in the East for the brothers of patients than for the brothers of reference subjects depending on whether the diagnosis of myocardial infarction in the patient had first been established before the age of 46 years, age 46 to 50 years, or at age 51 to 55 years. Hypertension and hyperlipidemia, but none of the other risk factors studied (relative weight, diabetes, and smoking), were most common among the relatives of the youngest patients and diminished in frequency with advancing age of the patient. Most of the strong familial component in coronary heart disease of early onset thus appears to be mediated by familial hyperlipidemias and hypertension. Smoking habits also appear to modify the manifestation of the disease in these high risk persons; coronary heart disease developed in heavy smokers an average of 4 years earlier than in their nonsmoking siblings. It is suggested that the risk of premature coronary heart disease in the persons at highest risk could be largely eliminated if information about family history were used to identify such persons at an early stage and if they were treated properly for their correctable risk factors. (Auth. Abs. Mod.)

80-0127. Thomas, P. C.; Cady, L. D.; O'Connell, E. R.; Bischoff, D. P.; Kershnar, R. K. **Heart Disease Risk Factors in Los Angeles County Safety Personnel.** *Journal of Occupational Medicine* 21(10): 683-687, October 1979.

CARDIOVASCULAR DISEASES

Eight generally accepted heart disease risk factors (age, sex, family history, blood pressure, cholesterol, weight, smoking, and exercise) were used in the computation of a cardiac index (RISKO) for 4,066 safety personnel of the County of Los Angeles. Seventy-one individuals subsequently developed coronary disease. These individuals were older, had a slightly higher prevalence of family history of heart disease, had higher resting blood pressures, had higher fasting serum cholesterol levels, had more excess fat, smoked cigarettes more frequently (60 percent vs. 37 percent), and were more inactive than individuals in the asymptomatic group. Thirty percent of the coronary group had high RISKO scores (above 31), whereas less than 10 percent of the asymptomatic individuals had scores of this magnitude. The index had significant value in identifying the individuals who were to develop symptomatic coronary disease, and its discriminative power was comparable to the power of the Framingham index.

80–0128. Vaisrub, S. **Another Kick in the (Cigarette) Butt.** *Journal of the American Medical Association* 242(11): 1175, September 14, 1979.

The association of smoking with malignant hypertension and subarachnoid hemorrhage is reviewed. In one study 69 percent of the patients with malignant hypertension were smokers compared to 37 percent of the nonmalignant hypertensives. The difference was even more significant in another study where the prevalence of smoking was 82 percent among the patients with malignant hypertension compared to 43-52 percent among the controls. The association between smoking and subarachnoid hemorrhage became apparent in a retrospective study of patients with ruptured cerebral aneurysms. Smoking was found to increase the risk of subarachnoid hemorrhage 3.9 times for men and 3.7 times for women. An even higher risk was reported in the Walnut Creek Contraceptive Study where subarachnoid hemorrhage was 5.7 times more common among smokers than among nonsmokers.

See also, **80–0076, 80–0085**

PREGNANCY AND INFANT HEALTH

80–0129. Bodendorfer, T. W.; Briggs, G. G.; Gunning, J. E. **Obtaining Drug Exposure Histories During Pregnancy.** *American Journal of Obstetrics and Gynecology* 135(4): 490-494, October 15, 1979.

One hundred and fifty-three pregnant clinic patients were included in this study to determine the drugs ingested during their pregnancies. Drug histories were obtained through interviews conducted by the hospital pharmacist and through the use of home diaries. In addition, each patient's medical chart was reviewed by the pharmacist to obtain a chart drug history. The results demonstrate that pregnant patients are exposed to a variety of potentially teratogenic agents. Sixty-nine percent of the patients had taken aspirin during their pregnancies. The next most frequently ingested drugs were vitamins, iron supplements, antacids, and cigarette smoke. Forty-three percent were cigarette smokers (5-1/2 percent smoked one pack of cigarettes or more a day). The medical charts recorded only 23 to 26 percent of the drugs recorded by the pharmacist's history and are therefore grossly inadequate in giving an accurate assessment of a patient's exposure to drugs during pregnancy.

80–0130. Edwards, L. E.; Alton, I. R.; Barrada, M. I.; Hakanson, E. Y. **Pregnancy in the Underweight Woman: Course, Outcome, and Growth Patterns of the Infant.** *American Journal of Obstetrics and Gynecology* 135(3): 297-302, October 1, 1979.

The obstetric performance and pregnancy outcome of 354 underweight patients were compared with matched control subjects of normal weight. The growth patterns of their infants were also compared. The underweight women had significantly higher rates of cardiac/respiratory problems, anemia, premature rupture of membranes, and endometritis but were less prone to develop pre-eclampsia. Prematurity and low Apgar scores were significantly more frequent in the infants of underweight women, with greater incidence of low Apgar score infants born to underweight smokers (19.3 percent) than born to control subjects (6.5 percent, $p<0.02$). This finding was observed only in infants of underweight women who smoked more than one pack of cigarettes per day. There was no difference in the frequency of intrauterine growth retardation and in perinatal mortality rates. The mean birthweight of the infants of underweight women was 231 grams less than that of infants of control subjects. Underweight women, particularly if they were anemic, had a higher incidence of low-birthweight infants despite adequate weight gain. Appropriate for gestational age infants of underweight women were more likely to be below the twenty-fifth percentile for weight correlated with length by 12 months of age. (Auth. Abs. Mod.)

80–0131. Ericson, A.; Kallen, B.; Westerholm, P. **Cigarette Smoking as an Etiologic Factor in Cleft Lip and Palate.** *American Journal of Obstetrics and Gynecology* 135(3): 348-351, October 1, 1979.

A case-control study has been made on smoking habits in women who, during 1975, gave birth to infants with closure defects of the central nervous system (ASB) or with cleft lip or cleft palate (CLP). For each case, two control subjects with nonmalformed infants were selected and matched for delivery unit, time of delivery, maternal age, and maternal parity. Smoking habits were routinely included in hospital records at the first visit to a maternity health clinic during pregnancy. Data were studied for 66 cases of CLP, 66 cases of ASB, and 261 control subjects. Significantly more women who had infants with CLP smoked than did control women ($p<0.01$), but women with ASB infants showed a normal smoking pattern. Drug use did not explain the findings. It is suggested that maternal smoking is one of many important factors in the etiology of cleft lip and cleft palate in humans. (Auth. Abs.)

80-0132. Haas, J. F.; Schottenfeld, D. **Risks to the Offspring From Parental Occupational Exposures.** *Journal of Occupational Medicine* 21(9): 607-613, September 1979.

Risks to the offspring of workers exposed to occupational chemicals are reviewed. These risks may result from parental exposure to industrial agents which are mutagenic, teratogenic, or carcinogenic. Evidence is reviewed for hazards to the offspring of parents with occupational exposure to vinyl chloride, benzene, chloroprene, radiation, and petroleum-derived hydrocarbons. Other behavioral and environmental factors (i.e., smoking, alcohol, and drug exposures) should also be considered when investigating the role of occupational factors in pregnancy outcome. Smoking has been shown to increase the risk of spontaneous abortion by a factor of 1.8, yet the percentage of aborted fetuses which are chromosomally abnormal is close to that expected. The absolute risk of a chromosomally abnormal fetus appears to be higher in smoking mothers. A survey of professional medical women identified a relative risk of 1.7 for spontaneous abortion and a risk of 2.3 for congenital abnormalities in heavy smokers. An approach to the surveillance of chromosomal abnormalities in the offspring of occupationally exposed parents is outlined. Future investigations should take into account other environmental and behavioral factors which can affect fetal development if causal associations between occupational exposure and pregnancy outcome are to be identified.

80-0133. Harlap, S.; Shiono, P. H.; Ramcharan, S.; Berendes, H.; Pellegrin, F. **A Prospective Study of Spontaneous Fetal Losses After Induced Abortions.** *New England Journal of Medicine* 301(13): 677-681, September 27, 1979.

The incidence of spontaneous abortions was observed among 31,917 women followed from their first prenatal visit. Life-table analysis showed that losses in the first trimester were not significantly affected by previous induced abortions, nor was any change in the risk of second-trimester losses detected among the 1,493 parous women who reported having had induced abortions after childbirth. There was, however, an increase in the incidence of midtrimester losses among the 2,019 nulliparous women with previous induced abortions; the age-adjusted rate of loss was 59.9 per 100,000 women at risk per day, as compared with 24.2 among the 12,042 control nulliparous women ($p<0.001$). The relative risk increased with the number of previous induced abortions and was not explained by the distribution of demographic and social variables. When the social variable smoking was considered, the relative risk for spontaneous fetal losses in women with previous abortions was 0.98 for nonsmokers and 1.30 for smokers in the first trimester. In the second trimester, the relative risk increased to 1.77 in nonsmokers and 2.48 in smokers. The risk decreased from 3.27 (95 percent confidence limits, 1.72 to 6.23) after what were mainly dilation and curettage induced abortions prior to 1973, to 1.42 (0.76 to 2.65) after the more gentle technique of cervical dilation by use of laminaria was introduced since 1973. These findings indicate that there is little or no risk of spontaneous abortions after induced abortions when performed by current techniques. (Auth. Abs. Mod.)

80-0134. Rantakallio, P. **The Effect of Maternal Smoking on Birth Weight and the Subsequent Health of the Child.** *Early Human Development* 2(4): 371-382, 1978.

The effect of maternal smoking during pregnancy on birthweight was studied in 12,068 births. Of the mothers, 1,819 were smokers. The children of smokers were compared with those of controls of similar age, parity, marital status, and place of residence. Maternal smoking reduced birthweight in a dose-related manner. Birthweight was least affected in infants of young, primiparous mothers who smoked only slightly, a difference which was, however, entirely caused by those mothers who stopped smoking for the last 3 months of pregnancy. Children in this subgroup had similar postneonatal mortality and morbidity to those of controls, up to the age of 5. Postneonatal mortality was higher in the total group of smokers' infants than among controls: infants of primiparous or young mothers not differing in this respect from the others. Morbidity up to the age of 5 was significantly higher ($p<0.001$) among the children of smokers: the children of primiparas and young mothers being affected in a similar way to the others. The low birthweight infants of smokers had a higher mean birthweight and lower perinatal mortality than the low birthweight infants of controls, but morbidity up to age 5 and postneonatal mortality were higher among the infants of smokers for both low birthweight infants and others. (Auth. Abs.)

80-0135. Ritchie, J.W.K. **Fetal Breathing and Generalized Fetal Movements in Normal Antenatal Patients.** *British Journal of Obstetrics and Gynaecology* 86(8): 612-614, August 1979.

Fetal breathing movements (FBM) and generalized fetal movements (GFM) were recorded for 30 minutes using a real time B-scan instrument in 46 women with uncomplicated pregnancies. Of the patients 32 were nonsmokers and 14 were smokers (cigarettes smoked/day ranged from 5 to 30, mean = 17). The interval between smoking and recording was noted in 12 patients and the mean was 99 ± 11 minutes. The time occupied by FBM was not significantly related to smoking, (22.5 ± 5.0 percent for smokers and 29.9 ± 4.6 percent for nonsmokers), meal times or plasma glucose concentrations. There was also no correlation between the time occupied by FBM and the time interval between the last cigarette and recording. FBM were absent during the observation period in 15 percent of patients and present for less than 10 percent of the time in 33 percent of patients. (Auth. Abs. Mod.)

80-0136. Saxton, D. W. **The Behaviour of Infants Whose Mothers Smoke in Pregnancy.** *Early Human Development* 2(4): 363-369, 1978.

The Brazelton Neonatal Behavioral Assessment Scale was used to compare the behavior patterns of infants of mothers who smoked during pregnancy and those whose mothers did not smoke. Smokers were defined as those who had smoked more than 15 cigarettes/day throughout their pregnancy. Over a period of 9 months a total of 32 infants (15 whose mothers smoked and 17 whose mothers did not smoke) between 4 and 6 days of age were examined and the results compared. The two groups were matched for maternal age, social class and parity. All infants were spontaneously delivered at term and were of normal birthweight. Sex distribution was equal. Duration of labor and analgesia during labor were similar for smokers and nonsmokers. Examinations were carried out in a warm, quiet semi-dark room, the examiner being unaware of which category the infant was in. There was evidence to suggest that the behavioral patterns of infants can be influenced by smoking in pregnancy and that in particular the auditory senses are affected. (Auth. Abs.)

80-0137. Younoszai, M. K. **Effect of Maternal Smoking on Hemoglobin and Hematocrit of the Newborn. (Letter).** *American Journal of Clinical Nutrition* 32(10): 1983, October 1979.

Reported confirmation of raised hematocrit values in infants of smoking mothers (*American Journal of Clinical Nutrition* 31(4): 557-558, April 1978) is of interest. Mean carbon monoxide saturation of hemoglobin in 10 cigarette smoking mothers was 8.3 percent, and in 11 nonsmoking mothers was 1.2 percent. Corresponding mean cord vein blood levels were 7.3 and 0.7 percent. See also 79-0493.

OTHER DISEASES AND CONDITIONS

80-0138. Camarasa, G.; Alomar, A. **Menthol Dermatitis From Cigarettes.** *Contact Dermatitis* 4(3): 169-170, June 1978.

A case of menthol dermatitis from cigarettes is reported in a 26-year-old female typist. The patient was suffering from a mild scaly erythema with irregular edges, which had been recurring for 2 years. It affected the upper lip, the perioral area, and the dorsal aspect of the central fingers of both hands. She was referred to a contact dermatitis clinic where she participated as a control in a clinical experiment. Four pharmaceutical products of unknown composition which were to be used as disinfectants and mouth fresheners were tested on healthy subjects and on others with known sensitivities. The patient exhibited a strong positive response to one of the products which contained menthol and essence of mint. The patient regularly smoked menthol cigarettes, as well as other types of cigarettes and tobacco. Since the patient has stopped smoking menthol cigarettes, she has been totally free of symptoms.

80-0139. Landecker, K. D.; Crawford, J.; Hunt, J. H.; Gillespie, P.; Piper, D. W. **Cimetidine and Gastric Ulcer Healing. A Double-Blind Controlled Trial.** *Medical Journal of Australia* 2(1): 43-45, July 14, 1979.

The effect of cimetidine in the daily dose of 1,200 mg on the healing rate of chronic gastric ulcer was assessed in a randomized double-blind trial in 48 patients. Cimetidine was found to accelerate the healing of chronic gastric ulcers in the ambulant patients, but it conferred no additional benefit on the hospitalized patients. Neither hospitalization, sex, smoking, analgesics, or alcohol had a statistically significant effect on the rate of healing. No significant side effects were observed. (Auth. Abs.)

80-0140. Pullan, P. T.; Clappison, B. H.; Johnston, C. I. **Plasma Vasopressin and Human Neurophysins in Physiological and Pathological States Associated With Changes in Vasopressin Secretion.** *Journal of Clinical Endocrinology and Metabolism* 49(4): 580-587, October 1979.

Using sensitive specific radioimmunoassays (RIAs) for vasopressin (AVP) and the two major human neurophysins, the relationship between AVP and the individual human neurophysins was investigated in man by measuring changes in plasma concentrations in physiological and pathological states known to be associated with changes in AVP secretion. Dehydration, water loading, and hemorrhage produced small but significant changes in plasma AVP concentrations without changes in the individual human neurophysins. In response to the stimulus of cigarette smoke inhalation, large parallel changes in plasma AVP and human neurophysin I (HNPI) levels were seen in two of the five subjects without change in plasma human neurophysin II (HNPII) levels. Plasma AVP and HNPI concentrations correlated significantly in each of these two subjects ($p<0.01$ and $p<0.001$, respectively). In the pathological states of diabetes insipidus and the syndrome of inappropriate antidiuretic hormone secretion, the observations more strongly supported a specific association between AVP and NHPI. In eight patients with central diabetes insipidus, plasma AVP and HNPI levels were low or undetectable, while plasma HNPII levels were normal. There was a clear distinction of both plasma AVP and HNPI levels in patients with central diabetes insipidus and in patients with nephrogenic diabetes insipidus. In 14 patients with the syndrome of inappropriate antidiuretic hormone secretion due to causes other than ectopic AVP production from tumors, plasma AVP and HNPI levels were elevated or normal, while plasma HNPII levels were normal. There was a highly significant positive correlation ($r = 0.99$) between plasma AVP and HNPI levels in these patients, with a 1:1 molar ratio. These data suggest that the secretion of AVP and HNPI in man are functionally related, while the secretion of HNPII is independent of AVP secretion. (Auth. Abs. Mod.)

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–0141. Benatar, S. R. **Smoking and Chronic Respiratory Symptoms in 11-15-Year-Old Children.** *South African Medical Journal* 56(8): 301-303, August 25, 1979.

A two part questionnaire was used to assess the prevalence of smoking and chronic respiratory symptoms in 315 white (W) and 290 colored (C) children between the ages of 11 and 15. The first part considered smoking, chronic respiratory symptoms, and history of previous hospitalization for chest diseases. The second part covered the same areas for the parents and the siblings. The quantity and duration of smoking, reasons for starting the habit, and social influences were examined. The peak expiratory flow rate, forced expiratory volume in one second, forced vital capacity, and maximum mid-expiratory flow rate were measured for each child. Two and one-half percent of the W children (all male) and 15.2 percent of the C children (68 percent male) were smokers. The overall prevalence of smoking in C fathers and mothers (85 and 56 percent, respectively) was greater than that in W fathers and mothers (50 and 43 percent, respectively), but within each ethnic group, there were no significant differences in the prevalence of smoking by parents of smoking and nonsmoking children. Both groups of children who smoked had significantly more sibling smokers than did nonsmoking children (W, $p<0.01$; C, $p<0.0005$). Twelve of the 52 children who smoked had chronic respiratory symptoms. The incidence of chronic respiratory symptoms in the parents was 13.6 percent in W parents and 34.3 percent in C parents ($p<0.0005$).

80–0142. Chien, C.-P.; Townsend, E. J.; Ross-Townsend, A. **Substance Use and Abuse Among the Community Elderly: The Medical Aspect.** *Addictive Diseases* 3(3): 357-372, 1978.

Two hundred and forty-two community living elderly persons with an average age of 73 years were studied in order to disclose the prevalence and scope of use and abuse of substances by this age group. A specially designed questionnaire was used to obtain information on socioeconomic status, degree of social and familial isolation, health status, and the substance usage pattern. The 242 individuals were taking a total of 301 kinds of prescribed and over-the-counter drugs, of which 60 percent were prescription drugs. The mean number of drugs taken per interviewee was 3.8 with a range of 0-15 drugs. Of the study group, 83 percent were taking 2-6 drugs and 14 percent were on 7-15 drugs. Of the subjects, 15 percent smoked 1/2 to 1 pack per day, 4 percent 1 pack or less per week, and 6 percent 2-3 packs per day; the rest were nonsmokers. Abuse of alcohol and narcotics was not found in this group, at least with the study method utilized. Although intentional abuse was not found, iatrogenic hazards of drug interaction from polypharmacy could be a serious and often unreported problem. Interference of laboratory tests by drugs could also delay or mask the accurate diagnosis of hidden illness. (Auth. Abs. Mod.)

80–0143. Clark, R. R. **Cigarette Smoking in Social Interaction.** *International Journal of the Addictions* 13(2): 257-269, 1978.

The correlation of behavior patterns by smokers and others in social interaction with various behaviors comprising cigarette smoking was studied. Field observation methods were used in observing 91 smokers in 117 social interaction settings. Settings were chosen to vary in degree of intimacy between participants in social interaction, and in degree of physical activity. Smoking characteristics (light-up, drag, exhalation, tap of ashes, holding of cigarette, put-out) were noted in a variety of social interactions. Reliability of observations was established using independent field observers and raters of a videotape of one interaction. Findings generally were that smoking behaviors occur at points in the interaction when the smoker is relatively uninvolved and passive in the context of the social interaction. It is speculated that smoking is a form of self-involvement which is incompatible, on a momentary basis, with active social involvement. (Auth. Abs. Mod.)

80-0144. Dicken, C.; Bryson, R. **Psychology in Action: The Smoking of Psychology.** *American Psychologist* 33(5): 504-507, May 1978.

Academic psychologists (N = 344) were studied in regard to cigarette smoking and its correlates. Male psychologists were less likely to smoke than unselected U.S. adult males but were similar to most professional comparison groups. The women psychologists were more likely to smoke than the men because relatively fewer had stopped. Most smokers reported the expectation of reducing or stopping smoking in the future. Substantial proportions of both sexes who had ever smoked and who had reached middle age had in fact stopped. Among those of advanced academic rank, smokers were significantly higher in book and article authorship. The sex differences are interpreted in terms of sex roles and role stress. The productivity data are interpreted relative to personality differences, work roles, and the arousal effect of nicotine. (Auth. Abs.)

80-0145. Eiser, J. R.; Sutton, S. R. **Smoking as a Subjectively Rational Choice.** *Addictive Behaviors* 2(2/3): 129-134, 1977.

In order to identify variables which might influence adult smokers to attempt to give up smoking, questionnaires were distributed to a sample of 45 public employees who were cigarette smokers. These offered subjects the chance to apply for treatment at a nearby smokers' clinic and also asked questions about their own smoking history, their perceived dependence on smoking, their perceptions of utilities associated with continuing smoking or attempting to stop smoking and succeeding or failing in the attempt. Of the 45 subjects, 18 chose to apply for treatment, and a discriminant analysis was performed to see if these could be distinguished from the 27 "nonchoosers" in terms of their questionnaire responses. The most important predictor was the number of cigarettes smoked per day, with choosers on the average smoking over 30 and nonchoosers under 18 per day. In addition, choosers tended to be slightly older, and enjoy the activity of smoking less than nonchoosers. Nonchoosers, as compared with choosers, were relatively confident in their own ability to stop smoking without help, if they wanted to. Although subjects as a whole tended to rate the overall consequences of continuing to smoke as worse than the overall consequences of successfully stopping, this was not an important predictor of which subjects chose to apply for treatment. (Auth. Abs.)

80-0146. Eiser, J. R.; Sutton, S. R.; Wober, M. **Smokers, Nonsmokers and the Attribution of Addiction.** *British Journal of Social and Clinical Psychology* 16(4): 329-336, 1977.

Questionnaires concerned with attitudes towards cigarette smoking were completed by 368 respondents who were taking part in a survey of audience reactions to one week's television programs. Cigarette smokers were asked how difficult it would be for them to give up cigarettes, whether they would like to do so, whether they felt they were addicted to cigarettes, and how often a cigarette gave them real pleasure. Nonsmokers were asked parallel questions to determine their perceptions of the average cigarette smoker. In comparison to how smokers saw themselves, nonsmokers saw the average smoker as more addicted, and as deriving less pleasure from cigarettes. Smokers who saw themselves as more addicted felt it would be more difficult for them to give up, smoked more cigarettes per day, said they would like to give up more, and derived somewhat more pleasure from cigarettes. Among those who had never smoked cigarettes, the attribution of addiction to the average smoker was related only to perceived difficulty of giving up. Lesser self-attributed addiction was also relevant to the prediction of which smokers claimed to be trying to reduce or give up smoking. Implications of these data for attribution theory, and for health education, are discussed. (Auth. Abs.)

80-0147. Eiser, J. R.; Sutton, S. R.; Wober, M. **Smokers' and Non-Smokers' Attributions About Smoking: A Case of Actor-Observer Differences?** *British Journal of Social and Clinical Psychology* 17(2): 189-190, 1978.

Smokers' and nonsmokers' attributions about cigarette smoking were compared in a sampling of 314 subjects, aged 18 years and over, in Great Britain. Of these, 144 were males and 170 females, 115 were classified as smokers, 117 as never-smokers, and 82 as ex-smokers. Those who had never smoked underestimated the amount of pleasure claimed by smokers ($p<0.01$) and the degree to which smokers were concerned about the health risks ($p<0.001$). Smokers attributed less influence to advertisements and to seeing actors smoke than did never-smokers ($p<0.01$ and $p<0.05$, respectively). Younger subjects attributed more importance than older subjects to the influence of family members ($p<0.01$) or friends ($p<0.001$) who were smokers, but viewed smoking as less pleasurable ($p<0.05$).

80-0148. Erickson, L.G.W.; Cramer, J. A. **Smoking Behavior Development and Modification: An Empirical Application of Three Social Psychological Theories.** *Journal of Applied Social Psychology* 6(4): 369-386, 1976.

Three major social psychological theories (reference group theory, psychoanalytic theory, and cognitive dissonance theory) were applied to data obtained from interviews with a random sample of 50 ex-smokers and 50 smokers. Current smokers were persons who had smoked at least 5 packs of cigarettes during their lifetime and were still smoking. Persons who had smoked at least 5 packs of cigarettes during their lifetime and were no longer smoking were defined as ex-smokers. Subjects were questioned about demographic information, smoking history, time of quitting smoking, parents' and friends' smoking habits, and who influenced them to start and to stop smoking. Two sets of hypotheses were derived from the three theories: one set compared the ex-smokers' smoking and nonsmoking periods of their lives; and another set consisted of comparisons between smokers and ex-smokers. Reference group hypotheses were supported by the former set. During the ex-smokers' smoking period, 78 percent had family members who smoked, 78 percent had close friends who smoked, and 82 percent felt that smoking was socially acceptable. During the ex-smokers' nonsmoking period, 30 percent had family members who smoked, 20 percent had close friends who smoked, and 32 percent felt that smoking was socially acceptable. Additional hypotheses tested showed a positive relationship between a person's smoking behavior and his/her family's smoking patterns. Significant differences existed between behavior patterns of smokers and ex-smokers. The majority of the subjects who stopped smoking started some other oral habit. The results indicate that fewer current smokers have changed their opinions concerning smoking and health. It is possible that cognitive dissonance may not always exist for them. While 54 percent of the ex-smokers had gained self-satisfaction, only 8 percent of the smokers thought that they would gain self-satisfaction if they quit smoking. If it is assumed that dissonance begins when the person becomes aware of the dangers of smoking, then the evidence of widespread awareness among smokers does not support a cognitive dissonance explanation of motivation for quitting. The reference group theory hypotheses were strongly supported by the study results. The psychoanalytic theory and the cognitive dissonance theories were supported to a lesser extent, but the psychoanalytic hypotheses were the most difficult to test, and they remained inconclusive.

80-0149. Fuller, R.G.C.; Forrest, D. W. **Cigarette Smoking Under Relaxation and Stress.** *Irish Journal of Psychology* 3(3): 165-180, 1977.

Although nicotine is a stimulant, cigarette smoking is often associated with relaxation. Nicotine may have opposite effects depending on the level of arousal of the smoker, tending to shift him away from both extremes of the arousal continuum. To test this hypothesis, the effects of smoking a cigarette were compared with a simulation condition in subjects either relaxing on a couch or watching a stressful film. The measures of arousal used were heart rate, skin resistance level, and isometric muscle tension. Eighty subjects of each sex comprising 40 light and 40 heavy smokers were assigned to either low or high arousal conditions. In the low arousal condition smoking led to large and significant increases in heart rate ($p<.01$), no change in skin resistance level, and a small but significant decrease in muscle tension after the cigarette had been extinguished ($p<.01$ and $p<.05$ for two sites). Under high arousal smoking again led to significant increases in heart rate ($p<.01$), but the impact of the stress episodes of the film was the same for both smoking and simulation conditions. No differences between conditions were observed in the skin resistance level of the electromyogram, although a tendency to lower muscle tension under the smoking condition was noted. Lack of covariation in the physiological measures suggested that they were not simply reflecting changes in the same underlying variable, arousal, and it was argued that smoking-induced increases in heart rate may be interpreted as facilitating rejection of the environment. (Auth. Abs.)

80-0150. Garfinkel, L. **Changes in the Cigarette Consumption of Smokers in Relation to Changes in Tar/Nicotine Content of Cigarettes Smoked.** *American Journal of Public Health* 69(12): 1274-1276, December 1979.

Over a 13-year period, 59 percent of 28,561 smokers decreased the tar and nicotine (T/N) level in the cigarettes they smoked without changing the number of cigarettes smoked to any important extent (34 percent smoked the same number of cigarettes, 32 percent increased, and 34 percent decreased their cigarette consumption). On the other hand, more than 54 percent of the "less than one pack a day" smokers as compared to only 25 percent of the "one pack or more a day" smokers increased the number of cigarettes smoked. Nicotine dependency plays a minor role in determining the smoking habits of those who continue to smoke on a long-term basis. (Auth. Abs.)

80-0151. Herman, C. P.; Kozlowski, L. T. **Indulgence, Excess, and Restraint: Perspectives on Consummatory Behavior in Everyday Life.** *Journal of Drug Issues* 9(2): 185-196, Spring 1979.

A regulatory model of consumption is outlined, with an emphasis on upper and lower regulatory limits which govern consumption. It is argued that aversive physiological symptoms counteract failure to maintain consumption within regulatory limits, but that consumption within these limits is affected by various situational and cognitive factors. This model is applied to the consumption of food, alcohol, tobacco, and caffeine, with particular attention to the distinctive features of regulation that pertain to each. Nicotine is the drug responsible for the pharmacological attraction and addictive nature of the smoking habit. It appears that smokers attempt to achieve a blood-nicotine level that falls between the upper (dizziness, sweating, nausea, general ill health) and lower (characteristic withdrawal symptoms) regulatory boundaries. Implications are derived regarding the multiple meanings of "excessive" and "insufficient" consumption in the regulation framework. Also discussed are the relative (perceived) contributions of physiological and "personal" mechanisms of regulatory control and ensuing effects of self-perception and attribution. Concluding remarks concern the complexities of multiple substance use. (Auth. Abs. Mod.)

80-0152. Hunt, P. T. **A Study of Three Cognitive-Behavioral Indices of Self Control in Smoking Cessation.** Doctoral Dissertation, Columbia University, University Microfilms International, 79-24910, 1979, 107 pp.

Developments in reinforcement-based self-control theory and techniques provided rationale and a conceptual framework for organizing the observations of the population made in this study. This framework consisted of a closed-loop sequence of three behaviors: self-monitoring, self-evaluation, and self-reinforcement. Using an ex-post facto design, two groups (N=64) were defined as ex-smokers or recidivists. Group membership was assigned on the basis of (1) having been abstinent for a minimum of 6 months prior to participation in the study, or (2) being a current smoker who had attempted to quit in the past 3 years. The two groups were compared on a range of measures: demographic characteristics (age, sex, and schooling) and habit characteristics (duration, amount of consumption, and previous attempts to quit); personality trait scores bearing presumed relevance to the notion of self-control (ego-strength, superego strength, guilt proneness, and control); self-report measures which tapped: (1) self-monitoring of smoking and its outcomes, (2) self-evaluation in relation to one's smoking habit, and (3) self-reinforcement of smoking and cessation activity; and analogue tasks measuring predisposition towards: (1) accuracy of self-monitoring, (2) positive versus negative self-evaluation of performance, (3) self-reward, and (4) self-punishment. Groups did not differ on demographic, habit, or personality measures. Ex-smokers exhibited a predisposition towards more positive self-evaluation, more self-reward, and less self-punishment than recidivists. The groups also showed no differences on the analogue tasks of self-monitoring accuracy or on the self-report scales which tapped self-evaluation and self-reinforcement in smoking and cessation. The interpretation of the results which was most consistent with the theory of self-regulation suggested that self-evaluative and self-reinforcing behaviors contribute to an understanding of self-control insofar as they reflect an overall coping style rather than transitory phenomena associated with specific concerns. Results were also discussed as they related to general self-control issues and in terms of their implications for further research and practice. (Auth. Abs. Mod.)

80-0153. Kaur, H. **Drug Taking Among University Students.** *Nigerian Medical Journal* 8(2): 112-113, 1978.

A survey of students at the University of Nigeria was undertaken in 1973. Out of 700 questionnaires, 683 were returned completed. It was found that 38 students (5.4 percent) acknowledged having taken drugs and continued to do so. No one acknowledged having taken drugs and having subsequently stopped. Marihuana was the most commonly used drug, with glue and cocaine following. Regular use of alcohol was reported by 245 students (35.9 percent) while 135 (19.8 percent) were regular tobacco smokers. Drug abuse was reported by 13.3 percent of the smokers and 3.7 percent of the nonsmokers. There was an association between drug-taking and family background. (Auth. Abs. Mod.)

80-0154. Kozlowski, L. T. **The Role of Nicotine in the Maintained Use of Cigarettes.** Paper presented at the Technical Review on Cigarette Smoking as an Addiction, August 23-24, 1979, National Institute on Drug Abuse, Rockville, Maryland, 1979, 13 pp.

The key addictive ingredient in tobacco may be nicotine. Reasons for this belief and problems with testing it are discussed. Research on whether smokers smoke to regulate their intake of nicotine (the nicotine-titration hypothesis) are reviewed in the light of a new model of drug self-administration. According to this model, both psychosocial and pharmacological factors have powerful yet varying effects on drug taking; in some situations the psychosocial forces dominate, while in others the pharmacological are more important. Variations in nicotine dose are argued to have an important effect on smoking behavior when they occur in the neighborhood of either of two boundaries. The lower boundary of biologically determined withdrawal effects is challenged when the smoker is smoking as little as possible; the upper boundary of biologically determined toxic effects is challenged when the smoker is smoking as much as possible. At either boundary, changes in nicotine dose are more likely to have biological and behavioral consequences than they would if they took place within the region of relative indifference to the biological effects which is contained by these two boundaries. Within the range of relative indifference, psychosocial influences (e.g., self-control, the presence of others smoking) have, for the most part, free reign over smoking behavior. (Auth. Abs.)

80-0155. Mechanic, D. **The Stability of Health and Illness Behavior: Results From a 16-Year Follow-Up.** *American Journal of Public Health* 69(11): 1142-1145, November 1979.

In 1977, 302 young adults who were among 350 children first studied in 1961 responded to a questionnaire related to their health and illness behavior. Ten types of adult health and illness behaviors and orientations are examined including seat belt use, smoking, exercise, drinking, and risk taking. Smoking was related to more drinking, a poorer perception of physical health, and less preventive medical care. Smokers were also less likely to enjoy physical activity and to use seat belts. These patterns are associated only to a modest degree, and patterns of health and illness behavior appear to have low levels of continuity over a 16-year period (correlations varying from .02 to .20). (Auth. Abs. Mod.)

80-0156. Newman, A. **The Effect of Reinforcement of Intention Statements and/or Execution of Self-Control in Smokers and Ex-Smokers.** *Addictive Behaviors* 2(1): 15-20, 1977.

The present study investigated the differential effects of rewarding intention statements to execute self-control in ex-smokers, smokers who never attempted to quit, and smokers who attempted to quit but failed. Subjects signed a contract to engage in a cold pressor task and then executed the contract. Subjects were either rewarded after the contract execution, both contract and execution, or no reward was administered. Subjects were then given a second trial with no reinforcements. Ex-smokers displayed greatest tolerance of cold when external support was lacking. Smokers who tried to quit but failed, however, showed greatest tolerance when they received both contract and execution reinforcement. Finally, subjects who never tried to quit tolerated cold best when execution reinforcement was given and contract reinforcement was withheld. Clinical applications of these results are discussed. (Auth. Abs.)

80-0157. Schwebel, A. I.; Kaemmerer, W. F. **Smoking, Alienation, and Locus of Control Factors.** *Omega* 8(3): 239-246, 1977.

Several studies have reported conflicting results concerning the relationship between Rotter's Locus of Control Scale and smoking behavior. In order to clarify the relationship, college students' responses to Rotter's scale were factor analyzed, and smokers', ex-smokers', and nonsmokers' factor scores were compared. A predicted significant difference ($p<.005$) between smokers and nonsmokers was found on the politically unresponsive world factor, suggesting that the relationship between locus of control and smoking is centered in the concept of "alienation" rather than "will-power". Cross-validation data from a community sample were corroborative. It was reasoned that smokers are "alienated from their bodies" and thus have not internalized their intellectual knowledge that smoking endangers their bodies, and that they have preventative control over their own death. The findings suggest the usefulness of the concepts of "alienation" and "alienation from one's body" in understanding life-threatening behaviors. (Auth. Abs.)

80-0158. Wechsler, H.; Gottlieb, N. H.; Demone, H. W., Jr. **Lifestyle, Conditions of Life, and Health Care in Urban and Suburban Areas.** *Public Health Reports* 94(5): 477-482, September-October 1979.

The association among social and economic characteristics, use of the health care system, health habits, life problems, and reported health status were examined in a survey of 1,043 Greater Boston area residents. The respondents were classified according to their residence in the core city, the remainder of the city, the inner suburbs, or the outer suburban areas. For all social and economic variables studied, significant differences among the four subgroups increased with increasing distance from the inner city, where respondents were most likely to be of lower socioeconomic status. No statistically significant differences were found with regard to use of health services, although the places where medical care was obtained differed. In terms of health habits, the findings suggest that outer suburban respondents exhibit somewhat healthier lifestyles, particularly with respect to smoking cigarettes and exercising. The difference in cigarette smoking was significant among the groups ($p<.01$). The proportion of respondents who reported that they were cigarette smokers was highest within the city (close to half of both core city and other Boston respondents) and lowest in the outer suburbs group (less than one-third). Respondents in the core city reported significantly more problems encountered, the greatest concern with their neighborhoods, and the least satisfaction with their neighborhoods. They were also most likely to report poorer health. (Auth. Abs. Mod.)

80-0159. Whichelow, M. J.; King, B. E. **Breast Feeding and Smoking. (Letter).** *Archives of Disease in Childhood* 54(3): 240-241, March 1979.

The prevalence of breast feeding at delivery and 3 months postpartum and the relationship of smoking to the choice of bottle vs. breast feeding was studied in 330 women in Cambridge, England. Of the 95 mothers who were classified as belonging to the manual class, 27 percent were smokers, whereas only 13 percent of the nonmanual group smoked. Breast feeding declined from 82 percent at delivery to 46 percent 3 months later. Smokers were significantly less likely to start breast feeding than nonsmokers ($p<0.01$) and more likely to have changed to bottle feeding by 3 months ($p<0.05$ in the nonmanual class). Comparison of bottle vs. breast feeders at 3 months showed more smokers who were bottle feeders ($p<0.001$, overall; $p<0.05$, nonmanual class; $p<0.05$, manual class)

80-0160. Wills, D. **A Study of Smoking Habits and Attitudes in Relation to the Nursing Profession.** *New Zealand Nursing Forum* 6(2): 8, December 1978-January 1979.

A questionnaire concerning smoking habits and attitudes toward smoking control measures was mailed to a random sample of members of the Nurses Society of New Zealand. Only 27 percent of the nurses were cigarette smokers. The great majority of nurses (96 percent) acknowledged that smoking is a health hazard and most also considered that nurses who smoke set a bad example as health professionals for the lay public. Further, most nurses (90 percent) supported increased measures such as health education, pricing policies, controls on advertising, controls on smoking in public places, and controls on smoking in hospitals, to discourage smoking. (Auth. Abs. Mod.)

80-0161. Worden, J. K.; Sweeney, R. R.; Waller, J. A. **Audience Interest in Mass Media Messages About Lung Disease in Vermont.** *American Journal of Public Health* 68(4): 378-382, April 1978.

This study pretested audience interest in 25 potential message concepts to be used in a mass media campaign designed to change knowledge, attitudes, and behavior regarding smoking and lung disease. A group of 150 respondents reflecting specific target audiences (smokers, older persons) rated each concept on the basis of a two-sentence description using Haskins' 0-100 scale. Results indicated that older persons were most interested in message concepts suggesting ways to deal with various lung disease symptoms, and smokers showed highest interest in concepts offering positive and straightforward advice on how to quit smoking, rather than concepts that were negative, cute, or satirical in approach. Recommendations based on audience interest were made for the design of future lung disease media campaigns. (Auth. Abs.)

80–0162. Wright, G. R.; Shephard, R. J. **Carbon Monoxide Exposure and Auditory Duration Discrimination.** *Archives of Environmental Health* 33(5): 226-235, September-October 1978.

Two young adults performed an auditory duration discrimination task while sitting in an open office (i.e., without sensory isolation). The percentage of errors rose when carboxyhemoglobin (HbCO) readings of 3.2 percent or 4.7 percent were produced by a rebreathing method; there was a parallel but statistically insignificant trend at 2.0 percent HbCO. Analysis of the data by psychophysical measures, however, showed no significant changes of difference threshold, criterion value, or point of subjective equality. After the tape was rearranged to meet criteria of a constant interval, a balanced order of presentation, and psychological rather than physical equality of tone length, eight young adults performed the task better in an isolation booth than in an open office, scores in both situations being unaffected by increases of HbCO to >8 percent. Further trials with five young adults compared responses before and after administration of 80 ml of CO (HbCO 4.92 percent); again, no effect of carbon monoxide was seen relative to control experiments. The error score of nonsmoking young adults can be increased by a level of HbCO as low as 3.2 percent. The likelihood of such a response, however, is markedly influenced by task difficulty, environment, and the arousal of the subjects. Furthermore, the nature of the functional disturbance remains uncertain; psychophysical measures show no significant impairment of auditory duration discrimination. (Auth. Abs.)

See also, **80–0043, 80–0064, 80–0136, 80–0165, 80–0169**

SMOKING CESSATION METHODS

80–0163. Baer, P. E.; Foreyt, J. P.; Wright, S. **Self-Directed Termination of Excessive Cigarette Use Among Untreated Smokers.** *Journal of Behavior Therapy and Experimental Psychiatry* 8(1): 71-74, 1977.

Fifty-one letters from former long-term severely dependent cigarette smokers were analyzed for factors and procedures they thought to have been effective for termination of smoking. All respondents had stopped smoking without professional help. Most respondents used multiple techniques such as: challenge from others, fear, preventive measure, or motivation. The probability of techniques occurring together was examined for the 19 subjects reporting two techniques. Only the fourfold occurrence of the categories, challenge to self, and motivation approached statistical significance ($p = 0.053$). However, this pattern of findings did not appear to support pairing of techniques at a level much above chance. (Auth. Abs. Mod.)

80–0164. Brockway, B. S.; Kleinmann, G.; Edleson, J.; Gruenewald, K. **Non-Aversive Procedures and Their Effect on Cigarette Smoking.** *Addictive Behaviors* 2(2/3): 121-128, 1977.

The majority of smoking reduction studies have focused on the use of aversive control procedures to reduce or eliminate smoking behavior. Relatively few studies have analyzed the effect of positive reinforcement despite evidence which suggests that positive retraining may be more effective over the long run than aversive controls in reducing cigarette smoking. This study examines the long-term effect of nonaversive procedures to reduce smoking behavior and describes a low-cost, reliable method for validating self-report data. Twenty-seven smokers were randomly assigned to experimental or control groups. Experimental subjects were divided into two groups which met for six consecutive weekly sessions and two biweekly sessions. Followup data was taken at 3, 6, and 12 months after treatment. A series of nonaversive stimulus control and positive reinforcement procedures comprised the treatment package. Included were alternate response training, gradual smoking reduction via individualized situational hierarchies, behavior rehearsal of verbal no-smoking requests, contingency contracting and in vivo practice of nonsmoking in high anxiety situations. Significant differences in smoking reduction rates were noted at the 3 and 6 month followups but not after 12 months. Tests measuring serum thiocyanate levels in the blood revealed discrepancies in self-reported data. (Auth. Abs.)

80–0165. Christen, A. G.; Cooper, K. H. **Strategic Withdrawal From Cigarette Smoking.** *CA; Cancer Journal for Clinicians* 29(2): 96-107, March-April 1979.

Cigarette smoking can be viewed mainly as a psychological habit with accompanying pharmacological and social satisfactions and reinforcements. It is mainly a learned behavior, occurring in response to direct and indirect social pressures. Though each person's smoking habit is individualized, studies show that smokers find at least six psychological satisfactions in the habit: stimulation, handling, relaxation, tension reduction, craving, and habit. To quit smoking, the individual must undergo physical and psychological withdrawal. Physical withdrawal symptoms last about a week, but it may take months or years to restructure a life without cigarettes. Quitting is a long-term learning process involving a great deal of unconscious, constructive, and painful conflict. Social pressure, lack of a plan, alcohol consumption, expectation of failure, stress situations, alternative forms of tobacco, weight gain, and nicotine dependence are some of the potential obstacles to quitting. One can stop smoking if suitable satisfying alternatives and involvements are discovered. Physical exercise in the form of aerobics is proposed as an especially effective alternative behavior. (Auth. Abs. Mod.)

80–0166. Flaxman, J. **Quitting Smoking Now or Later: Gradual, Abrupt, Immediate, and Delayed Quitting.** *Behavior Therapy* 9: 260-270, 1978.

This study evaluated gradual versus abrupt smoking cessation techniques, the value of delaying the smoking quit date, and the use of aversive conditioning by means of warm, smoky air and rapid smoking compared to an attention control treatment. Sixty-four subjects were recruited from the community. The smoking rate of subjects was 26 cigarettes per day, mean years of smoking was 20.3, and past abstinence from smoking averaged 5 months. Subjects were assigned to immediate, target date, gradual, and partially gradual treatment groups prior to the quit date and to either an aversive conditioning or attention control treatment group after quitting. Target date quitting was significantly more effective than gradual quitting ($p<.05$). Delaying the quit date was more effective than quitting immediately for women, but less so for men ($p<.01$). Aversive conditioning was markedly more effective only in the partially gradual group ($p<.01$). In both the gradual quit and the immediate quit groups aversive conditioning helped men and hindered women in their attempts to stop smoking ($p<.01$, $p<.10$, respectively).

80–0167. Harrup, T.; Hansen, B. A.; Soghikian, K. **Clinical Methods in Smoking Cessation: Description and Evaluation of a Stop Smoking Clinic.** *American Journal of Public Health* 69(12): 1226-1231, December 1979.

This study reports the results of the Kaiser-Permanente Stop Smoking Clinic and describes the philosophy and methods employed by the clinic in treating addictive smoking behavior. The clinic is an educational program that uses group counseling provided by the group leader and a group-support counseling component. The counselors are all ex-smokers. The clinic has two major goals: cessation of cigarette smoking, and cessation of the desire to smoke. The clinic presently covers an 8-week period with 13 90-minute meetings. Group reunions are held at 3 months, 6 months, and 12 months after quitting. Of the 1,128 clients (43 percent male, 57 percent female) who registered for the group program, 57 percent were abstinent 6 months after quitting smoking and 47 percent were abstinent at 1 year. Men were only slightly more successful than women in quitting (49 vs. 46 percent, respectively). The clinic methods used are described in detail. They attempt to relate smoking behavior to the larger phenomenon of addiction. (Auth. Abs. Mod.)

80–0168. Lamontagne, Y.; Gagnon, M.-A.; Trudel, G.; Boisvert, J.-M. **Thought-Stopping as a Treatment for Reducing Cigarette Smoking.** *International Journal of the Addictions* 13(2): 297-305, 1978.

Three treatments, thought-stopping, group discussion, and the wearing of a badge saying "I don't smoke," each combined with self-monitoring, were compared to one another and to self-monitoring alone for their ability to modify the smoking behavior of 60 male volunteers who smoked an average of 27.3 cigarettes per day and whose mean addiction duration was 17.9 years. All procedures led to an important reduction of the smoking rate at the beginning of treatment, the reduction being significantly greater with thought-stopping. During the first two weeks of treatment, the smoking rate decreased almost 50 percent for the self-monitoring and the badge-wearing groups, 59 percent for the discussion group, and more than 70 percent for the thought-stopping group. The latter remained the most successful treatment during the 6-month followup, although it did not differ significantly from self-monitoring alone. (Auth. Abs. Mod.)

80–0169. Liberman, R. P. **Discussant for Section on Behavioral Change. Behavioral Change: A Stopped Smoker Is Not a Non-Smoker.** In: Jarvik, M. E.; Cullen, J. W.; Gritz, E. R.; Vogt, T. M.; West, L. J. (Editors). *Research on Smoking Behavior.* National Institute on Drug Abuse Research, Monograph Series 17, December 1977, pp. 373-381.

One approach to designing treatments with more durable outcomes is to analyze the experiences of smokers who have quit and relapsed, and to compare their process of relapse with the successful experiences of smokers who quit and remain abstinent. A behavioral-empirical analysis of the relapse phenomenon is recommended. There are a host of possible relapse factors to be considered, including the return of the temporarily abstinent ex-smoker to a family and/or work situation where others still smoke (modeling effects), and the gradually more sparse receipt of social reinforcement from significant others as time passes from the point of withdrawal and people take the ex-smoker's abstinence for granted. In a recent study of 48 individuals who attempted to stop smoking on their own, a comparison was made between 24 people who were successful in quitting cigarettes and remained abstinent for at least 4 months, and 24 others who were unsuccessful in staying off cigarettes. The evaluation utilized an interview whose focus was the subjects' experiences during the process of quitting cigarettes. The successful ex-smokers could be distinguished from those who failed by their (1) persisting longer in their efforts at quitting, (2) using a greater variety of techniques and employing them more frequently in their daily life, (3) obtaining more social reinforcement for their efforts at abstinence from significant others, and (4) giving themselves more positive self-reinforcement for efforts at abstaining. The results of this interview study and others underscore the importance of duration of treatment efforts, multiple techniques, self-control methods, and social support or reinforcement from one's social world in maintaining abstinence. Treatment efforts should target both a decrease and cessation in smoking behaviors and an increase or strengthening of behaviors that can be linked to abstinence.

80-0170. McGrath, M. J.; Hall, S. M. **Self-Management Treatment of Smoking Behavior.** *Addictive Behaviors* 1(4): 287-292, 1976.

Twenty-nine habitual smokers were randomly assigned to one of three treatments: (1) self-management treatment (SMT), (2) self-monitoring plus social reinforcement (SMSR), and (3) no-treatment control (NT). An analysis of covariance of mean smoking frequency, with pretreatment smoking frequency as the covariate, revealed that at treatment termination (7 weeks) SMT and SMSR means were significantly lower than the NT mean ($p<0.01$) and there was no significant difference between the former two groups ($p<0.05$). At followup, 80 days after treatment termination, the SMT mean was significantly lower than either of the other two group means and there was no significant difference between these latter two groups. Further analyses indicated that both SMT and SMSR groups had significantly reduced means ($p<0.005$) at both treatment termination and followup, relative to pretreatment. Further, neither SMT nor SMSR groups revealed significant relapses over the followup interim. Self-report data revealed significant relapses over the followup interim. Self-report data revealed a substantial (0.77) and significant partial correlation between consistency of use of SMT techniques and treatment gains. Results were interpreted as suggesting the efficacy of a "comprehensive" self-management treatment package and the potential importance of extended treatment durations. (Auth. Abs.)

80-0171. Rogers, R. W.; Deckner, C. W.; Mewborn, C. R. **An Expectancy-Value Theory Approach to the Long-Term Modification of Smoking Behavior.** *Journal of Clinical Psychology* 34(2): 562-566, April 1978.

Despite the research interest in modifying smoking behavior, therapeutic treatments that can produce long-term cessation have not been demonstrated rigorously. A followup study of two attitude change experiments ($N = 173$) examined the effects of a fear appeal, that is, increasing smokers' awareness and appreciation of the highly noxious consequences of smoking. Although this familiar type of information may be an integral component of many smoking treatment programs, its long-term suppressive effect has not been demonstrated in well-controlled experiments. High-fear manipulation significantly increased the percentage of smokers who were able to stop smoking completely at 3 months (22 percent) and 1 year (18 percent) after treatment. Low-fear manipulation resulted in abstinence in 7 percent of the smokers at 3 months, but, at the 1-year followup none of the smokers were abstinent. (Auth. Abs. Mod.)

80-0172. Vogt, T. M.; Selvin, S.; Billings, J. H. **Smoking Cessation Program: Baseline Carbon Monoxide and Serum Thiocyanate Levels as Predictors of Outcome.** *American Journal of Public Health* 69(11): 1156-1159, November 1979.

One hundred and sixty-two smokers who participated in an aggressive smoking cessation program underwent analysis of expired air carbon monoxide (CO) and serum thiocyanate (SCN) at entry and 1 year later. Persons who failed to quit smoking had higher baseline CO and SCN and smoked more cigarettes per day than did those who succeeded in quitting. The use of CO, SCN, number of cigarettes smoked, age, and blood pressure at baseline permitted accurate classification of over 70 percent of the subjects into groups which would succeed and fail in their effort to quit smoking. Persons who failed to quit reduced the reported number of cigarettes smoked per day by one-third, but there was no corresponding reduction in CO and SCN, suggesting that self-reporting of a reduction in the number of cigarettes smoked may not lead to a corresponding decline in exposure. (Auth. Abs.)

80-0173. Whieldon, D. **How You Can Help Patients Stop Smoking.** *Behavioral Medicine* 5(3): 12-15, March 1978.

This study suggests a number of ways in which physicians can help their patients to quit smoking: (1) physicians can act as models of healthy living by not smoking themselves; (2) they can explain the risks of smoking to their patients; (3) they can refer patients to smoking withdrawal consultants or organizations; and (4) physicians can themselves get involved in the management of smoking cessation efforts. The following guidelines are suggested for the physician: (1) a firm agreement should be made with the patient about procedures, office visits, and a cessation date; (2) the patient should clearly understand his reasons for wanting to stop smoking; (3) the patient should record the number of cigarettes he smokes for about 1 or 2 weeks; (4) the Smoker's Self-Testing Kit developed by the Department of Health, Education, and Welfare should be utilized; (5) the patient should be urged to substitute other activities for smoking, such as chewing gum, reading, or exercising; and (6) after the patient is ready to stop completely, he should be seen again or telephoned as a followup procedure. Experience has shown that intervention by physicians can be both economical and relatively effective in cessation efforts.

TOBACCO MANUFACTURING AND PROCESSING

80–0174. Halter, H. M.; Ito, T. I. **Effect of Tobacco Reconstitution and Expansion Processes on Smoke Composition.** In: *Recent Advances in Tobacco Science* , Series 4, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1978, pp. 113-131.

This paper reviews the published literature on tobacco reconstitution and expansion processes, with emphasis on the relationship between physical structure and smoke composition. Using a single reconstitution process, the tobacco slurry process, it is possible to alter cigarette physical and combustion relationships by varying sheet structure and composition. Data is presented for a foamed low-density sheet indicating substantial reductions in tar constituents compared to unfoamed sheet or natural tobacco. A study of commercial sheet processes illustrates a broad range of smoke compositions resulting from different structures, additives, and processing conditions. A comparison of paper process and slurry process sheets made without additives showed the slurry process sheet was close in smoke chemistry to that of the starting tobacco, while the paper process was reduced in tar, nicotine, and other smoke components. The paper process results are likely related to substantial changes in tobacco chemistry during processing. Tobacco expansion processes reviewed include comparative data for two tobacco puffing processes and freeze drying. The tobacco and smoke chemistry of the two puffed samples were more dissimilar than similar. The freeze-dried sample had the lowest smoke phenols, but was otherwise quite similar to the puffed sample in smoke chemistry. The potential for smoke modification via reconstitution and expansion processes is apparent. (Auth. Abs.)

80–0175. Mattina, C. F.; Selke, W. A. **Influence of the Paper on the Smoke of Cigarettes.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 533-552.

Diffusion, dilution, carbon monoxide delivery, additives, and coal resistance are among the factors considered in this discussion of the effect of permeability and burn additives on cigarette smoke yield and composition. Cigarettes made with untreated paper burned slower than those treated with phosphate which in turn burned slower than those treated with citrate. Cigarettes made with papers that were treated with high levels of chemicals such as MAP or aluminum chloride delivered levels of carbon monoxide (CO) disproportionately higher than predicted levels. Diffusion measurements indicated that 20 to 40 percent of the CO generated by burning tobacco could diffuse out of the cigarette during a puff, depending on the paper permeability. Coal resistance, a function of the burn additive in the paper, is partially responsible for this phenomenon.

See also, **80–0033**

TOBACCO ECONOMICS

80–0176. Foreign Agriculture. **U. K. Data Show Drop in Tobacco Consumption.** *Foreign Agriculture* 16(25): 11, June 19, 1978.

Tobacco consumption in the United Kingdom fell in 1977, compared with 1976 and earlier years. Total manufactured weight of all tobacco products sold for consumption in the United Kingdom was 104,327 tons, 3,719 tons below the 1976 level, and lower than any previous year, at least since the early 1950's. Total cigarette consumption in 1977 declined by 4,700 million pieces from 1976 levels to 125,900 million pieces. Nonfilter cigarette consumption dropped more sharply (down 3,050 million) than did filter cigarette consumption (down 1,650 million). The cigarette sector is under increasing pressure from the smoking and health lobby and the Government. Most of the brands are in the middle-to-high tar range, which the Government intends to tax at a supplementary rate. Elderly nonfilter cigarette smoker attrition will further diminish the market. Cigarillo and cigar consumption also declined in 1977 by 25 million and 10 million pieces, respectively.

80–0177. Howland, K. E. **U.K. Smokers Spurn Tobacco Substitutes.** *Foreign Agriculture* 15(49): 6, December 5, 1977.

On July 1, 1977, cigarette manufacturers in Great Britain launched 12 new brands of king-size cigarettes containing up to 25 percent tobacco substitute material. In spite of an expensive advertising campaign, the new cigarettes fell far short of achieving anticipated sales levels. Only 1 in 10 British smokers chose the new brands in the early weeks following their introduction, and by the end of October, the market share for the new brands had fallen to just over 1 percent. Among the reasons given for these disappointing sales are: (1)

the massive advertising campaign which raised too many expectations; (2) the refusal of the British Government to permit advertising of substitute-containing cigarettes as being safer than conventional cigarettes; and (3) equal taxation for the new brands and for regular cigarettes.

80-0178. Krawczyk, L. **Turkey: Land of Potential and Problems.** *Foreign Agriculture* 15(49): 10-12, 14, December 5, 1977.

Agricultural exports in Turkey, chiefly cotton, tobacco, filberts, and wheat have been running about $1.0 to $1.3 billion a year. Tobacco is likely to replace cotton as the primary agricultural export this year. The Turkish Tobacco Monopoly, a government agency, is the sole importer and manufacturer of tobacco products in Turkey. Turkey has always pursued a very restrictive policy with regard to imports of leaf tobacco and tobacco production. Official imports are extremely small. Contraband cigarette imports flourish, but still make up a very small percentage of Turkey's total consumption. Turkey's current problems in tobacco are due to massive overproduction in 1976, and until recently, official minimum export prices that were unrealistically high. The Government of Turkey is now reducing tobacco export prices (through various export subsidy schemes) to clear surplus 1975 and 1976 crop stocks. But the consequences of surplus production in recent years will probably be felt through 1979.

80-0179. U. S. Department of Agriculture. **World Tobacco Production Up 2.6 Percent; Stock Increase Expected in 1978.** *Foreign Agriculture Circular* 2:1-19, January 1979.

An 8 percent increase in world oriental leaf production paced the total world tobacco crop in 1978 to 5.59 million tons. (Based on the past 5 years this figure will not need to be revised by more than 2 percent.) All types except fire-cured and dark air-cured cigar tobacco contributed to the 2.6 percent boost. The 1978 crop fell only 1 percent short of the record 1976 crop of 5.66 million tons. World flue-cured output rose 2.2 percent to 2.3 million tons. Among major flue-cured producers, Canada, the United States, Brazil, and India had larger crops. The quality of the United States flue-cured crop harvested in 1978 is described as the best in 4 years and will help relieve the tight supply situation for high-quality flue-cured. Global burley output reached a record 600,674 tons during 1978, up 3 percent from the 1977 level and 17 percent above the 1972-76 average. Spain's burley production rose by 55 percent. The 2.6 percent increase in the farm-sales-weight production of leaf tobacco will yield an approximate increase of 125,000 tons in the 1978 supply of unmanufactured tobacco. Consumption is expected to expand by less than 50,000 tons, thus stocks will probably rise by 75,000 tons. The somewhat higher stock level will not likely influence prices for the 1979 crop. (Auth. Abs.)

BILLS AND LEGISLATION

80-0180. Greene, G. E. **Nonsmokers' Rights. A Public Health Issue.** *Journal of the American Medical Association* 239(20): 2125-2127, May 19, 1978.

The Howard County Indoor Clean Air Act of 1977, requiring public places in Howard County, Maryland, to provide separate areas for smokers and nonsmokers, or, if desired, to prohibit smoking altogether, is an example of numerous laws to restrict smoking in public places. Medical and lay testimony was presented before the Howard County Council. Medical testimony covered research findings and clinical experience related to the health effects of sidestream smoke for nonsmokers; lay testimony came from nonsmokers who described their reactions to being exposed to tobacco smoke. Four groups of persons were identified whose health would be adversely affected by sidestream smoke: (1) allergic and asthmatic persons, (2) infants and children, (3) persons with chronic heart or lung disease, and (4) contact lens wearers.

GENERAL

80-0181. Abraham, J. L. **"Safe" Cigarettes and Fortified Alcohol. (Letter).** *New England Journal of Medicine* 299(20): 1137, November 16, 1978.

Two recent studies, one reporting development of a "safer" cigarette (*Newsweek* 42: 25-28, August 21, 1978) and the other presenting a cost-benefit analysis of the prevention of a severe neurologic syndrome by addition of vitamin B to alcoholic beverages, received quite different responses. While the cigarette study was criticized for discussing safety in connection with cigarettes, the alcoholic beverage report was acclaimed for its value in preventive medicine. The respondent comments on the inconsistency in the response and points out that as long as both these harmful habits are supported by strong political forces, objective scientific studies may not lead to the most cost-effective or healthful solutions.

80-0182. Ayers, R. **"You've Come a Long Way, Baby!" (Letter).** *Cancer Nursing* 1(1): 13, February 1978.

An ex-smoker colleague relates that the percentage of women smokers in the nursing profession has risen from 37.7 in 1969 to 38.9 in 1975, despite the amount of available literature on smoking and lung cancer. In 1975, for the first time in Connecticut history, there was more lung cancer among women than men aged 35 to 44. Quitting to benefit health and to enable nurses to practice prevention is encouraged. Good health care (e.g. sputum cytology and X-ray examinations) is recommended if quitting is not possible.

80-0183. Ball, K.; Stevenson, A. **Hospital Action on Smoking.** *British Medical Journal* 2(6193): 777-778, September 29, 1979.

The Central Middlesex Hospital in London has established a new policy restricting smoking on its premises. Measures designed to enact this policy include: (1) the restriction of smoking in all acute wards and public areas of the hospital; (2) the creation of special smoking areas; and (3) a "smoking action week" to introduce staff and patients to the new hospital policy. Complaints about the restrictions were found to be few, as most people, including both smokers and nonsmokers, favor a ban on smoking in hospital wards.

80-0184. Cohen, S. B. **Should Medical Associations Discourage Smoking?** *Journal of the American Medical Association* 239(20): 2158, May 19, 1978.

Medical associations are urged to set an example in the prevention and elimination of smoking. Smoking has been viewed by the medical community as an unimportant issue not of concern to busy men trying to save lives. However, by reducing the incidence of chronic smoking-related diseases like emphysema and hypertension, physicians might be able to reduce a demand for their services, which would translate into a relative increase in medical manpower. Since the image of physicians is a prime factor in the behavior of patients, members of the medical profession should not permit smoking at association meetings, in medical institutions, and in their own offices.

80-0185. Falkson, J. L. **The "New" Politics of Health.** *Journal of Health Politics, Policy and Law* 2(4): 607-611, Winter 1978.

From clean air standards to proposed controls on carcinogens in drinking water, the Federal government has expressed many concerns about occupational, environmental, and public health. The Secretary of the Department of Health, Education, and Welfare launched an antismoking campaign to be targeted at education, regulation, incentives and research. Among his proposals were a general increase in the Federal excise tax on cigarettes with a graded increase according to nicotine levels, and a suggestion that insurance companies might consider offering health, life, fire, or other policies at an advantage to nonsmokers via lowered or special premium rates. The politics of health appear to be part of a new trend toward preventive public health care.

80-0186. Humphries, B. **DDT Residue Levels Show Sharp Decline.** *Research and Farming* 36(1/2): 12-13, Summer-Autumn 1977.

Average DDT residues in flue-cured tobacco offered for sale by Tar Heel farmers have dropped from 53 parts per million in 1968, to just above one-tenth of a part per million in 1976, according to studies by the N.C. Agricultural Experiment Station. This is a decline of 99.8 percent in total DDT residues on tobacco. Present DDT residue levels on flue-cured leaf are now acceptable in both domestic and foreign markets.

80-0187. Kaufman, J. D.; Lumnitz, J. S.; Jonas, S.; Wynder, E. L.; Hoffmann, D. **Stopping Smoking. (Letters).** *New England Journal of Medicine* 301(7): 389-390, August 16, 1979.

Various approaches to antismoking programs for adults have been considered (*New England Journal of Medicine* 300(16): 894-903, April 19, 1979). In addition to those described, a suggestion was made that aerobic exercise programs be added to the list, as they have had high success rates in motivating smokers to quit by substituting running for smoking. A different approach, the use of a heavy tax on cigarettes to pay for the health problems associated with smoking, was proposed by another respondent, as the average smoker is often unwilling to quit since the dangers associated with smoking are neither swift nor certain. Other measures suggested for inclusion in antismoking programs were: (1) a ban on all cigarette advertising; (2) ending subsidies for growing tobacco; and (3) limiting the availability of cigarettes. In response, the authors of the original article agreed that aerobic exercise was a preventive behavior approach which does tend to have a "ripple" effect. They questioned whether a ban on cigarette advertising would really be effective in reducing smoking. In regard to motivation to quit, the importance of the physician presenting an example as a nonsmoker and practicing primary disease prevention is stressed. See also 79-1061.

80-0188. Mitchell, R. S.; Judson, F. N.; Moulding, T. S.; Weiser, P.; Brock, L. L.; Kelble, D. L.; Pollard, J. **Health Effects of Urban Air Pollution. Special Consideration of Areas at 1,500 m and Above.** *Journal of the American Medical Association* 242(11): 1163-1168, September 14, 1979.

Sources of common air pollutants and their health effects are summarized. Epidemiologic studies of the effects of common air pollutants are reviewed. State and National Ambient Air Quality standards are discussed. Pollution—carbon monoxide and oxidants in particular—is capable of aggravating preexisting chronic heart and lung diseases. Pollution and tobacco (especially cigarette) smoke act synergistically in a causative role in chronic bronchitis and in an aggravating role in emphysema. Susceptibility to the effects of urban air pollution varies widely, with the most susceptible being fetuses, the newborn, the elderly, the infirm, those with chronic heart and lung diseases, and those who smoke. (Auth. Abs. Mod.)

80–0189. Sapolsky, H. M. **The Political Obstacles to the Control of Cigarette Smoking in the United States.** Presented at the M.I.T./C.N.R.S. Conference on Science and Decision, Paris, France, February 1-3, 1979, M.I.T. Document 05A, December 1978, 25 pp.

The opportunity to affect significantly the consumption of cigarettes in the United States through government action appears quite limited. Fifty million Americans smoke cigarettes. The United States is a leading producer of tobacco leaf and utilizes a price support system which is designed to protect tobacco growers. The industry is profitable and politically well connected. Several states are important producers of tobacco while others benefit from the excise tax imposed on cigarettes. The opposition to smoking is relatively weak. Action on Smoking and Health (ASH), Group Against Smokers' Pollution (GASP), and religious groups compete for the same limited financial support, and are internally divided. (Auth. Abs. Mod.)

AGRICULTURE

80–0190. Alam, M.; Babar, S. A.; Said, M. **Effect of Nitrogen on the Quality Controlling Factors of Flue-Cured Virginia Tobacco Leaf.** *Pak Tobacco* 1(1): 24-25, January 1977.

Sugars, nicotine, chlorides, and protein are the important quality determining elements in flue-cured tobacco. A field experiment was conducted at the Research Station, Kalpani, Mardan, to study the effect of different doses of nitrogen in combination with a basal dose of phosphorus and potassium at the rate of 60 lbs each per acre on the leaf quality of variety Coker 254. Average chloride content and percentage of nitrogen increased with increased dosage of nitrogen. Decrease in reducing sugar occurred with the increase of nitrogen, while potash content of the leaves was not affected. Nicotine and phosphorus contents were so irregularly distributed among the treatments that no significant treatment relationship could be stated. (Auth. Abs.)

80–0191. Brod, G.; Fischbach, E.-D. **Ruckstandsprobleme bei der Blauschimmel- und Blattlausbekampfung im Tabakbau. [Residue Problems in the Control of Blue Mold and Aphids in Tobacco Cultivation.]** *Der Deutsche Tabakbau* 58(14): 174-176, July 1978, German.

Tobacco plants in experimental lots in eight different West German locations were sprayed with 0.05 and 0.10 percent solutions of antracol (dithiocarbamate fungicide) or 0.1 percent solution of metasytox R (oxydemeton methyl, phosphorothioic dimethyl ester) to control blue mold or aphids, respectively. A total of 120 kg of tobacco leaves were harvested from each experimental lot over a 3-year period (1974-76) and were analyzed for fungicide and insecticide residues and their degradation products following roof-top drying, conditioning, or fermentation. Average dithiocarbamate (CS_2) residue levels in roof-top dried tobacco are tabulated for each year's crop. Typically, the residue level in 1974 increased by 226 percent when the antracol dose was increased from 2.4 to 4.0 kg/ha. Temperature was found to be even more important than dosage for the level of residues. In 1974, when there were only 7 hot days (30°C or higher), the average dithiocarbamate residue concentration was 21.88 mg CS_2/kg and blue mold disease increased. In 1975 and 1976, with 15 and 23 hot days, residue levels decreased to 5.33 and 2.22 mg CS_2/kg, respectively, and blue mold disease was absent. In 1974, the tolerance levels of 50 mg/kg proposed by the German tobacco industry was exceeded in one third of the experimental lots. No detectable residues were found in tobacco samples dried for 3 to 4 months, when metasystox R alone or mixed with antracol, parathion, and mevinphos were used to control aphids. The chlorinated hydrocarbon content of the tobacco samples was well below the legal limit or even negligible.

80–0192. Bush, L. P.; Saunders, J. L. **Accumulation, Manipulation and Regulation of Nicotine Content in Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 389-425.

The control of the nicotine content in commercial tobacco is reviewed. An evaluation is made of those environmental factors (e.g., water supply, fertilization, topping, and suckering) that influence nicotine content and can be controlled during tobacco production. Physiological factors of nicotine and alkaloid production in tobacco such as germination, site of alkaloid synthesis, and amino acid incorporation are also discussed. The review focuses on the regulation of nicotine levels in tobacco through an examination of genetic control, the effects of growth regulation and modification, and metabolic regulation.

80–0193. Carey, A. E.; Gowen, J. A.; Tai, H.; Mitchell, W. G.; Wiersma, G. B. **Pesticide Residue Levels in Soils and Crops, 1971—National Soils Monitoring Program (III).** *Pesticides Monitoring Journal* 12(3): 117-136, December 1978.

Data from the 1971 National Soils Monitoring Program are summarized. Composite samples of soil and mature crops (including tobacco) were scheduled for collection from 1,533 4-hectare sites in 37 States. Analyses were performed on 1,486 soil samples for organochlorines, organophosphates, polychlorinated biphenyls (PCBs), and elemental arsenic. Organochlorine pesticides were detected in 45 percent of the soil samples in the following order of frequency: dieldrin, ΣDDT, aldrin, chlordane, and heptachlor epoxide. Most pesticide levels ranged from 0.01 to 0.25 ppm. Crop samples were collected from 729 sites, and all were analyzed for organochlorines. Crop samples were analyzed for organophosphates and atrazine only when pesticide application data indicated current-year use. Organochlorines were detected in 42 percent of the crop samples analyzed, organophosphates in 13 percent, and atrazine in 1 percent. (Auth. Abs.)

80–0194. Chaplin, J. F. **Breeding for Varying Levels of Nicotine in Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 328-339.

Two systems of genetic control are involved in the quality and level of total alkaloids in tobacco (*Nicotiana tabacum* L.). A single locus controls the conversion of nicotine to nornicotine and two loci control total alkaloid levels. These levels may be further modified by minor genes or quantitative factors. Breeding lines and cultivars of flue-cured tobacco have been developed with varying levels of total alkaloids (nicotine). Certain agronomic characteristics, chemical and smoke constituents associated with total alkaloids, have been determined. Low nicotine lines had reduced yields, a lower quality index, less total nitrogen, and the plants ripened slower than normal nicotine lines. Taste and aroma of the smoke were not appreciably affected by nicotine levels. In a separate interspecific study, genetic factors that control nicotine production in *N. rustica* L. were transferred to *N. tabacum* , and breeding lines with four nicotine levels were obtained. (Auth. Abs.)

80–0195. Chouteau, J. **Incidence de la Teneur en Chlore de l'Eau d'Arrosage sur Certaines Caracteristiques Physico-Chimiques des Tabacs. [Incidence of Chlorine Content of Sprinkling Irrigation on Physical and Chemical Characteristics of Tobaccos.]** *Annales du Tabac* 13(2): 103-108, 1976, French.

As part of a study on French tobacco combustibility, experiments have been carried out on different farms so as to show the influence of chlorine content of sprinkling irrigation on the physical and chemical characteristics of tobacco crops. The chlorine content varied from 10 to 150 mg/l, and the volume of water ranged from 200 to 750 m³/hectare, according to the weather conditions of the year and the farms involved. It was discovered that about 50 percent of the chlorine supplied by water was found in the tobacco leaves. Considering the ill effects of this element on the quality of tobacco and particularly on its combustibility, it seems advisable to dissuade growers from sprinkling tobaccos with waters whose contents in chlorine are greater than 50 mg/l. (Auth. Abs.)

80–0196. Collins, G. B.; Legg, P. D. **Genetic Control of Chemical Constituents in Tobacco.** In: *Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.* Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, pp. 290-327.

This review briefly discusses genetic studies of chemical constituents in tobacco. Success in breeding for alkaloid content, the finding of genetic variation for other components (nicotine, nornicotine, sterols, phenols, proteins, nitrates, and other nitrogenous compounds), and the existence of large variations among tobacco cultivars, introductions, and species indicated that chemical constituents could be manipulated genetically. However, manipulation is difficult because genetic traits are not independent and complex interactions occur among various biochemical pathways in plant metabolism.

80–0197. Court, W. A.; Elliot, J. M. **Influence of Nitrogen, Phosphorus, Potassium, and Magnesium on the Phenolic Constituents of Flue-Cured Tobacco.** *Canadian Journal of Plant Science* 58(2): 543-548, April 1978.

A field experiment was conducted in 1974 and 1975 on Fox loamy sand to study the effects of different rates (0, 22.4, 44.8, and 67.2 kg/hectare) of nitrogen (N) fertilization on selected chemical constituents of flue-cured tobacco (*Nicotiana tabacum* L. cv. Delhi 34). Nitrogen fertilization increased the levels of total alkaloids, total N, and scopoletin in cured leaf. Chlorogenic acid, neo-chlorogenic acid, 4-caffeoylquinic acid, rutin, and scopolin were found to be inversely related to the rate of nitrogen. Phenolic constituents and inorganic nutrients in the leaves and inorganic nutrients in the soil were studied in 1975 and 1976 in another field experiment. Treatments included selected omissions of phosphorus, potassium, or magnesium from field plots grown continuously with tobacco since 1967. Marked nutrient deficiency symptoms were observed only in plots in which potassium had been omitted. Tobacco from these plots was generally lower in phenolic constituents. (Auth. Abs.)

80–0198. De Baets, A. **Influence de Quelques Techniques Agricoles sur la Teneur en Nicotine et le Potentiel en Condensat du Tabac en Feuille. [Influence of Some Agricultural Practices on Nicotine and Condensate of Tobacco Leaf.]** *Annales du Tabac* 13(2): 91-101, 1976, French.

During the years 1972 and 1973, experiments were carried out in order to study the influence of some agricultural practices on the alkaloid content and potential for smoke condensate formation of *Nicotiana tabacum* L., dark air-cured type, cultivar P.B. 91-1. Normal percentages of leaf nicotine, smoke nicotine, and smoke condensate under normal growing conditions (30,000 plants/hectare, 200 kg of nitrogen/hectare, topping and leaf harvesting) ranged from 0.6-1.0, 0.4-0.8, and 8-11, respectively, in the lower leaves; from 0.7-1.1, 0.5-0.9, and 9-12, in the middle leaf cutters; and from 1.6-3.0, 1.4-2.6, and 13-18 in the middle leaf wrappers. In the tips, these components ranged from 2.6-3.3, 1.8-3.0, and 16-33, respectively. Changing the number of plants per hectare (25,000, 30,000, 35,000, or 40,000), or changing the rate of nitrogen fertilization (100-150 or 200 kg/hectare, did not significantly influence these components. Nontopping induced a decrease of the nicotine content in the wrappers and tips of 50 percent or more. The condensate content only decreased 10 to 20 percent. Topping on a higher number of leaves per plant had the same influence, but the differences were not as large. Stalk-cutting compared to priming gave a higher nicotine content. Curing under dark plastic sheets, compared to transparent sheets, decreased the condensate forming potential of the leaf. (Auth. Abs. Mod.)

80–0199. Enzmann, J.; Siabatto-Pinto, T. **Untersuchungen uber den Borgehalt von *Nicotiana tabacum* L. unter Berucksichtigung des kritischen Grenzbereiches. [Studies on the Boron Content of *Nicotiana tabacum* L. With Reference to the Critical Margins.]** *Beitraege zur Tropischen und Subtropischen*

Landwirtschaft und Tropen Veterinaermedizin 14(2): 163-174, 1976, German.

Vegetation experiments were used to investigate the influence of different boron fertilizations on the boron content of various plant organs and on the yield. A highly significant positive correlation ($r = 0.94$) was found between the boron content of the upper leaves and the boron concentration in the nutrition substrate. The critical margin derived from the boron content of the leaves and the dry matter yield was 25 to 30 ppm in the upper and 50 to 60 ppm of boron in the lower leaves at the time of flowering. For assessing the boron supply of *N. tabacum*, only the margin of the upper leaves is recommended, since various influences (liming) can increase the boron content in the lower leaves and simulate an adequate supply. (Auth. Abs.)

80–0200. Gebri, P. **Leveltragyazas Hatasa a Dohany Hozamara, Minosegere, Asvanyi Osszetetelere. [Effect of Foliar Nutrition on Yield, Quality, and Mineral Composition of Tobacco.]** *Dohanyipar* (1): 19-25, January-February 1978, Hungarian.

As a result of foliar nutrition with nitrogen (N), phosphorus (P), and potassium (K), and with the microelements boron, magnesium, and iron, the yield and quality of Szabolesi and Virginia tobacco can be improved 1 to 9 percent. As a result of foliar nutrition with microelements the quantity of P and K in the leaf increased significantly and there was an accumulation of calcium, boron, and magnesium. With microelement nutrition and large doses of fertilizers, the mineral matter quantity of leaf is increased as well. In view of the microelement supply, foliar nutrition proved to be more effective than fertilization. The applied treatment combinations with foliar nutrients influenced favorably the nicotine concentration and the combustibility, but influenced unfavorably the texture quality of leaf. The jointly used microelements as fertilizers and foliar nutrients improved the mentioned three measures of value properties of tobacco leaf. By the propagation of stable industrial nutrient solutions containing microelements the possibility of microelement supply is more favorable as well. (Auth. Abs. Mod.)

80–0201. Jones, J. L.; Tramel, J. L., Jr. **Effects of Nitrogen Fertilization and Leaf Population on Yield and Quality of Virginia Dark-Fired Tobacco.** *Tobacco International* 181(4): 57-59, February 23, 1979.

Field experiments were conducted in 1972 and 1973 in Virginia to determine the response of Virginia dark-fired tobacco (*Nicotiana tabacum* L.) to three leaf populations (148,800; 185,000; and 222,000) per hectare (ha) at four nitrogen (N) rates (78, 122, 146, and 179 kg/ha). Significantly higher yields were obtained as the leaf populations were increased from 148,800 to 222,000 leaves per hectare; however, price (dollars/45.4 kg) significantly decreased at the 222,000 leaf population. Maximum value per hectare (yield x price) was obtained at the 185,000 leaf population. There was a trend for total alkaloids and total nitrogen to decrease as leaf population increased. Yields were increased as nitrogen rate was increased from 78 to 179 kg/ha. Price (dollars/45.4 kg) was significantly reduced as nitrogen rate was increased above 146 kg/ha. Total alkaloid and total nitrogen concentrations in the cured leaf were generally increased as nitrogen rates were increased. The results of this work indicate that approximately 150,000 leaves/ha and about 145 kg of N/ha is optimum for the production of Virginia dark-fired tobacco. (Auth. Abs.)

80–0202. Khan, P.; Hussain, T.; Khalil, J. K. **The Chemical Composition of Flue-Cured Virginia Tobacco at Leaf Stalk Position as Influenced by Potassium.** *Pak Tobacco* 2(2): 43-47, July 1978.

The effect of potassium on the moisture, ash, chloride, crude protein, sugars, nicotine, and potassium contents of tobacco leaves at three stalk positions of Coker 254 cultivar was studied under local conditions. The amount of moisture, ash, protein, sugars, and potassium showed a steady increase with increasing amounts of potassium fertilizer, while chloride and nicotine tended to decrease. The top leaves were found rich in nicotine, protein, and potassium, the middle leaves were rich in sugars, and the bottom leaves were rich in moisture, ash, and chloride. (Auth. Abs.)

80–0203. Leidy, R. B.; Sheets, T. J. **Residues From Two Formulations of Acephate on Flue-Cured Tobacco.** *Tobacco International* 180(13): 103-106, June 23, 1978.

Immediately after application of the emulsifiable concentrate (EC) of acephate, residues on bottom leaves of flue-cured tobacco cured by conventional methods averaged 16.7 and 51.4 ppm at the 0.56 and 1.12 kg/hectare rates, respectively. Corresponding values for the soluble powder (SP) formulation were 20.0 and 58.2 ppm. Middle leaves contained residues approximately 2.6 times greater than bottom leaves, and upper leaf residues were about 2.6 times greater than those for middle leaves. Between the time of application and 1 day after application, residues from both rates of the SP formulation declined 47 percent. After 1 day, residues from the 0.56 kg/hectare rate of the EC formulation did not decline, and the residue level for the 1.12 kg/hectare rate of EC was 76 percent of day 0, suggesting a difference between formulations the first 24 hours after application. Residue levels were similar for the two formulations during the remainder of the sampling period. Residues of methamidophos, a major metabolite of acephate, increased through day 5 on green tobacco and then declined by day 9. Methamidophos residues on cured tobacco were greater than those for acephate at day 3 and day 5. The total residue (acephate plus methamidophos) on cured leaf averaged 0.20 ppm on day 5 samples. Losses of total residues during curing averaged 97 percent. (Auth. Abs.)

80–0204. Malik, I. A. **Studies on Agronomic and Chemical Characteristics of Certain Flue-Cured Tobacco Varieties.** *Pak Tobacco* 1(2): 9-13, 1977.

Flue-cured tobacco varieties in Pakistan were compared for certain growth characteristics and yield per acre. Varieties Coker 347, Coker 254, Coker 316, Speight G-28, and Coker 213 were among the comparatively high-yielding group. Considerable differences were obtained in the cured leaf content percentage of total alkaloids, reducing sugars, total nitrogen, ash, and chlorides in the different varieties. All the varieties were found to be generally low in total alkaloids and reducing sugars. Optimum sugar-nicotine ratios of 10.17 to 12.17 were obtained in varieties Coker 254, Coker 347, and Speight G-28, whereas high ratios of 21.88 and 19.70 were obtained in varieties Coker 213 and Coker 316 indicating considerable mildness in smoking. Varieties Speight G-28 and Coker 347 appeared to be most promising with respect to yield, quality characteristics, and disease resistance. (Auth. Abs.)

80–0205. Murty, K.S.N.; Narasayya, K. L.; Sastry, A. S. **Effect of Different Fertiliser Treatments on the Chemical Composition and Quality of Flue-Cured Tobacco.** *Tobacco Research* 2(2): 126-133, December 1976.

The effect of different fertilizer treatments on the chemical composition and quality of flue-cured tobacco was studied using three levels of nitrogen (N) as ammonium sulphate (20, 40, and 60 kg N/hectare) and two levels of phosphorus as the single superphosphate (0 and 100 kg P_2O_5/hectare). These studies show that application of N above 20 kg/hectare in black soils results in a thicker leaf which is high in nitrogenous constituents and low in sugars and quality ratios. Thus 20 kg N/hectare appears to be adequate in black soils for producing flue-cured tobacco of acceptable quality. (Auth. Abs. Mod.)

80–0206. Mylonas, V. A.; Pangos, E. A. **Effects of Chemical Agents on Sucker Control and on Certain Agronomic and Chemical Characteristics in Burley Tobacco.** *Tobacco International* 180(15): 167-170, July 21, 1978.

Field experiments were conducted in Greece in 1974 and 1975 to evaluate the effects of the chemical agents Antak, AC-92390, Amchem 71-A25, Amchem 72-A45, Amchem 72-A146, and Off-Shoot-T on sucker control, agronomic characteristics, and chemical characteristics of the cured leaf of Burley tobacco. All sucker control chemicals resulted in reduced sucker weight and number per plant than the topped and untreated checks. The topped and hand-suckered or chemically suckered treatments resulted in higher yield per hectare, and in higher price and value per hectare over the untopped check. Statistically significant differences among the chemically suckered treatments were observed for suckers per plant, dry and fresh sucker weight per plant, yield (kg/hectare), price ($/kg), value ($/hectare), concentration of total alkaloids, and potassium concentration. There was no difference in total nitrogen content among the treatments although burning capacity tended to be lower with topping and hand or chemical suckering when compared to no topping. (Auth. Abs.)

80–0207. Neas, I.; Brown, G. W.; Dickerson, J. P.; Henderson, R. M.; James, W. B.; Line, W. B.; Threatt, H. C., Jr. **Evaluation of Once-Over Low-Profile Harvested Tobacco: Part II. Smoke Chemistry and Smoking Panel Evaluation.** *Tobacco International* 180(12): 28-31, June 9, 1978.

Smoke chemistry and smoking panel evaluation were determined on cigarettes for normal harvest (NH) and low-profile once-over harvest (LP) management systems. Puff counts of cigarettes for both harvest systems were similar except for LP-6 which exhibited a low number of puffs. The first two leaf primings of the NH system had lower FTC "tar" and total particulate matter (TPM) deliveries than any of the LP harvest treatments. The low nicotine and ammonia deliveries from the tobacco from the NH system were not available from the LP harvest treatments. A range of TPM water deliveries was observed for the NH treatments, whereas all the LP harvest treatments delivered nearly identical values. The atypical smoke values may mean a chemical imbalance in tobacco from the LP harvest system. The smoke analyses suggest that the system produces tobacco which is more like up-stalk tobacco from the NH system. The expert smoking panel preferred the low-profile early harvest treatments to the NH-1 priming treatment. No preference was evident between mid-harvest low-profile treatments and the NH-2 treatment. The panel did prefer the NH-3 and NH-4 priming treatments to the late dates of harvest of the low-profile treatment. (Auth. Abs.)

80–0208. Schmidt, J. A. **Fundamental Study Within the Framework of Simulation Experiments on the Tobacco Plant to Reduce Nicotine, Condensate and Pesticide Residues.** *Tabak Journal International* (3): 153-154, 156, June 1977.

Over the last 5 years the Institute for Tobacco Growing and Tobacco Research in Forchheim has developed new techniques that permit measurement of the assimilation activity of tobacco plants and the control of important growth parameters. By means of electronic measurements of ground moisture it has been possible to determine the degree of groundwater saturation which is most suitable for tobacco and increases the absorption of phosphoric acid and potassium. Feeding a chelated solution of trace elements leads to improved availability of natural elements in the plant which has a favorable effect on reduced absorption of nitrogen. Plants fed in this way are not attacked by aphids, have a higher resistance to diseases, and develop only small amounts of flowers or shoots. The harvested leaves have an improved filling capacity after curing and produce a very satisfactory output; the amounts of nicotine, condensate, and carbon monoxide in the smoke are reduced. Phytotron experiments confirmed the influence of the light spectrum on the strengthening of resistance and showed systemic infection in blue mold. (Auth. Abs. Mod.)

80–0209. Sievert, R. C. **Effect of Time of Infection With Potato Virus Y on Yield, Quality, and Chemical Constituents of Leaves of Burley Tobacco.** *Tobacco International* 180(11): 75-77, May 26, 1978.

Infection of "Burley 49" with potato virus Y (PVY) during the growing season caused significant reductions in tobacco leaf yield, quality, and value per hectare. Cured leaves from diseased plants had significantly more total nitrogen and acid insoluble nitrogen than did healthy leaves. Early inoculation resulted in leaves that had higher levels of nicotine and α-amino nitrogen than leaves from uninoculated or late inoculated plants. Total phenols, pH, water soluble acids, nitrate nitrogen, and soluble nitrogen were not significantly affected by infection with PVY. The effects of the virus were generally proportional to the length of time the virus was associated with the host, with the earliest infections having the greatest effect. Plants which became infected in June were shorter and flowered later than healthy plants or plants infected in July. (Auth. Abs.)

80–0210. Sievert, R. C. **Effect of Early Harvest of Burley Tobacco Infected With Potato Virus Y on Yield, Quality, and Chemical Constituents.** *Tobacco International* 180(10): 87-90, May 12, 1978.

Infection of Burley tobacco with potato virus Y reduced the yield, grade, and crop indices, and the value of the cured leaf. Harvesting the crop up to 3 weeks early did not reduce the losses. Diseased tobacco leaves had higher levels of total nitrogen, acid insoluble nitrogen, and nitrate nitrogen than healthy leaves. The amount of phenols and the percent total soluble nitrogen were generally lower in infected than in healthy leaves. The leves of α-amino nitrogen, total alkaloids, and water soluble acids were similar in both healthy and diseased tobacco harvested at the same time. Early harvesting altered the levels of most of the constituents but did not affect the amounts of phenols, water soluble acids, and nitrate nitrogen. (Auth. Abs.)

See also, **80–0214, 80–0235, 80–0236, 80–0237, 80–0238, 80–0242, 80–0244**

CHEMISTRY

80–0211. Behr, D.; Wahlberg, I.; Aasen, A. J.; Nishida, T.; Enzell, C. R.; Berg, J.-E.; Pilotti, A.-M. **(1*S*,2*E*,4*R*,6*E*,8*R*,11*S*,12*R*)-and (1*S*,2*E*,4*S*,6*E*,8*R*,11*S*, 12*R*)-8,11-Epoxy-2,6-Thunbergadiene-4,12-diol. Two New Diterpenoids of Greek Tobacco.** *Acta Chemica Scandinavica. Series B* 32(3): 221-227, 1978.

Two new diterpenoids were isolated from Greek *Nicotiana tabacum* L. One of them was shown to be (1*S*,2*E*,4*S*,6*E*,8*R*,11*S*,12*R*)-8,11-epoxy-2,6 thunbergadiene-4,12-diol by synthesis and X-ray analysis, while the other was tentatively assigned the structure (1*S*,2*E*,4*R*,6*E*,8*R*,11*S*,12*R*)-8,11-epoxy-2,6-thunbergadiene-4,12-diol by spectroscopic means. The implication of these results for the biogenesis of tobacco thunberganoids is discussed. (Auth. Abs.)

80–0212. Behr, D.; Wahlberg, I.; Nishida, T.; Enzell, C. R. **Structure Determination and Synthesis of 5(13),7*E*-**Megastigmadien-6,9-diol, a New Constituent of Greek Tobacco. *Acta Chemica Scandinavica. Series B* 31(7): 609-613, 1977.

A new C_{13} nor-carotenoid was isolated from Greek *Nicotiana tabacum* L. and shown by spectroscopic methods and synthesis to be 5(13),7*E*-megastigmadien-6,9-diol. The additional compounds obtained in the reaction of singlet oxygen with (±)-β-ionol were also characterized. (Auth. Abs.)

80–0213. Behr, D.; Wahlberg, I.; Nishida, T.; Enzell, C. R. **(3*E*,6*E*)-2,6-Dimethyl-10-Oxo-3,6-Undecadien-2-ol and (2*E*)-3-Methyl-4-Oxo-2-Nonen-8-ol. Two New Constituents of Greek *Nicotiana tabacum* L.** *Acta Chemica Scandinavica. Series B* 31(7): 573-576, 1977.

Two new compounds were isolated from Greek *Nicotiana tabacum* L. and shown to be (3*E*,6*E*)-2,6-dimethyl-10-oxo-3,6-undecadien-2-ol and (2*E*)-3-methyl-4-oxo-2-nonen-8-ol by spectroscopic methods. One of these belongs to the group of nor-compounds derived from acyclic isoprenoids, while the biogenetic origin of the other compound is uncertain. (Auth. Abs. Mod.)

80–0214. CORESTA. **CORESTA Recommended Method No. 1 ter. November 1978. Revised Method for the Determination of Dithiocarbamates in Tobacco.** *CORESTA Information Bulletin* (2): 28-29, 1978.

A revised method for determining dithiocarbamates in tobacco is proposed. The dithiocarbamates are decomposed on heating with hydrochloric acid in the presence of stannous chloride. The carbon disulfide (CS_2) which is formed is transferred with a current of nitrogen into a trap containing concentrated sulfuric acid to remove interfering substances, and then into a trap containing a methanolic solution of potassium hydroxide. The concentration of potassium *o*-methyl dithiocarbonate formed under these conditions is measured by spectrophotometry. As it is not normally known which dithiocarbamate is present in tobacco, results are expressed as carbon disulfide, and the values obtained using this method are taken to indicate the dithiocarbamate residue level. (Auth. Abs.)

80–0215. Demole, E.; Enggist, P. **A Chemical Study of *Virginia* Tobacco Flavour (*Nicotiana tabacum* L.). II. Isolation and Synthesis of *cis*-2-Isopropenyl-8-Methyl-1,2,3,4-Tetrahydro-1-Naphthalenol and 3-Isopropenyl-5-Methyl-1,2-Dihydronaphthalene.** *Helvetica Chimica Acta* 61(4): 1335-1341, April 1978.

Gas liquid chromatography was used to isolate *cis*-2-isopropenyl-8-methyl-1,2,3,4-tetrahydro-1-naphthalenol (A) and its dehydration product, 3-isopropenyl-5-methyl-1,2-dihydronaphthalene (B), from two small subfractions of Virginia tobacco condensate. Both these norsesquiterpenes were identified spectrally and synthesized from 1-methylnaphthalene in nine steps which resulted in the nonnatural, *trans*-alcohol A′. The possible biogenesis of A and B in tobacco is briefly outlined. (Auth. Abs.)

80–0216. Demole, E.; Enggist, P. **Identification of Twenty-One Novel Constituents of Oriental Tobacco Flavour (*Nicotiana tabacum* L.) Including (*E*) -3-Methyl-non-2-en-4-one, Pentadecan-15-Olide, 8α,13:9α,13-Diepoxy-15,16-Dinorlabdane, (*Z*)-Octadec-9-en-18-Olide, and (*E*)-2-Ethylidene-6,10,14-TrimethylPentadecanal.** *Helvetica Chimica Acta* 61(7): 2318-2327, November 1978.

Investigation by gas liquid chromatography of a small but organoleptically typical subfraction of Oriental tobacco condensate led to the identification of 47 compounds. Of these, 21 have not been previously reported as Oriental tobacco constituents, and 14 appear to be novel to all tobacco types. Two macrolides isolated represent the first musk compounds detected in tobacco. Identifications were made by direct comparison with the authentic chemicals synthesized whenever necessary. (Auth. Abs. Mod.)

80–0217. Devreux, M. G.; Bourcier, F. **Methode de Dosage Ponderal des Hydrocarbures du Tabac. [Method for Weight Determination of Tobacco Hydrocarbons.]** *Annales du Tabac* 13(1): 111-115, 1975, French.

A quick technique for the weight determination of tobacco hydrocarbons is described. Separation is done by partition column chromatography. The apolar components are eluted with petroleum ether and the weight is determined after evaporation. (Auth. Abs.)

80–0218. Drwiega, J.; Kuter, E. **Zastosowanie Chromatografii Gazowej do Oznaczania Weglowodanow Rozpuszczalnych w Tytoniu. [The Use of Gas Chromatography for Determination of Tobacco Soluble Carbohydrates.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* 1-2: 75-84, 1978, Polish.

A method for quantitative determination of soluble carbohydrates in native bright and dark tobaccos using gas chromatography is described. Identification was obtained by comparison of relative retention times of test and standard carbohydrate solutions. Glucose, fructose, and saccharose were identified. Prior to chromatographing, carbohydrates were transformed to silyl ethers with the use of a reaction mixture composed of pyridine, hexamethyldisilazane and trimethylchlorosilane. (Auth. Abs.)

80–0219. Dumas, C.; Badre, R.; Guillerm, R. **Methodologie du Dosage de la Nicotine et de la Cotinine Dans les Milieux Biologiques. [Methodology for Determining Nicotine and Cotinine in Biological Media.]** *Annales du Tabac* 13(1): 5-14, 1975, French.

A nitrogen-specific thermionic detector is used in a sensitive assay for nicotine in biological media. Extraction conditions (e.g., pH and solvents) for nicotine and cotinine in blood and urine are described as is a detailed gas phase chromatographic technique. Two applications are given: the kinetics of the plasma levels of five subjects after smoking one cigarette, and the measurement of nicotine impregnation (ng/ml) in plasma of active and passive smokers confined in the same enclosed space. (Auth. Abs. Mod.)

80–0220. Federal Trade Commission. **Report of "Tar" and Nicotine Content of the Smoke of 176 Varieties of Cigarettes.** Federal Trade Commission, Washington, D.C., December 1979, 12 pp.

The Federal Trade Commission's Laboratory has determined the tar (dry particulate matter) and total alkaloid (reported as nicotine) content of 176 varieties of cigarettes. The laboratory utilized the Cambridge filter method with some modifications. Of the 176 varieties tested, 18 were smoked to 23 mm. The butt length of the other 158 varieties tested ranged from 24.1 mm to an average of between 47.7 and 49.5. The butt lengths of 135 of the 176 varieties tested exceeded 30 mm. The samples used were obtained by attempting to purchase two packages of each variety of cigarettes as distributed by the seven domestic cigarette manufacturers during April 1979 in each of 50 geographic locations throughout the country. All varieties of cigarettes were not available in each of the 50 geographic locations and in these instances, one or more additional packages of cigarettes were purchased in those geographic locations where respective varieties were available. The samples utilized in the tests were representative of the 176 varieties of cigarettes as available throughout the country at the time of purchase. In the table listing the cigarette varieties in alphabetical order the tar content is reported to the nearest 0.1 mg and the nicotine to the nearest 0.01 mg, each with appropriate statistical values. The average weight is reported in gm per cigarette and the butt length range to the nearest 0.1 mm. In all other tables the average weight and butt length columns and the figures representing the standard deviation of the mean have been eliminated. The tar figures have been rounded to the nearest mg (0.5 mg and greater rounded up, 0.4 mg and less rounded down), and the nicotine figures have been rounded to the nearest tenth of a mg (0.05 mg and greater rounded up, 0.04 mg and less rounded down). Two tables respectively list varieties in increasing order of tar values and in increasing order of nicotine values. Accordingly, tar and nicotine figures in the tables and list represent rounded off averages without indication of their precision. (Auth. Abs. Mod.)

80–0221. Glenn, D. F.; Edwards, W. B., III. **Synthesis and Mass Spectrometry of Some Structurally Related Nicotinoids.** *Journal of Organic Chemistry* 43(14): 2860-2870, July 7, 1978.

The synthesis and mass spectrometry of a group of structurally related nicotinoids have been investigated. A detailed discussion is presented of their complex electron-induced fragmentation mechanisms, established with the aid of 27 site-labeled deuterium analogues, high-resolution measurements, and metastable ion studies. Compounds related to nicotine, cotinine, nornicotine, and myosanine were synthesized. (Auth. Abs. Mod.)

80–0222. Green, C. R. **Neutral Oxygenated Compounds in Cigarette Smoke and Their Possible Precursors.** In: *Recent Advances in Tobacco Science.* Series 3, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1977, 94-120.

This paper reviews neutral oxygenated compounds such as esters, lactones, aldehydes, ketones, and furans, which account for about 40 percent of known smoke constituents. These compounds are important to the flavor and aroma of cigarette smoke and arise in smoke from either direct transfer or pyrolysis of oxygen containing tobacco constituents such as carbohydrates.

80–0223. Griest, W. H.; Guerin, M. R. **Influence of Tobacco Type on Smoke Composition.** In: *Recent Advances in Tobacco Science.* Series 3, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1977, pp. 121-144.

This paper reviews information on Bright and Burley tobacco cigarettes and compares characteristics such as weight, puff number, length, cut, paper, blend, and smoke volume. Quantitative analyses of their smoke and smoke condensate components (e.g., carbon monoxide, nicotine, total nitrogen, phenols, sugars) are listed and discussed. Data from literature which compares the influence of more than one tobacco on smoke composition and which uses the same analytical procedure and smoking conditions are also discussed. Finally, a summary of the influence of other variables (tobacco, origin, tobacco variety, curing practice, reconstitution, substitution) on smoke composition is listed.

80–0224. Hsieh, T.C.Y.; Kaul, K.; Laine, R. A.; Lester, R. L. **Structure of a Major Glycophosphoceramide from Tobacco Leaves, PSL-I: 2-Deoxy-2-Acetamido-*D*-Glucopyranosyl (α1—4)-*D*-Glucuronopyranosyl(α1 —2)Myoinositol-1-*O*-Phosphoceramide.** *Biochemistry* 17(17): 3575-3581, August 22, 1978.

The chemical structure of a major glycophosphoceramide from tobacco leaves, called PSL-I, has now been characterized as 2-deoxy-2-acetamido-*D*-glucopyranosyl-(α1—4)-*D*-glucuronopyranosyl (α1—2)myoinositol-1-*O*-phosphoceramide. Sites of glycosidic linkage were determined by methylation analysis on a trisaccharide isolated by degradation of carboxyl-reduced PSL-I and periodate oxidation experiments on PSL-I. The resulting products were identified with gas chromatography/mass spectrometry. Anomeric configurations were determined by resistance of the sugars in the peracetylated trisaccharide to chromium trioxide treatment. (Auth. Abs.)

80–0225. Ivanov, N.; Ognianov, I. **Izsledovane na Eterichnoto Maslo i Smolite v Bulgarskite Orientalski Tiutiuni. Suobshtenie I. Neutralna Fraktsiia. [Investigation of the Essential Oil and Resins Contained in Bulgarian Oriental Tobaccos. Communication I. Neutral Fraction.]** *Akademia Naselskostopanskite Nauki. Izvespia na Instituta po Tiutiuna (Plovdiv),* 3: 227-235, 1963, Bulgarian.

The essential oil and the resins contained in a brand of Bulgarian oriental tobaccos were extracted by means of ethyl ether. The extractive substances were divided into several fractions. The following substances were isolated from the neutral fraction by means of adsorption chromatography, infrared spectroscopy, and elementary analysis: a mixture of saturated aliphatic carbohydrates, aromatic monocyclic diterpene, neophitadiene, and two aliphatic alcohols. (Auth. Abs.)

80–0226. Jimenez, S. M. **Recopilacion Sobre la Relacion Contenido Potasico/Calidad del Tabaco. [Review of the Relationship Between Potassium Content and Tobacco Quality.]** *Instituto Nacional de Investigaciones Agrarias Anales, Serie Tecnologia Agraria,* 4: 149-166, 1977, Spanish.

This is a compilation of research concerning problems of tobacco quality (combustion, burning properties, mineral and organic composition) in relation to potassium content. The multiple influences potassium content has on tobacco quality (water content, insect resistance, carbohydrate content) are discussed. The potassium soil levels, the symptoms of potassium deficiency, the apportionment and equilibrium of potassium in soil, and the potassium gradings in tobacco soils are also studied. (Auth. Abs.)

80-0227. Johnson, W. R. **The Pyrogenesis and Physicochemical Nature of Tobacco Smoke.** In: *Recent Advances in Tobacco Science*. Series 3, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1977, pp. 1-27.

This paper reviews information published on the physical mechanisms of cigarette smoke formation. The distribution of radioactively labeled cigarette smoke, the gas concentration within a cigarette, the temperature distribution in the coal, the atmospheric oxygen incorporation (using ^{18}O) into sidestream and mainstream smoke, and the combustion zone airflow pattern are some of the subjects discussed.

80-0228. Levins, R. J. **Isolation of Polyaromatic Hydrocarbons From Whole Smoke Condensate: A Simple Two-Step Procedure.** *Chromatographia* 11(12): 736-741, December 1978.

A simple two-step procedure for isolating polyaromatic hydrocarbons from cigarette whole smoke condensate has been devised. Preliminary separation of a blue fluorescent zone from whole smoke condensate by preparative thin-layer chromatography yields a crude polyaromatic hydrocarbon fraction. A highly enriched polyaromatic hydrocarbon fraction is obtained by passage through a short Sephadex LH-20/isopropanol column. The isolation procedure and a temperature-programed gas chromatographic scan can be run in less than 8 hours. (Auth. Abs.)

80-0229. Mueller, G. **Schwermetallgehalte (Cd, Zn, Pb, Cu, Cr) im Tabak haufig in der BR Deutschland gerauchter Zigaretten. [Heavy Metal Content (Cd, Zn, Pb, Cu, Cr) of Tobacco in Commonly Smoked Cigarettes in the Federal Republic of Germany.]** *Chemiker-Zeitung* 103(4): 33-37, 1979, German.

Tobacco of 15 commercial cigarette brands, commonly smoked in the Federal Republic of Germany, were analyzed for various heavy metals. The following minimum, mean, and maximum concentrations (in μg/g = ppm) were found: cadmium (Cd) 1.07-1.46-2.30; zinc (Zn) 33-45-64; lead (Pb) 2.4-3.3-4.3; copper (Cu) 10-21.7-34; chromium (Cr) 3.4-4.9-6.7; iron (Fe) 292-428-572; and manganese (Mn) 71-133-176. Positive correlations are highly significant between Fe and Cr, significant between Zn and Cd, and probable between Mn and Cr. The very high Cd concentration in tobacco, which is about 10 times higher than in other plants or plant products, is emphasized with respect to a possible synergistic influence with harmful carcinogenic organic compounds known to be produced during cigarette smoking. (Auth. Abs.)

80-0230. Muramatsu, M.; Mikami, T.; Naito, N.; Tomita, H. **A Model Study on the Diffusion and the Dilution of Low Molecular Weight Gaseous Components Through Cigarette Paper During Smoking.** *Beitraege zur Tabakforschung* 9(3): 141-146, October 1977.

The effects of diffusion and dilution on the concentration of gaseous components have been studied by a model consisting of unlit cigarettes differing in paper porosity and length and a standard gas mixture. The difference in the concentration of gaseous components between the front and butt end of the cigarette during the puff increased with cigarette length and paper porosity, and was also dependent on the diffusion coefficient of respective components through the cigarette paper. These changes could be explained mainly by the diffusion loss through the paper in the case of the cigarette with ordinary paper, but by both dilution with air through the paper and diffusion loss in the case of the cigarette with perforated paper. Subsequently, the levels of gaseous components such as carbon monoxide in the exit stream become lower in the latter cigarette. An equation has been derived which can fully account for these changes. (Auth. Abs. Mod.)

80-0231. Nakane, M.; Hutchinson, C. R. **Biosynthetic Studies of Secondary Plant Metabolites With $^{13}CO_2$. *Nicotiana* Alkaloids. 2. New Synthesis of Nornicotine and Nicotine. Quantitative Carbon-13 NMR Spectroscopic Analysis of (2′, 3′, *N*-CH_3-$^{13}C_3$) Nicotine.** *Journal of Organic Chemistry* 43(20): 3922-3931, September 29, 1978.

An efficient synthesis of the tobacco alkaloids, nornicotine (1a) and nicotine (1b), is achieved by Michael condensation of the α-lithiomethoxime of 3-acetylpyridine with a ketene thioacetal monoxide providing all the pyrrolidine ring atoms of 1a in masked form. Subsequent reduction with diborane and reductive cyclization in refluxing 97 percent formic acid are used to produce *N*-formyl-1a in high yield. The latter is converted to 1a or 1b by literature procedures in 60 percent overall yield from 3-acetylpyridine. The chemistry of several alternative, but inefficient, synthetic approaches to 1a also is discussed, in particular a route from 3-acetylpyridine and mesylaziridine. The synthesis of (2′,3′,*N*-CH_3-$^{13}C_3$)-1b is achieved by this route from (1,2-$^{13}C_2$)acetic acid and (^{13}C)formaldehyde via (1′,2′-$^{13}C_2$)-3-acetylpyridine. Analysis of the proton-decoupled ^{13}C NMR spectrum of the triply ^{13}C-labeled 1b is done to certify the accuracy of the quantitative determination of the relative ^{13}C enrichment in 1b biosynthetically labeled by $^{13}CO_2$. An earlier conclusion about the symmetry of ^{13}C labeling of carbons 2′ and 5′ of 1b is circumstantially validated, i.e., that these four carbons are unequally ^{13}C labeled by $^{13}CO_2$ within experimental error. (Auth. Abs.)

80–0232. Norman, V. **An Overview of the Vapor Phase, Semivolatile and Nonvolatile Components of Cigarette Smoke.** In: *Recent Advances in Tobacco Science*. Series 3, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1977, pp. 28-58.

This is an overview of the volatile and nonvolatile components of the particulate and vapor phases of cigarette smoke. Quantitative methods for their determination, such as gas chromatography and nondispersive infrared, are briefly mentioned. The major classes of components found in smoke condensate (e.g., aldehydes, lactams, phenols, pyridines, sterols), their constituents (e.g., benzaldehyde, cotinine, catechol, nicotine, stigmasterol), and metals are listed. Hydrocarbons, ketones, pyridines, phenols, furans, pyrazines, and indoles are listed in decreasing order as major group components of semivolatiles.

80–0233. Pitner, T. P.; Edwards, W. B., III; Bassfield, R. L.; Whidby, J. F. **The Solution Conformation of Nicotine. A ^{1}H and ^{2}H Nuclear Magnetic Resonance Investigation.** *Journal of the American Chemical Society* 100(1): 246-251, January 4, 1978.

The ^{1}H nuclear magnetic resonance (NMR) spectrum of nicotine is analyzed in detail. Selectively deuterated nicotine analogues afford considerable simplification of the pyrrolidine ^{1}H resonances by allowing partition of this seven-spin system into three-, four-, and five-spin systems. In addition, the ^{2}H NMR chemical shifts of these analogues provide a means of assigning ^{1}H NMR chemical shifts to specific protons unambiguously. The vicinal coupling constants of the pyrrolidine ring protons suggest an envelope conformation for the five-membered ring, in which the methyl and pyridine moieties assume equatorial positions. A perpendicular spatial orientation of the pyridine and pyrrolidine rings is supported by two observations: a small long-range coupling constant (<0.05 Hz) between H(2′) and H(6), and nuclear Overhauser enhancements of 9 ± 2 and 5 ± 2 percent for the H(2′) resonances upon saturation of H(2) and H(4), respectively. (Auth. Abs.)

80–0234. Reynolds, J. H.; Wheeler, J. P. **A Method for the Study of Air Diluted Cigarettes.** *Tobacco International* 179(15): 137-142, July 22, 1977.

A useful method for the study of air-diluted cigarettes has been devised. The method consists of enclosing the ventilated portion of a cigarette within a vessel that separates it from the surrounding atmosphere in which the burning coal is located. The vessel is constantly swept with argon. Trapping of the mainstream gas-phase smoke and analysis of it for oxygen, nitrogen, and argon yield data which are used to calculate the percent air dilution and the amount of oxygen consumed. The trapped smoke can also be analyzed for other gas-phase components. Important advantages of the method include the same cigarette yields information regarding air dilution and oxygen consumption as well as on gas phase smoke components. Constant attention to the cigarette during smoking is not required. At very high levels of air dilution no resistances to flow are added to the ventilation stream. (Auth. Abs.)

80–0235. Richter, M. **Uber die Untersuchung von Organochlor-Insektizidruckstanden in Tabak und Tabakwaren. 1. Mitt. Die Analysenmethode. [On the Study of Organochlorine Insecticide Residues in Tobacco and Tobacco Products. Part I. The Analysis Method.]** *Nahrung* 22(9): 759-768, 1978, German.

An analytical method for quantitative serial estimation of organochlorine insecticide residues in tobacco is reported. This method is based on hexane extraction followed by column chromatographic clean-up and preseparation as well as qualitative and quantitative gas chromatographic analysis. The following 16 substances are analyzed: HCB, α-HCH, lindane, heptachlor, aldrin, heptachlor epoxide, *o,p′*-DDE, *p,p′*-DDE, *o,p′*-TDE, *p,p′*-TDE,*o,p′*-DDT, dieldrin, endosulfan, β-endosulfan, and endosulfan sulfate. In a sample of 5 g of tobacco the following substances can be detected: HCB, α-HCH, lindane, heptachlor, aldrin, heptachlor epoxide—0.001 ppm; *o,p′*-DDE, *p,p′*-DDE, dieldrin—0.003 ppm; *o,p′*-TDE, *p,p′*-TDE—0.01 ppm; *o,p′*-DDT, α-endosulfan, β-endosulfan—0.02 ppm; endosulfan sulfate—0.03 ppm; *p,p′*-DDT—0.05 ppm. (Auth. Abs.)

80–0236. Richter, M. **Uber die Untersuchung von Organochlor-Insektizidruckstanden in Tabak und Tabakwaren. 2. Mitt. Ruckstandsmengen der in der DDR verarbeiteten Rohtabake. [On the Study of Organochlorine Insecticide Residues in Tobacco and Tobacco Products. Part II. Residue Levels of Leaf Tobaccos Used in the GDR.]** *Nahrung* 22(9): 769-776, 1978, German.

The levels of insecticide residues in East German and imported leaf tobaccos are reported. With this analytical method 16 chlorinated hydrocarbons can be analyzed. No DDT was found in tobaccos from the People's Republic of Korea. The mean DDT level of tobaccos from Hungary was lower than 0.1 ppm, the possible value of the future tolerance. With few exceptions samples of other countries contained less than 1 ppm of the total DDT group. Only a cigar tobacco from Paraguay showed a very high residue level of 7.20 ppm. Except for this sample, the residue values are attributed to the DDT persistence in soil and the relatively high uptake by tobacco, since, with the exception of a few countries, DDT is no longer used for tobacco. The lindane concentration was very low in all tobaccos. In some tobaccos, traces or only very small quantities of other organochlorine residues were detected. (Auth. Abs.)

80–0237. Schiltz, P.; Albo, J.-P.; Cazamajour, F. **Relations Entre les Teneurs en Alcaloides et en Cendres chez les *N. tabacum*. Incidence Varietale sur les Taux D'Alcaloides et de**

Cendres. [Alkaloid and Ash Content Relationships in *N. tabacum*. The Influence of Varieties on Alkaloid and Ash Contents.] *Annales du Tabac* 13(2): 9-18, 1976, French.

The alkaloid and ash contents of a *Nicotiana tabacum* variety and of related tobaccos were studied under three experimental conditions: the alkaloid and ash cotyledon test, which provides some evidence of hereditary properties of tobacco, the leaf test, which permits comparison of the influence of three nutrition types, and finally, field culture with topping and air curing. In these experimental conditions, a variability was observed, but a positive correlation between alkaloid and ash levels is shown. It is assumed that this correlation reflects the hereditary properties of the variety. When a similar experiment was carried out with very different tobacco varieties (Burley, Kentucky, Dark tobacco, Oriental tobacco, Virginia) a negative correlation was established between alkaloid and ash contents. The varieties, however, can be arbitrarily grouped in families where a positive correlation is possible. This result suggests the hypothesis that families reveal hereditary relationships. Finally, these experiments gave an opportunity to verify the results obtained with the alkaloid and ash cotyledon test which facilitates selection of *N. tabacum* for the two properties under study. (Auth. Abs.)

80-0238. Schiltz, P.; Coussirat, J.-C. **Relations Entre les Teneurs en Alcaloides et en Cendres Chez les *N. tabacum*. Variations des Taux d'Alcaloides et de Cendres en Fonction de Modifications de l'Alcaloidogenese et de Certaines Conditions de Milieu. [Alkaloid and Ash Content Relationships in *N. tabacum*. Alkaloid and Ash Content Variations Following Modifications of Alkaloidgenesis and Some Environmental Factors.]** *Annales du Tabac* 13(2): 19-26, 1976, French.

A positive correlation between alkaloid content and potential for ash formation having been established, a study was made of their variation when tobacco was submitted to several light intensities, different concentrations of a gibberellic acid solution, various plant spacing densities, three vegetation durations, and finally, humidification of the aerial part of the plant. The relationship of alkaloid content to potential for ash formation is a recessive type and is linked to alkaloidgenesis. This normally positive relationship is modified when the plant genome is interfered with, and this may lead to a negative correlation when several situations are compared. (Auth. Abs.)

80-0239. Shannon, I. L., Trodahl, J. N. **Sugars and Fluoride in Chewing Tobacco and Snuff.** *Texas Dental Journal* 96(1): 6-9, January 1978.

Sucrose, glucose, and fluoride concentrations were measured in 23 brands of chewing tobacco (13 leaf-type and 10 plug), 8 brands of powdered tobacco products (snuff-type), and dried tobacco leaf that had not been processed. Leaf and plug chewing tobaccos contained relatively high levels of added sugars but the concentrations were quite low in the snuff-type products. Fluoride concentrations were in the 2-3 ppm range for the chewing tobaccos and less than 1 ppm in the powdered tobacco materials. Analysis of unprocessed tobacco leaf indicated no sucrose, a glucose concentration of 0.2 percent, and a fluoride level of 4.75 ppm. (Auth. Abs. Mod.)

80-0240. Takagi, Y.; Fujimori, T.; Kaneko, H.; Fukuzumi, T.; Noguchi, M. **Isolation of a New Tobacco Constituent, (3S, 5R, 6S, 9ξ)-3-Hydroxy-5,6-epoxy-β-ionol, from Japanese Domestic SUIFU Tobacco.** *Agricultural and Biological Chemistry* 42(9): 1785-1787, September 1978.

A new compound related to tobacco carotenoids (previously described as compound A) has been isolated from Japanese air-cured tobacco, *Nicotiana tabacum* cv. SUIFU, and identified as (3S, 5R, 6S, 9ξ)-3-Hydroxy-5, 6-epoxy-β-ionol. Mass, infrared, and nuclear magnetic resonance spectrometry were used in structural confirmation.

80-0241. Tancogne, J. **Methode Rapide d'Estimation des Residus de Carbaryl Dans le Tabac. [Rapid Method of Evaluating Carbaryl Residues in Tobacco.]** *Annales du Tabac* 13(2): 149-153, 1976, French.

A quick method of evaluating carbaryl residues in tobacco is described. The method is based upon thin-layer chromatography of a nonpurified tobacco extract, followed by an exposure of the insecticide to esterase inhibition. The lower determination limit is 0.5 ppm. The ability to sort out noncontaminated tobaccos with a net saving of labor is the main advantage of this method. (Auth. Abs.)

80-0242. Tancogne, J.; Chouteau, J. **Les Phytosterols du Tabac. Variation des Teneurs en Fonction de l'Etage Foliaire et de la Fertilisation Azotee. [Tobacco Phytosterols. Content Variations According to Leaf Level and Nitrogen Fertilization.]** *Annales du Tabac* 13(2): 79-89, 1976, French.

Desmethylsterols and β-amyrin have been quantitatively determined by thin-layer chromatography followed by gas chromatography. The first results of this study show that in the case of topped, stalk-harvested dark tobacco, the sterol contents decrease from the lower to the upper leaves. Furthermore, an increase of nitrogen fertilization results in a decrease of the sterol contents without modifying their individual proportions. The sterol contents of dark French tobaccos are relatively low. (Auth. Abs.)

80-0243. Tancogne, J.; Chouteau, J. **Les Phytosterols du Tabac. Variation en Fonction du Stade Vegetatif, de l'Ecimage et de l'Etage Foliaire. [Tobacco Phytosterols. Variations According to Stage of Vegetation, Topping and Position of the**

Leaf on the Stalk.] *Annales du Tabac* 14(2): 185-195, 1977, French.

Phytosterols in tobaccos coming from an experimentally staggered harvest of topped and nontopped plants have been measured. The results of this study show that total sterols increase with the age of the plants, whereas topping induces a marked decrease in the sterol contents. There is a rather close relationship between sitosterol and stigmasterol contents. These two sterols appear to be complementary; their ratio indicates a marked gradation according to the position of the leaf on the stalk. The upper leaves are relatively richer in sitosterol than the leaves in other positions. The free sterols/total sterols ratio decreases with maturity, but in the lower leaves it is markedly higher than in the middle and upper leaves. (Auth. Abs.)

80–0244. Tancogne, J.; Chouteau, J.; Cazamajour, F. **Residus Laisses par la Butraline Employee Comme Inhibiteur de Bourgeons. [Residues Left by Butralin Used as a Sucker Inhibitor.]** *Annales du Tabac* 14(2): 217-223, 1977, French.

A method designed for the rapid quantitative determination of organochlorine insecticides in tobacco was used to determine the residues left by Butralin employed as a sucker inhibitor and applied using various methods. Distributed in local sprayings restricted to the top of each plant, Butralin leaves only a slight amount of residues averaging about 1 ppm, with higher values for the lower leaves than for the middle and upper leaves, in air-cured tobaccos. Butralin applied through a sprinkling ramp at dosages of 5,000 and 10,000 gm of active matter per hectare results in higher residue levels which can reach 30 ppm in the lower leaves. (Auth. Abs.)

80–0245. Trong-Lang, N. T. **Alpha-Carbon Hydroxylation in the Metabolism of Tobacco Alkaloids.** Doctoral Dissertation, University of California, San Francisco, University Microfilms 77-13,876, 1977, 266 pp.

The work described in this dissertation is concerned with the further elucidation of the structures and the modes of formation of metabolites of the major nicotine alkaloids. For these studies a synthetic scheme was designed which provides for the introduction of deuterium at all sites of the five-membered ring of nicotine and cotinine, as well as the synthesis of nornicotine deuterium-labeled at the C_2' and C_3' positions of the pyrrolidinyl moiety. Studies on the metabolism of nornicotine by 10,000 x g rabbit liver supernatant fractions led to the discovery of two new metabolites, myosmine and 2′-(3-pyridyl)-$\Delta^{5'}$-pyrroline. (Auth. Abs. Mod.)

80–0246. Vajdics, Z.; Farkas, J.; Dezso, Z.; Ori, F. **Kulonbozo Porozitasu Cigarettapapirokkal Gyartott es Perforalt Cigarettak Vizsgalata, Kulonos Tekintettel a Zart Veggel mert Legellenallasra es a Fofust Nikotin Alakulasra. [Examination of Cigarettes Made From Papers Having Different Porosity.]** *Dohanyipar* (3): 108-112, May-June 1978, Hungarian.

The relationship between paper porosity and air resistance measured by closed end was studied by measuring nicotine formation in mainstream smoke. In the measured range the nicotine content of the mainstream can be reduced (by about 25 percent) by increase in paper porosity, but perforation had no influence on nicotine content of mainstream smoke.

80–0247. Viart, P.; Penven-Guilleau, R. **Indice Phenolique du Tabac. [Phenolic Index of Tobacco.]** *Annales du Tabac* 13(1): 21-32, 1975, French.

The Folin-Ciocaltau reagent can be used to characterize the phenolic content of tobacco samples. A study of the conditions resulted in a definition of detailed procedure giving reproducible and sufficiently specific results, after denicotinization of the tobacco extracts by silicotungstic acid. Several indices will probably be necessary for a complete study of phenolic compounds. Some of them are volatile and have a direct influence on the organoleptic properties of tobacco. The phenol functions of the less volatile components may be free or masked (glucosides, brown pigments); their influence on smoke composition, for example, probably depends on the importance of the structures represented in the tobacco. The method proposed corresponds theoretically to the free phenol functions. (Auth. Abs.)

80–0248. Wahlberg, I.; Curvall, M.; Enzell, C. R. Syntheses of (12*R*,13*S*)- **and (12*S*,13*R*)-8,12-Epoxy-14-Labden-13-ol and (12*S*,13*R*)-8,13-Epoxy-14-Labden-12-ol, Three Tobacco Diterpenoids.** *Acta Chemica Scandinavica. Series B* 32(4): 310-312, 1978.

The preparation of three tobacco labdanoids by peracid oxidation of (12*E*)-abienol is reported. Four tobacco diterpenoids have previously been synthesized from (12*Z*)-abienol, which has been proposed as a precursor of tobacco labdanoids. Since three of the products obtained by peracid oxidation of (12*E*)-abienol are tobacco constituents, and since none of these are formed by peracid oxidation or photo-oxygenation of (12*Z*)-abienol, it is reasonable to assume that both isomers of abienol may serve as precursors of the labdanes and nor-labdanes found in processed tobacco.

80–0249. Wahlberg, I.; Karlsson, K.; Curvall, M.; Nishida, T.; Enzell, C. R. **Sensitized Photo-Oxygenation of (12*Z*)-Abienol. Biomimetic Synthesis of Tobacco Labdanoids.** *Acta Chemica Scandinavica. Series B* 32(3): 203-215, 1978.

The reaction of (12*Z*)-abienol with singlet oxygen was found to occur with competition between four processes to give labdanic compounds. Since several of the photo-oxygenation products obtained have previously been identified as tobacco constituents, the present results provide experimental support for the view that (12Z)-abienol may be a precursor of the labdanic compounds found in processed tobacco. Tobacco cultivars often produce diterpenoids of the labdane and thunbergane types which are highly susceptible to light and oxidation. This fact is of particular interest from the tobacco flavor point of view, since it is likely that the products of the catabolism of these diterpenoids are low-molecular weight odoriferous compounds.

See also, **80–0194, 80–0204, 80–0207, 80–0262**

PHARMACOLOGY AND TOXICOLOGY

80–0250. Adamsson, E.; Piscator, M.; Nogawa, K. **Pulmonary and Gastrointestinal Exposure to Cadmium Oxide Dust in a Battery Factory.** *Environmental Health Perspectives* 28: 219-222, February 1979.

The elimination of cadmium in feces was studied in a group of 15 male workers exposed to cadmium oxide dust in a nickel-cadmium battery factory. The elimination of cadmium in feces was on the average of 619 and 268 μg/day in seven smokers and eight nonsmokers, respectively. The corresponding ranges were 97-2577 and 31-1102 μg/day. A significantly higher fecal concentration of cadmium was found in smokers compared to nonsmokers ($p<0.05$). Cadmium concentrations in blood were also significantly higher in smokers than in nonsmokers, both before and after 1 month of vacation ($p<0.01$ and $p<0.05$, respectively). Among the smokers, there was a significant decrease in the cadmium concentrations during the vacation period, but not among the nonsmokers. It was estimated that cadmium naturally occurring in food and cigarettes, cadmium excreted from the gastrointestinal tract, and cadmium transported from the lungs by mucociliary clearance to the gastrointestinal tract only could explain up to 100 μg of the cadmium in the feces. Since much higher values for fecal cadmium were recorded even among some nonsmokers, this was interpreted as being the result of ingestion of cadmium from contaminated hands and other body surfaces. Among the smokers, direct oral contact with contaminated cigarettes or pipes is an additional factor; the smokers also inhale cadmium in the tobacco smoke from contaminated cigarettes. Part of that cadmium is transferred to the gastrointestinal tract by mucociliary clearance and also adds to the fecal cadmium. (Auth. Abs. Mod.)

80–0251. Basrur, P. K.; McClure, S.; Zilkey, B. **A Comparison of Short Term Bioassay Results With Carcinogenicity of Experimental Cigarettes.** In: Neiburgs, H. E. (Editor). *Prevention and Detection of Cancer. Part 1. Prevention. Volume 2. Etiology. Prevention methods.* New York, Marcel Dekker, 1978, pp. 2041-2048.

The short-term tests currently used for assessing the carcinogenicity of chemical compounds include the mouse skin response test based on sebaceous gland suppression and epidermal hyperplasia indices and the mutagenicity test based on the ability of a substance to cause a reproducible change in the bacterial genome. In an attempt to determine the tumor predictive value of these tests, a set of four experimental cigarettes with known tumor induction potential in long-term experiments was subjected to these two short-term tests. A comparison of the data showed that the two samples which produced lower tumor response in long-term experiments can be separated from those with higher tumor response on the basis of a majority of criteria assessed in short-term studies. However, the ranks of the four cigarettes based on their relative potency to elicit each of these responses followed somewhat different trends. Mutagenicity ranks with condensates and smoke on a concentration basis showed similar trends for the extremes whereas the intermediates varied in ranks with smoke and condensate. The total tumor potency trend paralleled the mutagenicity ranks of condensate calculated on a per cigarette basis. The sebaceous gland suppression response with 10 percent condensates simulated the trend shown by bacterial mutagenicity response to smoke on a volume basis, and with higher condensate dose this response followed the trend of the carcinoma response elicited by these cigarettes in long-term experiments. The epidermal hyperplasia response assumed to be a measure of the promoters in cigarette smoke was not helpful in rating cigarettes, whereas the sebaceous gland suppression response which reflects the tumor initiator levels, and the mutagenicity response which represents one of the prerequisite steps in cancer induction process, may be used as indices of biological insult correlated with the tumor inducing potency of cigarettes. (Auth. Abs.)

80–0252. Bayne, C. K. **A Probability Prediction Model of Mouse Skin Tumors Based on Chemical Components of Cigarette Smoke Condensates.** U.S. Department of Energy, Oak Ridge National Laboratory, Publication No. ORNL/TM-7037, Oak Ridge, October 1979, 38 pp.

Smoke condensates generated from a large number of cigarette blends have been painted on the backs of mice to try to produce histopathological verified tumors. Corresponding samples of each smoke condensate were also analyzed for the concentrations of selected chemical constituents (nicotine, benzo(*a*)pyrene, benz(*a*)anthracene, phenol, *o*-cresol, *m*- and *p*-cresol, pH, weak acid, very weak acid). Relating the biological data to the chemical analyses sheds light on the undesirable smoke condensate constituents and can be used to design a less hazardous cigarette. This relationship is formulated as a mathematical model that predicts the probability that a mouse will be tumor free as a function of the concentration of the applied smoke condensate and eight of the chemical constituents of the smoke condensate. The prediction model is based on 196 data points from the four series of experiments conducted for the Towards A Less Hazardous Cigarette Program. Using a least squares analysis, a 17-term prediction model was found that predicted probabilities as well as a full 66-term second-order model. The prediction model accounted for 76.6 percent of the total variation and 70.4 percent of the observed values are within one standard deviation of the predicted values.

80–0253. Benignus, V. A.; Otto, D. A.; Prah, J. D.; Benignus, G. **Lack of Effects of Carbon Monoxide on Human Vigilance.** *Perceptual and Motor Skills* 45(3, Part 1): 1007-1014, December 1977.

Conflicting results have been reported concerning the effects of low levels of carbon monoxide (CO) on human vigilance performance. This article presents a critical review of the literature and the results of a study employing 52 human male subjects performing a numeric monitoring task. Carbon monoxide levels were 0, 100, and 200 ppm, which produced mean carboxyhemoglobin levels of 0.01, 4.61, and 12.62 percent, respectively. No CO-exposure levels produced any effect on vigilance performance. The power of the statistical test for CO effects was shown to be quite high, even for fairly trivial possible decrements of performance. (Auth. Abs. Mod.)

80–0254. Berg, K.; Borresen, A.-L.; Dahlen, G. **Effect of Smoking on Serum Levels of HDL Apoproteins.** *Atherosclerosis* 34(3): 339-343, November 1979.

Quantitative determination of high density lipoprotein apoproteins (apoA) in serum from 97 healthy men showed a significantly lower apoA-I and apoA-II level in smokers than in nonsmokers. Also a significant negative correlation was found between the apoA-I or apoA-II levels and the number of cigarettes smoked per day ($0.02<p<0.05$). (Auth. Abs.)

80–0255. Caille, E.-J.; Bassano, J.-L. **La Cigarette Dans le Champ Cerebral. [Cigarette Smoking Effect in the Cerebral Field.]** *Psychologie Medicale* 8(6): 893-908, 1976, French.

Operational behavior and cerebral electrogenesis were studied in a selected group of 12 adults, aged 23 to 26, at the same level of general vulnerability. The selected subjects exhibited a clearly modulated large amplitude cortical signal and a carboxyhemoglobin (COHb) level characteristic of habitual smokers. Each subject was deprived of smoking for 36 to 40 hours. The subject then smoked a cigarette at a rate of one puff every 10 sec during cerebral electrogenesis, or at a random rate during execution of psychomotor, decisionmaking, or cognitive tasks. The cigarettes contained either 0, 0.6, or 1.6 mg of nicotine. After smoking a nicotine containing cigarette, experimental data analysis showed improvement in all operational behavior parameters except visual motor coordination. The heart rate, critical fusion frequency, and mobility and complexity of the EEG signals accelerated proportionally to the nicotine content of a cigarette. These results signified a cortical desynchronization from a synchronized level induced by lack of smoking. This desynchronization and stimulation of the central nervous system, improvement of operational behavior, and mood after nicotine absorption make it appear that cortical arousal in a normal extrovert subject occurs from a state of underactivation.

80–0256. Card, J. P.; Mitchell, J. A. **The Effects of Nicotine on Implantation in the Rat.** *Biology of Reproduction* 20(3): 532-539, April 1979.

The effects of nicotine on blastocyst implantation were investigated in the rat. Flushing each uterine horn with a constant force and volume of saline at selected times during pregnancy demonstrated that, in control animals, implantation had progressed sufficiently by 1,200 hours of Day 5 (Day 0 = day of insemination) to prevent conceptus retrieval. In contrast, in animals receiving twice daily subcutaneous injections of nicotine (5 mg/kg body weight/injection), blastocysts could be retrieved as late as 2,100 hours of Day 5. Thus, in nicotine-treated rats, the development of blastocyst attachment sufficient to resist flushing was delayed approximately 9 hours. Simultaneous entrance of fertilized ova into the uterine lumen at 1,800 hours of Day 3 in both control and nicotine-treated animals demonstrated that this delay could not be attributed to altered transport through the oviduct. Associated with the altered time course of implantation in nicotine-treated rats were changes in blastocyst development: the time of zona pellucida loss and the growth of the inner cell mass were delayed. However, the number of blastocysts ultimately implanting in nicotine-treated animals did not differ from that of controls. (Auth. Abs.)

80–0257. Cohen, B. S. **The Magnitude, Lung Distribution and Significance of the Polonium-210 Inhaled in Cigarette Smoke.** Doctoral Dissertation, New York University, Department of Environmental Medicine, University Microfilms 79-18946, 1979, 165 pp.

This work evaluates the distribution of the radionuclide polonium-210 deposited in human lung by cigarette smoke and examines its possible significance with respect to lung cancer. The alpha activity was found to be associated with the relatively insoluble fraction of cigarette smoke tar. The cigarettes tested were produced from two types of USDA tobacco, Tennessee Burley (TN) and North Carolina Bright (NC). Perfusion of the tar with physiological saline resulted in no change in the mean measured activity, but there was more variability in the measured values for the perfused tar than for the initial tar samples. An upper limit of 20 percent and 16 percent loss of activity for TN and NC respectively may be estimated. A minimum of 85 percent of the alpha activity of the tar is from polonium-210. This low solubility confirms the possibility of long-term retention of this radioactive nuclide in the lung. The retention of polonium-210 in the lung was corroborated by radiochemical analyses of partitioned human lung specimens. Polonium-208 was used in each individual sample to determine chemical yield. The parenchymal concentration, P, is significantly higher in smokers than in nonsmokers; 9±2 and 3±1 fCi g^{-1} respectively. Measured concentrations of polonium-210 in the tracheobronchial tree, T, 10±4 fCi g^{-1} for smokers and 7±1 fCi g^{-1} for nonsmokers are not significantly different. The ratio, T/P of the concentration of polonium-210 measured in one compartment to that in the other is lower in smokers, $T/P = 1.1 \pm 0.2$, than in nonsmokers, $T/P = 2.7 \pm 0.7$, indicating retention of the inhaled nuclide in the lung parenchyma in smokers. The average concentration of the polonium-210 which results from inhaled activity retained in the lung parenchyma in smokers is estimated at approximately 5 fCi g^{-1}. Analyses of the lungs of a few ex-smokers showed alveolar concentrations similar to those in smokers. A new technique was developed to measure ultra low alpha activity on the bronchial mucosa, utilizing cellulose nitrate track etch film detectors. The lower limit of detection of this system for an individual sample is 0.15 fCi cm^{-2} when detectors are exposed to about one square centimeter of bronchial surface for 100 days. Measurements to determine the alpha activity along the bronchial mucosa were made in five individuals. A few areas of slightly elevated activity were measured in each of the lungs except for that of a young smoker, and one area of markedly elevated activity was measured in the lung of a smoker. From the multiple measurements on each bronchial tree a mean activity could be determined for each individual. The means ranged from 0.01±0.06 fCi cm^{-2} for a young smoker to 0.26±0.04 fCi cm^{-2} for an older smoker. The mean value for a nonsmoker was 0.02±0.06 fCi cm^{-2}. The average dose rates to the bronchial epithelium from the alpha activity estimated for each of these five individuals range from 0.1 to 2.0 millirad per year. Some areas of the bronchial tree are exposed to slightly higher dose rates (2-6 millirad per year). In addition, the small areas of elevated activity could deliver an annual dose of approximately 1 rad to the basal cells of the bronchial epithelium. The risk from these dose rates is evaluated in terms of the lung cancer experience of underground miners. (Auth. Abs.)

80–0258. Corberand, J.; Nguyen, F.; Do, A. H.; Dutau, G.; Laharrague, P.; Fontanilles, A. M.; Gleizes, B. **Effect of Tobacco Smoking on the Functions of Polymorphonuclear Leukocytes.** *Infection and Immunity* 23(3): 577-581, March 1979.

Eight tests investigating the function of circulating polymorphonuclear leukocytes were performed in 68 subjects. The subjects were divided into two groups: those who did not smoke and were not particularly exposed to passive inhalation of cigarette smoke (2 males, 32 females), and those who smoked at least 20 cigarettes per day (14 males, 20 females). In the latter group, 28 subjects smoked filter cigarettes, among which 4 used Virginia tobacco and 24 used black tobacco. The six nonfilter cigarette users smoked black tobacco. Among the whole smoker population, 30 inhaled the tobacco smoke and 18 had smoked one cigarette within 1 hour before blood withdrawal. To determine the effects of heavy smoking, the tests were performed in 10 smokers before and after smoking five consecutive cigarettes in a period not exceeding 60 min. Comparison of the two groups allowed determination of the in vivo effect of tobacco smoke on the nonspecific defense system of the body. Ingestion ability, oxygen consumption, and bactericidal activity were normal in smokers. Myeloperoxidase and neutrophil alkaline phosphatase activities also were unchanged. The nitroblue tetrazolium reduction and the serum lysozyme levels were slightly increased in smokers. The capillary tube random migration was depressed, and intensive smoking further aggravated this change. It is suggested that tobacco smoke acts directly on one (or several) unidentified target sites of polymorphonuclear leukocytes. This impairment, demonstrated in vivo, probably plays a role in the genesis of the bronchopulmonary diseases so frequent in heavy smokers. (Auth. Abs. Mod.)

80–0259. Dax, E. M.; Clappison, B. H.; Pullan, P. T.; Pepperell, R.; Johnston, C. I. **Individual Neurophysin Concentrations in the Pituitary and Circulation of Humans.** *Clinical Endocrinology* 10(3): 253-263, March 1979.

Specific, homologous human neurophysin I and II radioimmunoassays were established and used to measure the individual, immunoreactive neurophysin concentrations in human plasma. Circulating levels of human neurophysin I in normal individuals were less than 1 ng/ml and neurophysin II levels were 1-2 ng/ml. Eleven habitual or occasional smokers abstained from smoking for 12 hours prior to the test. All subjects were ambulatory and taking fluids ad libitum. Samples were withdrawn 20 minutes and 5 minutes prior to smoking two cigarettes with high nicotine content (1.2 mg nicotine/cigarette) over 20 minutes. Samples were withdrawn at 5 minute intervals for 40 minutes after smoking was commenced. Nicotine inhalation in 2 subjects out of 11 resulted in a marked increase in plasma neurophysin I without any detectable rise in plasma neurophysin II, indicating independent release of neurophysin I in response to this stimulus. These two subjects were the only ones to experience nausea and vasovagal symptoms during smoking, suggesting that nicotine inhalation caused release of neurophysin I via nausea and vagal reflexes rather than by direct hypothalamic stimulation. In the other nine subjects, plasma neurophysin I levels remained undetectable during smoking, while plasma neurophysin II levels showed no significant change. The mean plasma neurophysin II level before smoking was 2.2±0.4 ng/ml and was 1.9±0.3 and 2.32±0.3 ng/ml 20 and 40 minutes after the commencement of smoking. Neurophysin levels were also affected by conditions like dehydration, hemorrhage, pregnancy, oral contraception, lactation, renal failure and oxytocin release.

80–0260. Domir, S. C.; Foy, C. L. **Movement and Metabolic Fate of (^{14}C)Ethephon in Flue-Cured Tobacco.** *Pesticide Biochemistry and Physiology* 9(1): 9-22, August 1978.

The absorption, distribution, and metabolic fate of (^{14}C) ethephon in flue-cured tobacco (*Nicotiana tabacum* L.) was studied using autoradiography, thin-layer chromatography, high-voltage paper electrophoresis, and liquid scintillation spectrometry. Labeled ethephon penetrated mature leaf tissue easily and was translocated primarily in an acropetal direction. No ^{14}C activity was detected in any other plant part except the treated leaf. The first day after treatment, most of the translocated ^{14}C was detected in the midrib, and after 2 days radioactivity was noticed in veinal areas distal to the point of application. Four days later, however, ^{14}C was detected in slight amounts only in the midrib, indicating that (^{14}C)ethephon was rapidly degraded by the leaf tissue. Depending on leaf position on the stalk, as much as 92 percent of the radioactivity had disappeared from the leaf tissue during the first day after treatment, and as little as 5 percent of the applied radioactivity was recovered 4 days later. Methanol-extracted plant residues contained insignificant amounts of ^{14}C. All of the ^{14}C in methanol extracts was present in the form of a labeled compound with an R_f value corresponding to that of ethephon, indicating the absence of any detectable metabolites of the parent compound. Smoke analysis of cigarettes showed that more (^{14}C)ethylene than $^{14}CO_2$ was recovered in the mainstream, whereas the trend was reversed in the case of sidestream smoke. (Auth. Abs.)

80–0261. Elinder, C.-G.; Kjellstrom, T.; Linnman, L.; Pershagen, G. **Urinary Excretion of Cadmium and Zinc Among Persons From Sweden.** *Environmental Research* 15(3): 473-484, June 1978.

Cadmium and zinc concentrations were measured in the urine of 130 Swedes, including 50 pairs of identical twins. Atomic absorption spectrophotometry procedures were developed for the analysis. Of the subjects, 108 were male (96 nonsmokers, aged 2 to 89, and 12 smokers, aged 40 to 75) and 22 were female (all nonsmokers and between 3 and 59 years of age). Cadmium concentration increased with age ($p<0.01$) and was higher among smokers than nonsmokers and among women than men ($p<0.01$). Estimated 24-hour excretion of cadmium among nonsmokers increased from about 0.25 to 0.40 μg in persons from 20 to 70 years old. The 24-hour cadmium excretions among nonsmokers in different age groups fitted better to total kidney burden than to daily cadmium intake from food. Zinc excretion, on the other hand, decreased after the age of 20. Smokers tended to have higher zinc values than nonsmokers and men had higher values than women, but these differences were not statistically significant. (Auth. Abs. Mod.)

80–0262. Gleich, G. J.; Welsh, P. W. **Immunochemical and Physicochemical Properties of Tobacco Extracts.** *American Review of Respiratory Disease* 120(5): 995-1001, November 1979.

Because of the suspicion that antigens may be present in tobacco, extracts of green tobacco leaf, cured tobacco leaf, and cigarette smoke condensate were prepared and their physicochemical and immunochemical properties investigated. Analyses by isoelectric focusing revealed as many as 20 bands with green leaf extract; with cured leaf extract and smoke condensate extract there was a marked decrease in the number of bands. The pattern for smoke condensate extract was similar to that for cured leaf extract. Immunization of rabbits and guinea pigs with green leaf extract yielded antiserums that produced at least 10 bands with this material; these antiserums reacted weakly with cured leaf extract and not at all with smoke condensate extract. Antiserums to cured leaf extract reacted weakly with both cured and green leaf extracts and not to smoke condensate extract. Serum specimens from 6 rabbits immunized 3 times with 1 mg of smoke condensate extract in Freund's complete adjuvant at monthly intervals and reimmunized 8 months later by intravenous injection of smoke condensate extract produced precipitin bands when reacted with green leaf and cured leaf extracts. After isoelectric focusing of green leaf extract, immunofixation analysis using rabbit anti-green leaf extract showed that the antigens were acidic proteins with isoelectric points of 4 to 5. These results indicate that all of these tobacco extracts contain antigens able to stimulate antibody formation in laboratory animals. (Auth. Abs.)

80–0263. Grygiel, J. J.; Wing, L.M.H.; Farkas, J.; Birkett, D. J. **Effects of Allopurinol on Theophylline Metabolism and Clearance.** *Clinical Pharmacology and Therapeutics* 26(5): 660-667, November 1979.

The effects of allopurinol on the plasma clearance and metabolism of theophylline in man were investigated in 10 subjects (5 smokers, 5 nonsmokers) under single dose and in 4 subjects (all nonsmokers) under multiple dosing conditions. No change in theophylline clearance was found with the concomitant use of allopurinol in smokers, nonsmokers, or the group as a whole, but 1-methylxanthine (1MX), a theophylline metabolite not previously described in man, was detected in urine during control and allopurinol treatment phases under both single and multiple dosing conditions. Excretion of 1MX increased at the expense of 1-methyluric acid (1MU) during the allopurinol treatment phase. It is proposed that the secondary biotransformation of 1MX to 1MU is mediated by xanthine oxidase. The patterns of urinary metabolites of theophylline were similar in smokers and nonsmokers.

80–0264. Hagedorn, B.; Kostenbauder, H. B. **Studies on the Effect of Tobacco Smoke on the Biotransformation of Vasoactive Substances in the Isolated Perfused Rabbit Lung. II. Angiotensin I Conversion.** *Research Communications in Chemical Pathology and Pharmacology* 20(1): 195-198, April 1978.

Cigarette smoke administration was previously found to have a pronounced inhibitory effect on metabolism of nicotine and benzo(*a*)pyrene in the isolated perfused rabbit lung. These observations suggest the possibility of tobacco smoke inhibition of biotransformations associated with nonventilatory pulmonary functions such as the biotransformation of endogenous vasoactive substances. However, the present study provides no evidence of an effect of smoke on the conversion of angiotensin I to angiotensin II in the isolated perfused rabbit lung; the differences between control and smoke exposure steady-state angiotensin II concentrations were not significant. (Auth. Abs. Mod.)

80–0265. Harris, C. C.; Autrup, H.; Stoner, G. D.; Trump, B. F.; Hillman, E.; Schafer, P. W.; Jeffrey, A. M. **Metabolism of Benzo(*a*)pyrene, *N*-Nitrosodimethylamine, and *N*-Nitrosopyrrolidine and Identification of the Major Carcinogen-DNA Adducts Formed in Cultured Human Esophagus.** *Cancer Research* 39(11): 4401-4406, November 1979.

The wide variation in the world-wide incidence of esophageal carcinoma suggests that environmental agents including chemicals cause this cancer. Since the interaction between chemical procarcinogens and human esophagus has not been studied previously, the metabolic fate of benzo(*a*)pyrene (BP), *N*-nitrosodimethylamine (DMN), and *N*-nitrosopyrrolidine in cultured nontumorous esophagus from two patients with and six patients without esophageal carcinoma was studied. Esophageal explants were cultured in a chemically defined medium for 7 days prior to adding (^{3}H)BP (1.5 μM), (^{14}C)DMN (100 μM), or (^{14}C)*N*-nitrosopyrrolidine (100 μM) for 24 hours. Radioactivity was found bound to both mucosal protein (BP, DMN, and *N*-nitrosopyrrolidine) and DNA (BP and DMN). The major carcinogen-DNA adducts were: (a) with BP, N^2-(10β-(7β,8α, 9α-trihydroxy-7,8,9,10-tetrahydrobenzo(*a*)pyrenyl)) deoxyguanosine; and (b) with DMN, 7-methylguanine, and 0^6-methylguanine (ratio of 0^6-methylguanine to 7-methylguanine was 0.3). The interindividual variations in binding levels to mucosal DNA were 99-fold for BP and 10-fold for DMN. In the two cases studied, the variation in binding levels among the three major anatomical segments (proximal, mid, and distal) was less than 2-fold. The metabolism of BP into water-soluble metabolites varied among the eight patients from 1 to 68 percent of the total metabolism. The qualitative chromatographic patterns of water-soluble metabolites (sulfate esters (range, 21 to 55 percent), glucuronide conjugates (7 to 37 percent), and glutathione conjugates (24 to 66 percent)) and of organic-extractable metabolites were similar in all patients. Whether or not quantitative differences in carcinogen metabolism and in carcinogen bound to esophageal DNA will play a role in human susceptibility to environmental chemical carcinogens is not as yet known. (Auth. Abs.)

80–0266. Janoff, A.; Carp, H.; Lee, D. K.; Drew, R. T. **Cigarette Smoke Inhalation Decreases α_1-Antitrypsin Activity in Rat Lung.** *Science* 206(4424): 1313-1314, December 14, 1979.

Brief inhalation exposure of rats to three or six puffs of cigarette smoke significantly decreases elastase inhibitory capacity per milligram of α_1-antitrypsin in lung lavage fluid ($p<0.01$). This effect is not observed in ozone-tolerant rats and can be reversed by treating the lung lavage fluid from smoke-exposed rats with reducing agents. Samples of human serum obtained immediately after smoking also show decreased elastase inhibitory capacity per milligram of α_1-antitrypsin. Again, elastase inhibitory capacity can be restored by treatment with a reducing agent. Cigarette smoking may cause emphysema by inactivating α_1-antitrypsin through oxidation. (Auth. Abs.)

80–0267. Jayasundar, S.; Vohra, M. M. **Mechanism of Nicotine-Induced Release of Norepinephrine From Adrenergic Nerve Endings: Is Generation and Propagation of Impulses Necessary?** *Archives Internationales de Pharmacodynamie et de Therapie* 232(2): 202-210, April 1978.

The mechanism of release of ^{3}H-norepinephrine (^{3}H-NE) from adrenergic nerve endings by nicotine was investigated. Tetrodotoxin (5 x 10^{-7}g/ml) abolished the contractile response of adrenergically innervated tissues, namely, rat isolated vas deferens, cat splenic strips, guinea-pig atria to transmural stimulation but not to nicotinic agents, and it did not affect the release of ^{3}H-NE by nicotine from rat isolated vas deferens. Incubation of tissues in low Na$^+$(23 mM) and high K$^+$(50 mM) medium, which is known to inhibit propagation of nerve impulses, did not affect the nicotine-induced outflow of ^{3}H-NE. These observations support the contention that the generation of action potential in the postganglionic sympathetic nerve fibers is not the cause of release of norepinephrine by nicotinic agents. (Auth. Abs.)

80–0268. Jusko, W. J.; Gardner, M. J.; Mangione, A.; Schentag, J. J.; Koup, J. R.; Vance, J. W. **Factors Affecting Theophylline Clearances: Age, Tobacco, Marijuana, Cirrhosis, Congestive Heart Failure, Obesity, Oral Contraceptives, Benzodiazepines, Barbiturates, and Ethanol.** *Journal of Pharmaceutical Sciences* 68(11): 1358-1366, November 1979.

Pharmacokinetic, pathophysiologic, and historic data were systematically collected while monitoring theophylline therapy in adult pulmonary patients and from special studies in pediatric patients, normal volunteers, and patients with other diseases. Total body clearances (Cl$_T$) were estimated by nonlinear computer analysis after infusion dosage or from area under the curve data following single oral or intravenous theophylline doses. The Cl$_T$ values, primarily reflecting the theophylline biotransformation rate, averaged 58 (±30) ml/hr/kg and ranged from 4 to 143 ml/hr/kg. Factors examined for their effect on theophylline Cl$_T$ included age, sex, liver disease, congestive heart failure, obesity, renal function, history of drug, tobacco, marihuana, caffeine, or alcohol use, and pregnancy. The NYBAID (automatic interaction detector) computer program for analysis of variance was employed to determine the order, priority, and combinations of independent variables correlating with Cl_T. The major factors that affected theophylline Cl_T in this population included age, liver disease, smoking status, and congestive heart failure. Cigarette smokers were categorized as nonsmokers (n=107), light smokers (<1 pack/day, n=54), or heavy smokers (>1 pack/day, n=39). Marihuana users were categorized as nonusers (n=177), light (<1 joint/week, n=9), or heavy (>2 joints/week, n=14) users. Age showed strong correlation (r=-0.49) with Cl_T. Much additional variability was accounted for in specific subgroups. For example, young adult (20 to 40 years) marihuana users exhibited highest Cl_T values (83±29 ml/hr/kg) while older patients (>40 years) with liver disease had the lowest metabolism rates (22±10 ml/hr/kg). In particular subject types, sex (in teenagers), oral contraceptives (in smokers), obesity (in young nonsmokers), and barbiturates also affected theophylline disposition. In contrast, several common factors such as chronic theophylline, chronic corticosteroids, caffeine, sex (in adults), and pregnancy did not alter theophylline Cl_T. This unique clinical pharmacokinetic monitoring and statistical approach to drug clearance characterization in patients permits identification of criteria in a format that may be useful for the prediction of drug disposition rates and dosage regimens. (Auth. Abs. Mod.)

80–0269. Kjellstrom, T. **Exposure and Accumulation of Cadmium in Populations from Japan, the United States, and Sweden.** *Environmental Health Perspectives* 28: 169-197, February 1979.

Studies were carried out in Japan, the United States, and Sweden regarding the comparability of analytical methods for cadmium, daily intake of cadmium via food, daily amount of cadmium in feces, concentrations of cadmium in different tissues and the body burden of cadmium, urinary excretion of cadmium, and cadmium concentrations in blood. It was found that the cadmium intake via food among adults is about 35 μg/day in Japan (Tokyo) and about 17 μg/day in the United States (Dallas) and Sweden (Stockholm). It varies with age in a way similar to calorie intake. Body burden increases rapidly with age. The half-time of cadmium is longer in muscles than in liver or kidneys. In the cross-sectional population samples studied (smokers and nonsmokers mixed), the average cadmium body burden at age 45 was about 21 mg in Japan, 9 mg in the United States, and 6 mg in Sweden. Among nonsmokers in the United States and Sweden the body burden at age 45 was about 5 to 6 mg; for those in Japan about 16 to 17 mg. The difference in average body burden for smokers and nonsmokers is explained by differences in smoking habits. Cadmium excretion in urine was closely correlated with body burden, and about 0.005 to 0.01 percent of body burden is excreted daily in urine. Cadmium concentration in the blood was a good indicator of average recent intake over a 3-month period. Neither blood cadmium nor urine cadmium changed immediately after an increase in exposure level. (Auth. Abs.)

80–0270. Mackenzie, J. S.; Flower, R.L.P. **The Effect of Long-Term Exposure to Cigarette Smoke on the Height and Specificity of the Secondary Immune Response to Influenza Virus in a Murine Model System.** *Journal of Hygiene* 83(1): 135-141, August 1979.

The effect of long-term exposure to cigarette smoke on the height and specificity of the secondary humoral immune response to influenza was investigated in a murine model system. It was shown that if mice were pre-immunized with a sublethal infection of influenza virus and then exposed to cigarette smoke equivalent to human consumption of 20 to 30 cigarettes/day for approximately 8 minutes over 36 weeks, they were able to mount a secondary immune response of normal height on subsequent challenge with the homologous virus strain. The response, however, was less specific than that elicited in control mice, with high titers of cross-reacting antibody by hemagglutination-inhibition to the following strain in the same antigenic series. Recall of antibody to the previous strain in the antigenic series was not observed in either control or smoke-exposed animals. These results serve to correct an earlier discrepancy between the murine system and human studies in which the response to influenza infection in mice was depressed by prolonged exposure to cigarette smoke, whereas in man the response of smokers did not differ significantly from that of nonsmokers. This apparent discrepancy had been caused by a lack of previous experience of influenza in the mice, which had mounted a primary response, as compared with the secondary response observed in the human studies. (Auth. Abs. Mod.)

80–0271. Madden, J. J.; Falek, A.; Shafer, D. A.; Glick, J. H. **Effects of Opiates and Demographic Factors on DNA Repair Synthesis in Human Leukocytes.** *Proceedings of the National Academy of Sciences of the United States of America* 76(11): 5769-5773, November 1979.

DNA repair synthesis (DRS) in leukocytes stressed by far UV irradiation was studied in 90 normal individuals, 38 street-heroin addicts, and 18 methadone maintenance patients. Age, sex, coffee use, and alcohol use had no significant effect on the maximal repair synthesis response of the control subjects, but smoking tobacco significantly decreased the mean response and variance when compared with nonsmoking controls. The difference between the DRS means for smokers and nonsmokers was statistically significant ($p<0.002$) when examined by a one-way analysis of variance. The difference between smokers and nonsmokers was the result of a significant decrease in the variance as tested by the F test for the homogeneity of variance of the DRS response ($F=3.15$; $p<0.01$) and not from an increase in "poor repairers" among the smokers. There were too few self-reported marihuana users in the sample for analysis. Coffee was also found to have a slight inhibitory effect on DRS

($p < 0.07$), but multivariate analysis indicated that this finding was due to those coffee drinkers who were also smokers. Nonsmoking coffee abstainers and nonsmoking coffee drinkers had DRS mean scores that were not significantly different from one another (18,700 and 16,800 dpm/A_{260}, respectively). On the other hand, coffee drinkers who were also smokers had a significantly decreased mean DRS score (10,300 dpm/A_{260}, $p < 0.002$). Heroin addiction had an even more pronounced negative effect, and this may be related to the high rate of chromosome aberrations found in this population. Half of the addicts tested were incapable of repairing UV fluences one-quarter as large as those repaired by the control subjects (5 J/m^2 and 20 J/m^2, respectively) in the 2-hour assay period. Long-term methadone treatment ameliorated the effects of the street heroin, just as it resulted in a decrease of the chromosome aberration frequency. (Auth. Abs. Mod.)

80–0272. Marshall, M. **Atherogenese. Experimentelle Untersuchungen zur Pathogenese obliterierender Arteriopathien am Miniaturschwein. [Atherogenesis. Experimental Investigation of the Pathogenesis of Obliterating Arteriopathies in Miniature Pigs.]** *Munchener Medizinische Wochenschrift* 121(38): 1211-1218, September 21, 1979, German.

The development of obliterating arteriopathy (arteriosclerosis) and cardiovascular risk factors were studied in miniature pigs. Twenty-six miniature pigs inhaled cigarette smoke from a smoking machine or air containing 150, 200, or 500 ppm carbon monoxide (CO). The air-CO mixtures were forced through a smoking chamber at a rate of 30 l/min for the first 15 min, then at a rate of 6 l/min for 4 hours/day for 1 to 8 days. The duration of cigarette smoke exposure was 16 days. Maximum CO concentration was 80 ppm in the chamber. Carboxyhemoglobin levels were 5 percent with cigarette smoke, 10 percent with air—200 ppm CO, and 35 percent with air—500 ppm CO. Electron micrographs of the inner walls of the arteries showed an adhesion of thrombocytes to apparently unaffected endothelium after exposure to cigarette smoke or air—200 ppm CO. There was a significant ($p < 0.0125$) increase in blood viscosity after exposure to air—500 ppm CO. Blood viscosity also increased by 9 and 26 percent at a shear rate of 78 and 7.8 sec^{-1}, respectively, after intravenous injection of 0.0521 mg/kg nicotine. A corresponding increase in platelet aggregation suggests that nicotine is an important cofactor in obliterating arteriopathy initiation. An arterial electron micrograph taken after infusion of 10 ml/kg 0.1 percent sodium palmitate solution was almost identical to that obtained after cigarette smoke exposure. In contrast, a normal artery inner wall was found after infusion of oleic acid. An increase in undifferentiated free fatty acids was also determined in these experiments.

80–0273. Minozhedinova, N. S.; Lovkova, M. Ia.; Ibraeva, B. Sh. **Metabolism of (^{14}C)Nicotine in Wild Species of *Nicotiana*.** *Biology Bulletin of the Academy of Sciences of the USSR* 5(3): 299-305, May-June 1978.

Nicotine metabolism was studied in a number of wild species of the genus *Nicotiana*. It was shown that exogenous alkaloid, labeled with ^{14}C on the methyl, as well as in each of the heterocycles individually, is actively metabolized when introduced into the above-ground organs of these species during the period of their vegetative development. As a result of the conversions of exogenous nicotine, anabasine is formed in anabasine species of the genus *Nicotiana*, while nornicotine is formed in nornicotine and certain nicotine species. It can be assumed that the low content of alkaloids in wild species of the genus *Nicotina* is due to the fact that the ability of these species to metabolize the indicated compounds is considerably more pronounced than in cultivated tobaccos. (Auth. Abs.)

80–0274. Morosco, G. J.; Goeringer, G. C. **Pancreatic Elastase and Serum α_1-Antitrypsin Levels in Beagle Dogs Smoking High- and Low-Nicotine Cigarettes: Possible Mechanism of Pancreatic Cancer in Cigarette Smokers.** *Journal of Toxicology and Environmental Health* 5: 879-890, 1979.

Beagle dogs exposed to cigarette smoke for 600 days experienced a significant change in pancreatic elastase levels, as measured in tissue homogenates, compared with their sham-exposed controls ($p < 0.05$). Significantly greater elastase activity was found in the high-nicotine cigarette smokers than in the low-nicotine cigarette smokers ($p < 0.05$). Levels of serum α_1-antitrypsin, an antiprotease capable of complexing the excess elastase, were also investigated. Animals smoking high-nicotine cigarettes had significantly lower serum α_1-antitrypsin activities than controls ($p < 0.05$). Low-nicotine smokers showed α_1-antitrypsin activities that were not significantly different from those of controls. (Auth. Abs. Mod.)

80–0275. Muraki, T.; Tokunaga, Y.; Nakadate, T.; Kato, R. **Inhibition by Cholinergic Agonists of the Prolactin Release Induced by Morphine.** *Archives of Pharmacology* 308(3): 249-254, September 1979.

The cholinergic agonists, pilocarpine, physostigmine, and nicotine, inhibited the prolactin release induced by morphine in male rats in vivo. Pilocarpine also inhibited the release of prolactin induced by β-endorphin or metoclopramide without affecting the basal and haloperidol-stimulated serum prolactin levels. The inhibitory effect of pilocarpine on the morphine-stimulated release of prolactin was antagonized by concurrent administration of atropine but not by atropine methylnitrate or by mecamylamine, while the inhibition by nicotine was antagonized by mecamylamine but not by atropine. The stimulation of prolactin release by morphine and its reversal by pilocarpine were observed after the administration of haloperidol or α-methyltyrosine. These results suggest that the central cholinergic system exerts an inhibitory influence on the prolactin release induced by morphine or β-endorphin and the cholinergic inhibition is not mediated via catecholaminergic neurons. (Auth. Abs).

80–0276. Naquira, D.; Arqueros, L. **Water Intake in Rats Affected by Addition of Nicotine to Drinking Water.** *Acta Physiologica Latino Americana* 28(1): 75-78, 1978.

With the purpose of mimicking the slow and discontinuous self-administration of nicotine involved in cigarette smoking, nicotine was chronically administered to rats by addition of the alkaloid to the drinking water. Nicotine was added to the drinking water as follows: 3 groups of 10 rats were supplied with nicotine solutions of either 24, 48, or 96 mg/l for 14 days, and the animals were observed for 7 days after suppression of the drug. The rats of another group were injected subcutaneously with 2 mg/kg/day of nicotine for 14 days, and the animals were observed for 7 days after suppression of the drug. These injections were applied at 12-hour intervals in volumes of 1 mg/kg of body weight. Nicotine concentration was increased from 1 to 96 mg/l during successive days. Rats supplied with 24 mg/l of nicotine had a water intake similar to their respective controls. Those supplied with 48 mg/l of nicotine had a decrease in water intake ($p<0.01$), but in the lower limit of the 99 percent confidence interval of controls. The group of rats receiving 96 mg/l of nicotine showed an immediate reduction of water intake ($p<0.001$), which was maintained during the 14 days of treatment, but returned to control values at the second day of its interruption. Body weight did not change at the three drug concentrations studied. Rats injected with nicotine showed a water intake similar to their respective controls. However, the body weight of these rats was significantly decreased after 7 days of treatment and remained decreased until the suspension of nicotine administration when body weight returned to control values. The intake of water with nicotine 1 mg/l was similar to that of water without nicotine. However, when the concentrations were increased to 3 mg/l, the rats showed a significant ($p<0.001$) preference for the water without nicotine. Total rejection of the water containing nicotine was observed at concentrations of 48 and 96 mg/l. Nicotine (96 mg/l) in the drinking water produced an immediate decrease in water intake without modifying weight gain. In contrast, rats injected with nicotine for 14 days showed a significant decrease in body weight and no changes in water intake.

80–0277. Orlander, J.; Kiessling, K.-H.; Larsson, L. **Skeletal Muscle Metabolism, Morphology and Function in Sedentary Smokers and Nonsmokers.** *Acta Physiologica Scandinavica* 107(1): 39-46, September 1979.

In a group of 43 sedentary men, 18 smokers and 25 nonsmokers were compared with respect to morphological, metabolic, and functional characteristics of skeletal muscle. The percentage of type I fibers was significantly lower and that of type II B fibers significantly higher in smokers ($p<0.001$ and $p<0.01$, respectively). Fiber areas were almost equal in the two groups. Muscle oxidative capacity was lowered in smokers, as judged from decreased mitochondrial enzyme activities and a lowered fibrillar space mitochondrial volume fraction. Isometric and dynamic strengths were lower in smokers, except at the highest velocity of movement studied. Dynamic strengths expressed in relation to isometric strength were similar at all velocities except the highest, where smokers were relatively stronger. Muscular endurance, measured in short-term isometric and dynamic tests, did not differ in the two groups. It is suggested that the lowered muscle oxidative capacity and strength in smokers may be partly a consequence of the different fiber type distribution. A possibly lower physical activity level and tobacco smoke constituents (e.g., carbon monoxide) may also be instrumental. It is not clear whether the different fiber type distribution in smokers is an effect of smoking per se, or if background factors are responsible. (Auth. Abs. Mod.)

80–0278. Pleban, P. A.; Pearson, K. H. **Determination of Manganese in Whole Blood and Serum.** *Clinical Chemistry* 25(11): 1915-1918, 1979.

Methods for determination of manganese in whole blood and serum with Zeeman-effect flameless atomic absorption spectroscopy are described. These analyses were performed on a twofold or fourfold dilution of the specimen in Triton X-100, 1 g/l. No predigestion or extraction procedures were required. The method of standard additions was used for quantitation. Within-run coefficients of variation for whole-blood manganese were 7.0 and 5.5 percent for 2.29 and 5.67 μg/l, respectively. For determination of serum manganese, coefficients of variation were 10.3 and 5.3 percent for 0.97 and 3.01 μg/l, respectively. Manganese detection limits for the assays were 3.0 pg. Whole-blood manganese concentrations, determined for 60 subjects, yielded a mean (±SD) of 9.03 (±2.25) μg/l. Mean serum manganese concentration, determined for 20 subjects, was 1.82 (±0.64) μg/l. Mean whole-blood manganese concentrations for men vs. women were 9.22 ± 2.15 vs. 8.87 ± 2.35 μg/l, respectively. For smokers vs. nonsmokers, the values were 8.87 ± 2.19 vs. 9.24 ± 2.33 μg/l. No correlation was found between blood manganese concentrations and age, sex, or smoking status. (Auth. Abs. Mod.)

80–0279. Rostenberg, I.; Penaloza, R. **Serum IgG and IgD and Levels in Some Infectious and Noninfectious Diseases.** *Clinica Chimica Acta* 85(3): 319-321, May 2, 1978.

Serum IgG and IgD levels were determined in the following groups: (1) a Mexican group of professional blood donors and patients with acute hepatitis, acute salmonellosis and hepatic cirrhosis; and (2) a non-Mexican group of healthy smokers and patients with cancer (prostate, lung, and gastrointestinal tract) and with benign diseases of the same organs as the cancer patients. IgG was significantly increased in the Mexican groups of patients with hepatitis and hepatic cirrhosis and in the non-Mexican groups in the cancer patients in relation to healthy smokers ($p<0.01$). IgD values showed a wide dispersion in all the groups, which does not allow for comparisons among means. For that reason, linear regression analysis between IgG and IgD was done, the results being significant only in the two groups with infectious diseases (acute hepatitis and acute salmonellosis), which suggest that IgD could be involved in the immune response against their respective pathogenic agents. (Auth. Abs. Mod.)

80–0280. Sato, S.; Ohka, T.; Nagao, M.; Tsuji, K.; Kosuge, T. **Reduction in Mutagenicity of Cigarette Smoke Condensate by Added Sugars.** *Mutation Research* 60(2): 155-161, April 1979.

The effects of adding sugars to high- and low-tar cigarettes on the mutagenicity of their smoke condensates were studied using *Salmonella typhimurium* TA100 and TA98 with and without metabolic activation. The sugars tested were glucose, fructose, galactose, sorbitol, sucrose, and lactose. The lowest mutagenicities observed with these sugars per mg of smoke condensate assayed on TA98 with metabolic activation were 37 percent (high-tar cigarettes) and 22 percent (low-tar cigarettes) of that of smoke condensate from untreated cigarettes. Addition of sugars increased the total amounts of smoke condensates, but the mutagenicities of the total condensates were also decreased by all the sugars, the lowest values being 35 percent (high-tar cigarettes) and 36 percent (low-tar cigarettes) of that of smoke condensates from cigarettes without added sugar. On assay with TA100 with metabolic activation, decreases in both specific and total mutagenicities of condensates of high-tar cigarettes were observed with all the sugars tested except galactose and sucrose. Treatment with glucose, fructose, or sorbitol decreased the specific mutagenicity of condensates of low-tar cigarettes, and glucose and fructose reduced also their total mutagenicity. The effects of added sugars were more marked when assayed on TA98 than on TA100, and of the sugars tested, fructose and sorbitol had the greatest effects. Addition of sugars had no effect on the mutagenicity of cigarette-smoke condensates without metabolic activation. (Auth. Abs.)

80–0281. Schreiber, H.; Bibbo, M.; Wied, G. L.; Saccomanno, G., Nettesheim, P. **Bronchial Metaplasia as a Benign or Premalignant Lesion. I. Cytologic and Ultrastructural Discrimination Between Acute Carcinogen Effects and Toxin-Induced Changes.** *Acta Cytologica* 23(6): 496-503, November-December 1979.

The respiratory tracts of hamsters were exposed to either of two different chemical compounds contained in cigarette smoke, the carcinogenic chemical benzo(*a*)pyrene or the noncarcinogenic chemical formaldehyde. At various times during or shortly after the exposure, the acute cytologic, histologic, and ultrastructural responses to the two agents were compared. Both agents induced microscopically similar squamous metaplastic changes of the tracheobronchial epithelium. However, there were several cytologic and ultrastructural differences in the appearance of the chromatin, nucleoli, and cytoplasm that could be used to separate carcinogen-induced from noncarcinogen-induced acute metaplastic changes. Furthermore, after cessation of the exposures, squamous metaplastic changes induced by the noncarcinogenic agent formaldehyde regressed to normal, while the acute carcinogen-induced changes were replaced by other, more persistent metaplastic changes. (Auth. Abs.)

80–0282. Sindelar, R. D.; Rosazza, J. P.; Barfknecht, C. F. ***N*-Demethylation of Nicotine and Reduction of Nicotine-1′-*N*-Oxide by *Microsporum gypseum*.** *Applied and Environmental Microbiology* 38(5): 836-839, November 1979.

Several microorganisms were examined for their abilities to convert *S*-nicotine into nornicotine. Five microorganisms including *Microsporum gypseum* (ATCC 11395) produced nornicotine and three unknown metabolites. *M. gypseum* efficiently reduced nicotine-1′-*N*-oxide to nicotine, but no nornicotine was obtained when the *N*-oxide was used as substrate. (Auth. Abs.)

80–0283. Sirtori, C.; Paganuzzi, M.; Lombardo, C.; Scalese, S.; Ruzzon, T. **Presenza di Sostanze Mutagene Nelle Urine di Fumatori. [Presence of Mutagenic Substances in the Urine of Smokers.]** *Gaslini* 10(1): 18-20, 1978, Italian.

The mutagenic activity of the urine of smokers (9 men, 3 women) following 200-fold concentration with nonpolar resins was explored. The test of mutagenesis utilized *Salmonella typhimurium* as the test organism. The results were positive for the presence of mutagenic substances in 11 of the 12 subjects (8 men, 3 women). Pooled urine from 12 nonsmokers gave negative results. The age span was the same in the smoker and control group, namely from 25 to 40. (Auth. Abs.)

80–0284. Stuart, B. O.; Palmer, R. F.; Filipy, R. E.; Dagle, G. E.; McDonald, K. E. **Respiratory Tract Carcinogenesis in Large and Small Experimental Animals Following Daily Inhalation of Radon Daughters and Uranium Ore Dust.** U.S. Department of Energy, Technical Information Center, Publication No. BNWL-SA-5910, Oak Ridge, 1977, 5 pp.

Uranium ore miners of the Colorado plateau have high incidence rates of lung cancer (6X), and high mortality rates due to pneumoconiosis and emphysema (5X) than those in the general population. Beagles and Wistar rats were used to study the effect of radon daughter and uranium ore dust exposure on pulmonary diseases. The beagles were exposed to radon daughters with uranium ore dust (Group 1, 20 dogs), radon daughters with ore dust and cigarette smoking (Group 2, 20 dogs), cigarette smoking alone (Group 3, 20 dogs), and sham-smoking (Group 4, 9 control dogs). Minimal changes occurred in the respiratory tracts of the controls. Pulmonary fibrosis was prevalent in all Group 1 and Group 2 dogs. Emphysema was present in all Group 1 and Group 2 dogs. Subpleural cavities, occasionally exceeding 1 cm in diameter, were present in 11 Group 1 dogs and in 12 Group 2 dogs. These cavities appeared several months earlier in Group 2 dogs. The lungs of dogs in Groups 1 and 2 with >40 months exposure histories had large areas of epithelial changes, including large areas of adenomatosis frequently progressing to squamous metaplasia of the alveolar epithelium with atypical cells. At >50 months exposure, lungs from 50 percent of Group 1 and Group 2 dogs contained large cavities within the parenchyma, surrounded by bands of hyperplastic adenomatous epithelial cells. These findings in dogs indicate that radon daughter and uranium ore dust exposure is a severe hazard to uranium miners.

80–0285. Sula, J. **Experimental Data Concerning the Question of the Synergic Action of the Influenza Virus and of Some Chemical Carcinogens in the Pathogenesis of Lung Cancer.** *Neoplasma* 26(1): 17-22, 1979.

The importance of new discoveries in the field of molecular biology concerning the interaction of oncogenic material in the cell leading to a synthesis of knowledge in the field of viral and physicochemical blastomogenesis is underlined. A concise reference to earlier studies and to the present state of knowledge is presented, with a subsequent review of the results of experimental studies dealing with the problem of the synergic action of influenza viruses (type A_2, A/PRS, B, Sendai) and of chemical carcinogens (urethane, carcinogenic hydrocarbons, diethylnitrosamines, cigarette smoke) in the pathogenesis of lung cancer. Both the suggested and already demonstrated mechanisms of the joint action of viruses and chemical carcinogens, illustrating the interrelations between the two factors in the process of malignant transformation, are discussed. (Auth. Abs.)

80–0286. Wetter, C.; Bernard, M. **Identifizierung, Reinigung und serologischer Nachweis von Tabakmosaikvirus und Para-Tabakmosaikvirus aus Zigaretten. [Identification, Purification, and Serological Differentiation of Tobacco Mosaic and Para-Tobacco Mosaic Virus From Cigarettes.]** *Phytopathologische Zeitschrift* 90(3): 257-267, November 1977, German.

Tobacco mosaic virus (TMV) and para-tobacco mosaic virus (PTMV) were identified from 42 different brands of German cigarettes blended of light tobaccos. *Nicotiana sylvestris* and White Burley tobacco were used as local lesion hosts for PTMV. White Burley was a far more sensitive test plant for PTMV than *N. sylvestris*. Virus was purified from cigarettes by precipitation with ammonium sulfate, differential and density gradient centrifugation yielding about 0.1 to 0.29 mg virus per gm of tobacco. Using this procedure, virus from single cigarettes could be separated from brown-colored material. Purified virus from cigarettes had about 50 percent of the infectivity of virus from fresh tissue. Precipitin slide tests were used to determine both viruses. TMV and PTMV in purified preparations could be identified by intragel absorption tests and immune electrophoresis. The amount of TMV in the different brands varied from 0.10 to 0.29 mg/gm tobacco. (Auth. Abs. Mod.)

80–0287. Whitman, P. K.; Awokoya, F. O.; DeCicco, S. G.; Dweck, J. S. **Controlled Release of Carcinogens From Beeswax Pellets.** Oak Ridge Station, School of Chemical Engineering Practice, Massachusetts Institute of Technology, Publication No. ORNL/MIT-244, November 26, 1976, 60 pp.

Polymer-coated and uncoated beeswax pellets were investigated as systems for controlled release of benzo(*a*)pyrene (BaP) in interstitial fluids. The maximum solubility of BaP in beeswax was determined to be 0.65 weight percent. The cumulative rate of release for unsaturated uncoated beeswax pellets was found to be predicted by analytical solution of the equations for mass transfer from a finite cylinder. An approximate solution for the cumulative release rate from unsaturated cellulose acetate polymer-coated beeswax pellets was calculated with identical initial BaP concentrations in both the polymer and the beeswax and the restriction that the partition coefficients were unity. The diffusivity of BaP in unsaturated beeswax was determined to be $3.2 \times 10^{-9} \pm 1.0 \times 10^{-9}$ cm^2/sec. The partition function for BaP between beeswax and serum was noted to be a strong function of concentration, varying from 396 ± 47 to 138 ± 24 for serum equilibrium concentrations of 6.8 and 24.9 μg/ml, respectively. (Auth. Abs.)

80–0288. Wilcox, R. G.; Hughes, J.; Roland, J. **Verification of Smoking History in Patients After Infarction Using Urinary Nicotine and Cotinine Measurements.** *British Medical Journal* 2(6197): 1026-1028, October 27, 1979.

Urinary concentrations of nicotine and its major metabolite, cotinine were measured in 104 volunteers (58 smokers and 46 nonsmokers) to test the reliability of the measurements as indicators of current smoking. In the nonsmokers, detectable concentrations were always below the confidence limits set for the method, while in smokers the concentrations were always above these limits. After subjects stopped smoking, cotinine appeared in the urine for longer than nicotine and was still detectable at least 36 hours after the last cigarette had been smoked. In subjects with a constant daily cigarette consumption (3 to 45 cigarettes per day), the urinary cotinine-creatinine ratio correlated strongly with the number of cigarettes smoked. When the method was used to verify the smoking histories given by patients attending an infarction clinic it was estimated that 46 to 53 percent of previous smokers had actually stopped smoking compared with the 63 percent who said that they had done so. It is suggested that simultaneous assays of urinary nicotine and cotinine may be useful in verifying patients' current smoking habits. (Auth. Abs. Mod.)

80–0289. Yang, J. A.; Huber, S. A.; Lucas, Z. J. **Inhibition of DNA Synthesis in Cultured Lymphocytes and Tumor Cells by Extracts of Betel Nut, Tobacco, and Miang Leaf, Plant Substances Associated With Cancer of the Ororespiratory Epithelium.** *Cancer Research* 39(12): 4802-4809, December 1979.

The high incidence of oropharyngeal, esophageal, and laryngeal cancers in certain parts of the world has been ascribed to conjugated tannins found in certain folk medicinal herbs. Miang leaf and betel nut were extracted with phosphate-buffered saline (0.14 M NaCl, 0.15 M potassium phosphate buffer, pH 7.4) and found that the extracts inhibited (^{3}H)-thymidine incorporation by phytohemagglutinin-stimulated human lymphocytes and by rat mammary tumor and mouse L-cells in logarithmic growth. Pretreating the lymphocytes for 1 or 4 hours with the extracts inhibited phytohemagglutinin-induced thymidine incorporation 72 hours later. At concentrations of 2.5 volumes percent or lower, miang and betel nut extracts inhibited thymidine incorporation by 40 to 98 percent without any apparent signs of toxicity as demonstrated by the ^{86}Rb equilibrium assay. In addition, neither extract inhibited cytotoxicity of rat mammary tumor cells by immune syngeneic spleen cells. The molecular weights of the inhibitory factors were between 1,000 and 10,000 daltons as determined by ultrafiltration and were unaffected by boiling for 3 min or by treatment with alcohol and, therefore, are probably not proteins. This in vitro demonstration of inhibition of DNA synthesis by these plant extracts presumably enriched for conjugated tannins may relate to inhibition of growth of rats and chicks fed conjugated tannin-contaminated sorghum feed. The carcinogenic potential of either these extracts or conjugated tannins is not yet established. (Auth. Abs.)

See also, **80–0386, 80–04[illegible]9**

MORTALITY AND MORBIDITY

80–0290. Cowell, M. J.; Hirst, B. L. **Mortality Differences Between Smokers and Non-Smokers.** State Mutual Life Assurance Company of America, Worcester, Massachusetts, October 22, 1979, 43 pp.

Since the 1964 publication of the Surgeon General's Report *Smoking and Health*, the life insurance industry has had a growing interest in the mortality differentials between smokers and nonsmokers selected for individual coverage. This interest has been heightened with the publication of the Surgeon General's even more comprehensive 1979 Report. This paper traces the background of the 1979 Report, highlights its principal conclusions with respect to mortality, and summarizes the Report's findings regarding socioeconomic and demographic differences between smokers and nonsmokers. The paper then outlines the development of a nonsmoker life insurance policy by the State Mutual Life Assurance Company, and presents the mortality experience between 1973 and 1978 policy anniversaries under State Mutual's nonsmoker business compared to the company's corresponding business issued to individuals who did not meet the nonsmoker criterion. Analysis of the data shows that smokers' death rates are two and one-half times those of nonsmokers and that these differences are found at all ages, but particularly in the younger ranges. Smokers are experiencing 30 percent higher death rates than those shown in the actuarial tables widely used by the insurance industry, while nonsmokers are experiencing only one-half of the estimated mortality. For causes of death such as respiratory cancer, pneumonia, and influenza, the mortality rates for smokers are as much as 15 times higher than for nonsmokers. Death rates for other respiratory disorders and for cardiovascular diseases are consistently two to three times higher for smokers than for nonsmokers. The paper concludes that the differences between the mortality of smokers and nonsmokers are too large to be ignored, and suggests the recognition of these differences in individual life insurance underwriting and pricing. (Auth. Abs. Mod.)

80–0291. Lipfert, F. W. **The Association of Human Mortality With Air Pollution: Statistical Analyses by Region, by Age, and by Cause of Death.** Long Island Lighting Company, May 1978, 274 pp.

A cross-sectional analysis is presented for U.S. urban mortality rates, dealing mainly with the 1969-71 vital statistics for about 200 cities. A review of previous statistical analyses is included, dealing with episode studies, time series analyses, and cross-sectional analyses. The air quality data base developed for the cross-sectional analysis emphasized seven community air pollutants: total suspended particulates (TSP), sulfur dioxide (SO_2), sulfate ion (SO_4), benzo(*a*)pyrene, benzene soluble organics, iron, and manganese; seven other species were more briefly considered. The data base was checked for consistency with other localized measurements and with historical trends in some cities. The historical analysis concluded that previous levels were as much as four times higher in many major cities. In developing the socioeconomic data base to be used in this work, special attention was given to socioeconomic status indicators, including education, housing status, birth rate, and poverty status. Cigarette smoking has been implicated as a contributor to excess mortality in a number of studies. To determine its impact, cigarette consumption data must be obtained. For this purpose an index of cigarette smoking was developed on a statewide basis using tax receipt data adjusted for the differences in tax between adjacent states. A positive correlation between smoking and sulfate air pollution was shown ($p<0.05$), indicating higher cigarette sales in the more polluted areas. An analysis was also made of migration trends and it was concluded that migration was not important for older residents of the more polluted central cities. The mortality rate analysis dealt with total mortality for 1959, 1969, and the average of 1969-71, using data obtained from both States and the Federal Government. Subsets were analyzed according to geographic region, certain socioeconomic variables, 10-year and larger age groups, and certain causes of death. The effects of cigarette smoking were indicated to be much more important than air pollution for both lung cancer and respiratory disease. In general, the regression results for cigarette smoking appeared to underestimate the effects, in comparison to previous studies of mortality differences due to smoking habits. Cigarette smoking was significantly related to mortality in every age group except ages 65 to 74. No combined subsets (age groups within regions or cause of death categories) were analyzed. The results showed a small (5 percent) but statistically significant association between air pollution and urban mortality which was stronger:(1) in 1969 than in 1959; (2) in the Northeast and North Central States; (3) for persons 65 years of age and over; (4) for the nonspecific causes of death (mostly heart disease); and (5) for manganese and total suspended particulates. The analysis of sulfate air pollution was severely compromised by its correlation with other variables, both other pollutants and socioeconomic factors, and statistical significance was shown only in a few isolated cases. Also, using dummy variables to deduce whether a threshold concentration for mortality effects existed showed inconsistent results for sulfate (mid-range concentrations had the largest effect) while manganese showed no threshold and a threshold of 85-130 $\mu g/m^3$ was suggested for TSP. Recommendations for a future analysis using 1980 census data were developed. (Auth. Abs. Mod.)

80–0292. Rubino, G. F.; Piolatto, G.; Newhouse, M. L.; Scansetti, G.; Aresini, G. A.; Murray, R. **Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy.** *British Journal of Industrial Medicine* 36(3): 187-194, August 1979.

This study examines the 1946 to 1975 mortality rates of 952 male North Italian chrysotile asbestos workers employed for at least 30 days between 1930 and 1965. The total number of deaths included 9 attributed to asbestosis and 11 attributed to lung cancer (including 1 suspected case of pleural mesothelioma). Comparison with the national figures for all Italy did not reveal an excess of deaths from lung cancer, but during the last quinquennium of observation (1971-75), the standard mortality ratio for lung cancer rose to 206. Simulation experiments enabled a dust index in fiber/years to be attached to each man in the cohort. All but two of the deaths from lung cancer occurred in the higher exposure group. The relative risk of lung cancer in this group was 2.89. The 11 workers who died from lung cancer were all cigarette smokers. A significant excess of deaths from laryngeal cancer was seen over the whole period of observation. All of the workers dying from laryngeal cancer were smokers and two of them had less than 1 year of dust exposure. The total number of deaths in the study population exceeded expected deaths by approximately 50 percent.

80–0293. Seltzer, C. C.; Friedman, G. D. **Smoking and Mortality. (Letters).** *New England Journal of Medicine* 300(20): 1162-1163, May 17, 1979.

A study of premature mortality rates in middle-aged smokers is criticized by a former coinvestigator of the project (*New England Journal of Medicine* 300: 213-217, 1979). This study is claimed to contain unsatisfactory data, distorted analyses, and unwarranted conclusions. Among the objections to the report are instability of death rates for groups containing fewer than 10 expected deaths; no mention of major inconsistencies with other research, including the Framingham Study; and ignoring contradictions in their own multiple-logistic risk analyses. The authors ignored the results of one of their previous studies, which demonstrated that the value of smoking cessation is overestimated by the current practice of comparing coronary heart disease rates in smokers and ex-smokers, without correcting for baseline differences. Contradictions in the multiple logistic-risk analyses which show that many previously high mortality ratios disappear when covariate factors are examined concomitantly are also ignored. Although the use of constitutional factors as covariables reduced coronary heart disease mortality by 23 percent, the original article dismissed the constitutional hypothesis because it failed to eliminate any differences between smokers and nonsmokers. The constitutional hypothesis warrants a more careful investigation. In reply, the first author of the article in question states that limitations of the study, such as small numbers of deaths, were pointed out. In addition, it is stated that the relation between smoking and coronary heart disease mortality was stronger in this study compared to others because of the relatively young subject age. Further, the constitutional hypothesis was rejected because it did not illustrate the particular detrimental effect of smoking under consideration. Finally, the analysis was undertaken in good faith despite the possibility that the relations between smoking and mortality could be explained by other factors. See also 79-0419.

See also, **80–0298, 80–0312, 80–0317, 80–0320, 80–0373, 80–0377**

80-0294. Bingham, S.; Williams, D.R.R.; Cole, T. J.; James, W.P.T. **Dietary Fibre and Regional Large-Bowel Cancer Mortality in Britain.** *British Journal of Cancer* 40(3): 456-463, September 1979.

The relationship between food intake and cancer of the large bowel was assessed by calculating the average intakes of foods, nutrients, and dietary fiber in the different regions of Great Britain and relating these to the regional pattern of death from colon and rectal cancers between 1969 and 1973. To identify the effect of tobacco consumption, average regional expenditures for tobacco were also included. No significant associations were found with the consumption of fat, animal protein, or beer, nor with current estimates of total dietary fiber intake. Average intakes of the pentose fraction of total dietary fibers, and of vegetables other than potatoes, were negatively and tobacco expenditures positively correlated with the truncated age- and sex-standardized death rates from colon cancer ($r = -0.960$, -0.940, and 0.770, respectively). Specific components of dietary fiber may therefore inhibit colon carcinogenesis. (Auth. Abs. Mod.)

80-0295. Carreras Ruiz, O.; Alert Silva, J. **Cancer Laringeo. Estado Actual del Diagnostico y Tratamiento. [Laryngeal Cancer. The Current Status of Diagnosis and Treatment.]** *Revista Cubana de Cirugia* 15(6): 669-678, November-December 1976, Spanish.

Clinical records of 114 laryngeal cancer patients, in whom 44 superglottic, 68 glottic, and 2 subglottic carcinomas were found, were reviewed. The greatest incidence was reported in men in their sixties with histories of smoking. There were 98 smokers (85.9 percent) among the patients. In 77.2 percent of the patients, the symptom duration was longer than 3 months, and extensive lesions (stages III and IV) were found in 64.9 percent of the patients. The current survival rate was 57.6 percent at 5 years, and radiation was the treatment utilized most frequently. The best results were obtained with T1 and T2 carcinomas, and the combination of radiotherapy and radical surgery was the best therapeutical method. (Auth. Abs. Mod.)

80-0296. Carter, P. K. **Carcinoid Tumors at MMC—1955-1978. Part I—Bronchial.** *Journal of the Maine Medical Association* 70(10): 402-405, October 1979.

A review of the records of 21 patients (7 men, 14 women, ranging in age from 15 to 77 years) with bronchial carcinoids at the Maine Medical Center over the 1955-1978 period indicated several differences in incidence and clinical presentation from those reported in the literature: (1) a disproportionately higher incidence of carcinoids of the bronchi than of other sites, (2) a significantly higher percentage of females, and (3) a significantly smaller number of patients with hemoptysis, cough, or pneumonia. Approximately one-third of the patients were smokers. As in the case of other studies, no association between bronchial carcinoids and cigarette smoking was found.

80-0297. Doll, R. **Lung Cancer: Prospects for Control.** *British Journal of Diseases of the Chest* 73(1): 47-48, January 1979.

The following observations provide a basis for a lung cancer prevention program: (1) the rarity of lung cancer in nonsmokers, (2) the absence of a threshold smoking limit below which there are no ill effects, (3) the benefits of smoking cessation are evident within 5 years, and (4) the risks for ex-smokers are determined by the length of time they smoked before stopping. The Department of Health and Social Security in Great Britain has officially discouraged smoking since 1957, and despite a poor public response, three important changes have occurred: (1) men in the top social class (4 percent of the population) smoke less than half the cigarettes they did in 1958; (2) the majority of smokers are now smoking filter-tipped cigarettes, whereas only 2 percent did in 1955; and (3) the amount of tar in the average cigarette has been reduced by 43 percent since 1965. There has also been a general reduction in mortality from lung cancer. Women over 45-years-old were the exception; they experienced an increase in mortality. Three possible future approaches to the prevention of lung cancer are: (1) manufacturing of less noxious cigarettes, (2) governmental restrictions on sales, and (3) health education programs.

80-0298. Health and Welfare Canada. **Canadian Trends in Smoking Related Diseases: Lung Cancer Mortality.** Department of National Health and Welfare, Technical Report Series No. 3, Canada, July 1976, 18 pp.

There is strong evidence that smoking is causally related to lung cancer. However, the longitudinal lung cancer mortality data for the Canadian population suggest that many young and middle-aged Canadians, regardless of sex and for whatever reason, have chosen to ignore this fact. The proportion of deaths due to this disease increased steadily from 1961 to 1973 for both males and females aged 35 years and over. Specifically, the percentage of total deaths due to lung cancer increased for males from 2.9 percent in 1961 to 5.3 in 1973. Similarly, for females, the percentage rose from 0.6 in 1961 to 1.6 in 1973. Of equal interest is the fact that the percentage increases in the 5-year groups were most pronounced for middle-aged (35-64 years) females. Examining the percentage of lung cancer to total neoplasm deaths by sex indicates that, by 1973, more than one-third of middle-age male mortality and 9 percent of middle-age female mortality due to cancer resulted from lung cancer. Middle-age female cancer mortality due to this disease doubled during the 12-year period under examination, and middle-age male mortality increased by 41 percent. Considerable variation in lung cancer mortality rates exists among various regions of Canada. With few exceptions, during the period 1961 to 1973, the age-standardized mortality rate was consistently lower, relative to the national rate, for the province of Quebec and the Atlantic and Prairies Regions. In contrast, consistently higher rates were evident for the provinces of Ontario and British Columbia. An examination of percent change in lung cancer mortality rates between 1961 and 1973 indicates a regional percentage increase for the male population of 132.6 percent, 96.1 percent, 62.8 percent, 55.2 percent, and 40.7 percent for the Atlantic region, Quebec, Ontario, British Columbia, and the Prairie Region, respectively. For the female population, rates increased 138.2 percent in Quebec, followed in order by the Atlantic region (127.7 percent), Ontario (124.2 percent), British Columbia (93.8 percent), and the Prairie Region (59.0 percent).

80-0299. Jussawalla, D. J.; Jain, D. K. **Lung Cancer in Greater Bombay: Correlations With Religion and Smoking Habits.** *British Journal of Cancer* 40(3): 437-448, September 1979.

The resident population of Greater Bombay was analyzed for the incidence of lung cancer and for other variables of possible significance to lung cancer incidence. During a 10-year period from 1964 to 1973, 2,177 lung cancer cases (1,861 males, 316 females) were registered from a population pool consisting of 5.24 million persons (3.07 million males, 2.17 million females). The average annual lung cancer incidence was 13.6 per 100,000 males, but only 3.3 per 100,000 females, age-adjusted to the Standard World Population. The incidence in non-Parsi males (14.0) was almost double the figure in Parsi males (6.8). There was, however, no significant difference in incidence between non-Parsi (3.8) and Parsi females (3.3). Time-trend analyses did not reveal statistically significant differences in the incidence of lung cancer in any particular (male or female) age group. The death certificates data showed that the age-adjusted rates (standardized to the world population) were 11.0 and 3.3 per 100,000, for males and females, in the total population. In a retrospective study, 792 males with lung cancer (42.6 percent of 1,861 male cancer patients) for whom detailed smoking histories were available, were matched for age and community with randomly selected controls. Patients included 81.2 percent who were smokers (70.1 percent, bidi smokers; 19.6 percent cigarette smokers). The majority were Hindus. A significant statistical association was found between tobacco smoking and lung cancer ($p<0.001$). All smokers appeared to be at high risk (16.8) compared with nonsmokers. The relative risk in bidi smokers (19.3) was even higher than in cigarette smokers (8.6). Hindu, Muslim, and Christian smokers were apparently at identical risks. A dose—response relationship was found in bidi and cigarette smokers. Lung cancer patients smoked a significantly greater number of bidis (18.99) and cigarettes (19.89) than smokers in the control groups (12.73 bidis and 12.43 cigarettes), $p<0.001$. Lung cancer patients had also smoked bidis for a significantly longer period (24.94 years) than bidi smokers in the control group (18.03 years), $p<0.001$. Cigarette smokers among the lung cancer patients had been smoking for a significantly longer period (23.15 years) than those among the controls (19.56 years), $p<0.05$. (Auth. Abs. Mod.)

80-0300. Kakande, I. **Primary Lung Carcinoma in Kenyatta National Hospital, Nairobi.** *East African Medical Journal* 56(7): 325-330, July 1979.

A retrospective survey of bronchogenic carcinoma in 29 patients admitted to Kenyatta National Hospital (KNH) during the 3-year period 1975-77 is presented. The average age of the patients was 52.4 years, and the male:female ratio was 3:1. The majority of the patients had cough, hemoptysis, and chest pain. Of the 18 patients whose smoking habits were recorded, 10 were tobacco smokers. Among these, 6 had smoked 6 to 20 cigarettes per day for 20 to 32 years. Only 2 (6.9 percent) of the patients had resectable tumors. Eleven patients (37.9 percent) died while hospitalized. (Auth. Abs. Mod.)

80-0301. Kizilbash, M.; Mettlin, C.; Sciandra, R.; Murphy, G. P. **Community Cancer-Screening Clinic. Evaluation of Experiences.** *New York State Journal of Medicine* 79(11): 1703-1707, October 1979.

The results of an American Cancer Society (ACS) sponsored community cancer screening clinic in New York State are presented. In April 1978, the Erie County ACS Unit, Buffalo-area hospitals, and participating volunteer physicians cooperated in offering a series of free cancer screening clinics which were open to anyone aged 21 or older. The public was notified of the clinics through public service radio and television advertising, news releases, newspaper articles, and community center flyers and posters. Special efforts were made to reach the high-risk elderly population. Examinations included a history, physical examination, urinalysis, hemoccult test, and Papanicolaou smear. The results from one of these clinics are presented. Of a total of 517 subjects who attended, 75 percent were over the age of 40, 65 percent were female, 68 percent were married, and 64 percent had at least completed high school. More females (53 percent) than males (32 percent) were nonsmokers, and more males (34 percent) than females (17 percent) were ex-smokers; 15 percent of males, and 11 percent of females were smoking at least 1 pack of cigarettes per day. Abnormal clinical findings were discovered in 109 of the 180 males, and in 182 of the 337 females. From a followup survey of 146 of the subjects, it was learned that patient reaction to the clinics was highly favorable. While the educational value of the clinics appeared to be significant with regard to breast self-examination, only 8 of the 39 smokers reported that they were smoking less.

80-0302. Kono, S.; Ikeda, M. **Correlation Between Cancer Mortality and Alcoholic Beverage in Japan.** *British Journal of Cancer* 40(3): 449-455, September 1979.

Geographical correlations between standardized mortality ratios (SMRs) of cancers and consumption of different types of alcoholic beverages (sake, synthetic sake, shochu, beer, wine, and whisky), of cigarettes, and urbanization were examined for all 46 prefectures in Japan. Statistically significant correlations were seen between cigarette smoking and lung cancer in males ($r = 0.35$, $p < 0.05$) and breast cancer in females ($r = 0.55$, $p < 0.001$), but not between smoking and cancer in other sites. Suggestive correlations were observed between cancer of the esophagus in males and both shochu and whisky ($r = 0.27$ and 0.22 respectively), between cancer of the rectum in males and wine ($r = 0.45$), and between cancer of the prostate and shochu ($r = 0.50$). These correlations were also confirmed in the partial correlations between SMRs of cancers and consumption of alcoholic beverages, controlling for the two variables urbanization and consumption of cigarettes. Although cancers of other sites were also correlated with certain types of alcoholic beverages, their associations seemed to be secondary to other factors. The validity of higher-order partial correlations and the problems of correlation study are also referred to. (Auth. Abs. Mod.)

80-0303. Korsgaard, R.; Simonsson, B. G.; Stiksa, G. **Vad ar Lungcancer? [What is Lung Cancer?]** *Kvartalsskrift* 73(3): 79-87, 1978, Swedish.

From 1960 to 1975 the number of deaths due to lung cancer more than doubled in Sweden, while the total number of deaths only increased 17.5 percent. Deaths due to lung cancer are not limited to the higher age groups. In 1975, 3 men and 2 women aged 25 to 29 years, 14 men and 10 women aged 40 to 44, and 85 men and 30 women aged 50 to 55 died from lung cancer. The increased tobacco consumption among women is beginning to result in an enormous increase in lung cancer cases. Between 1960 and 1975 the number of women who died of lung cancer more than doubled, from 211 in 1960 to 492 in 1975. In 1976, at the Clinic of Lung Medicine in Lund, Sweden, 42 percent of the cases were treated for malignant tumors. The mortality rate is expected to increase rapidly during the coming 25 to 30 years, which is regarded as the latency period for lung cancer.

80-0304. Kreyberg, L. **Yrkesbetinget Lungekreft. En Vurdering. [Occupational Lung Cancer. An Evaluation.]** *Tidsskrift for den Norske Laegeforening* 99(12): 570-574, 596, April 30, 1979, Norwegian.

For the evaluation of the carcinogenicity of occupational carcinogens the use of the ratio Group I:Group II tumors (squamous cell and small cell anaplastic carcinomas: adenocarcinomas and most other types), as well as the male:female ratio as epidemiological analysis methods are advocated. The Group I:Group II ratio in Norway has risen from <1:1 over the years as well as the lung cancer mortality rate (from 2 per 100,000 males in the mid-1930s in both urban and rural areas to 50 and 20 respectively in the mid-1970s). The health effect of tobacco with occupational carcinogens is additive or multiplicative. Based on a large national sampling of lung cancer patients it was estimated that in a hypothetical nonsmoking population in 1950-64 the total male:female ratio for lung cancer would be 1.2:1 with the Group I:Group II ratio being 0.3:1 for males and 0.1:1 for females. With some occupational carcinogens even a moderate tobacco consumption resulted in a significant increase of the lung cancer rate. The tobacco consumption of farmers and school teachers was 13.8 g/day and the Group I:Group II ratio 0.9:1 and 0.8:1. The daily tobacco consumption for metal workers was 15.7 g and for carpenters/masons/ painters 17.3 g. The Group I:Group II ratios were 5.8:1 and 6.4:1 respectively.

80-0305. Kreyberg, L. **Lung Cancer in Workers in a Nickel Refinery.** *British Journal of Industrial Medicine* 35(2): 109-116, May 1978.

This study is an analysis of the occurrence of lung cancer in nickel workers, particularly with regard to development time, histological types, and tobacco smoking, in addition to specific exposure to nickel dust and fumes. The series consists of 44 cases of lung cancer occurring during the years 1948-74 in people currently or previously employed at Falconbridge nickel refinery. A 7-year period of reduced activity during the war enables lung cancer in workers who took up employment in 1927-39 to be compared with that in workers who started in or after 1946. Four smokers and four nonsmokers had Group II tumors (adenocarcinoma and other histological types). The mean age at diagnosis is consistent if the development time is related to the length of tobacco smoking. Tobacco smoking is an important factor in the development of lung cancer in nickel workers, and under the conditions described in this study the reduced carcinogenic influence may be attributable to reduced exposure to nickel and possibly also to tobacco. (Auth. Abs. Mod.)

80-0306. Kunze, M. **Neue Wege zur Bekampfung des Bronchuskarzinoms. [New Methods of Controlling Bronchial Carcinoma.]** *Mitteilungen der Osterreichischen Sanitatsverwaltung* 79(7/8): 1-3, 1978, German.

New possibilities for controlling bronchial carcinoma are discussed. These possibilities appear to be in the areas of prevention and early detection. It is generally recognized that cigarette smoking is the most significant factor contributing to lung cancer. In Austria, as elsewhere, the risk of lung cancer is greater for those who have smoked longer than 10 years than for nonsmokers, particularly for cancers of the Kreyberg I and Kreyberg II diagnostic groups. Changing the smoking habits of the Austrian population is the most important measure for preventing lung cancer. Already, publicity about smoking has resulted in reducing the number of male smokers and in increasing consumption of relatively less harmful ("light") cigarettes, without an increase in total cigarette consumption. It is anticipated that the mortality from lung cancer among males will decrease during the next decade. However, a slight increase in the incidence of lung cancer is expected among females. Continued efforts are needed to modify smoking behavior and, more importantly, to encourage abstinence from tobacco smoking among those who have not yet begun to smoke, as well as to control production and sales of cigarettes. Suggested measures include technological control of the manufacture of tobacco products and taxation of cigarettes according to their content of harmful substances. The 20x20 program of medical care for heavy smokers, which includes the search for tobacco-associated diseases and withdrawal therapy, could be used for the early detection of bronchial carcinoma. This new type of medical service belongs to the branch of "Life style medicine." The program could be used with ambulatory and inpatient facilities. Utilization of the existing tuberculosis facilities for the control of lung cancer has already begun in Vienna.

80-0307. Kunze, M.; Vutuc, C. **Teer-Exposition bei Tabakassoziiertem Lungen- und Blasenkrebs. [Tar Exposure of Patients with Smoking-Associated Lung and Bladder Cancers.]** *Munchener Medizinische Wochenschrift* 121(32/33): 1041-1042, August 10, 1979, German.

A survey of smoking habits was made among 200 male patients selected at random from 500 lung cancer patients and 150 bladder cancer patients. Significantly more lung cancer patients (95 percent) were smokers than bladder cancer patients (87 percent), $p<0.01$. The respective average age of each group was 63.2 and 68.7 years. The lung cancer patients had been smoking for 40.2 years and the bladder cancer patients had been smoking for 42.7 years. The bladder cancer patients had smoked cigarettes with tar contents >25 mg significantly more often ($p<0.01$) than the lung cancer patients.

80-0308. LaRiviere, W.; Pickett, A. B. **Clinical Criteria in Diagnosis of Early Squamous Cell Carcinoma of the Lower Lip.** *Journal of the American Dental Association* 99(6): 972-977, December 1979.

A survey of 22 cases of precancerous and cancerous lesions of the lower lip is presented. Subjective symptoms, clinical data, and definitive microscopic diagnoses are correlated. Most carcinomas appeared in occult and noncharacteristic forms. Most of the patients (85 percent) smoked excessively; only 4 smoked a pipe. Of the 15 patients who indicated a preferred side for holding the cigarette or pipe, squamous cell carcinoma developed at that site in 4 patients. Results indicate that squamous cell carcinoma of the lower lip may be unsuspected and undiagnosed in its early states. Recognition of subtle clinical changes may lead to a greater incidence of correct diagnosis and early treatment. (Auth. Abs. Mod.)

80-0309. Lawther, P. J.; Waller, R. E. **Trends in Urban Air Pollution in the United Kingdom in Relation to Lung Cancer Mortality.** *Environmental Health Perspectives* 22: 71-73, February 1978.

The substantial reduction in air pollution, and particularly in components such as benzo(*a*)pyrene in urban areas of the United Kingdom during the past few decades has presented an opportunity to consider further the possible role of carcinogens in the air in relation to lung cancer. While the overall trends in lung cancer mortality have undoubtedly been dominated by changes in smoking, the marked contrasts that at one time existed between these death rates in urban and rural areas have gradually diminished. This may indicate that air pollution contributed appreciably to the urban/rural differences in lung cancer at one time, but it is still difficult to disentangle any effects it may have had from those of changing smoking habits. (Auth. Abs.)

80-0310. Lessard, R.; Reed, D.; Maheux, B.; Lambert, J. **Lung Cancer in New Caledonia, a Nickel Smelting Island.** *Journal of Occupational Medicine* 20(12): 815-817, December 1978.

An epidemiologic study of lung cancer and its possible association with nickel refining was conducted in New Caledonia. This study was stimulated by a review of cancer incidence and mortality in 11 South Pacific countries and territories which indicated that New Caledonia had one of the highest lung cancer rates. Medical records were reviewed to ascertain new cases of lung cancer diagnosed between 1970 and 1974. The residences of the cases were classified according to three geographic zones, based on their distance from the nickel smelter. A control group was obtained from male patients, aged 35 or older, who were seen at a hospital laboratory during the summer of 1975 and from whom personal information (occupational exposure, smoking habits, residential history, and demographic characteristics) was obtained by interview. Since most of the study cases were deceased, this information had to be obtained from medical charts, death certificates, administrative files, and from nickel company records. During the 1970-74 period, 92 new cases of lung cancer were diagnosed. Of these, 67 percent were confirmed pathologically and 33 percent were confirmed clinically and radiologically. The incidence of lung cancer was found to increase inversely to the distance from the nickel smelter. A case-control analysis of the association of lung cancer, smoking, and nickel exposure was made in 68 of the 81 male cases for whom smoking information was available. Of the lung cancer cases, 29 were nickel workers (28 smokers, 1 nonsmoker), and 39 were non-nickel workers (38 smokers, 1 nonsmoker). Of the control cases, 22 were nickel workers (20 smokers, 2 nonsmokers), and 87 were non-nickel workers (49 smokers, 38 nonsmokers). Statistical analysis of the data indicated the following: (1) Lung cancer and nickel occupation were significantly associated ($p<0.05$) independently of the effects of age and cigarette smoking; the relative risk was 3.0. (2) Lung cancer and cigarette smoking were significantly associated ($p<0.05$) independently of the effects of age and nickel occupation; the relative risk was 22. (3) There was no significant interaction between cigarette smoking and nickel occupation which would increase the relative risk of each variable separately.

80–0311. National Academy of Sciences. **Health Effects of Alpha-Emitting Particles in the Respiratory Tract. Report of the Ad Hoc Committee on "Hot Particles" of the Advisory Committee on the Biological Effects of Ionizing Radiations.** National Academy of Sciences, National Technical Information Service Publication No. PB-273 111, October 1976, 129 pp.

In animals, all experimental data so far obtained indicate that when insoluble plutonium particles are inhaled, the major radiation dose in the lungs occurs in the pulmonary, i.e., alveolar, region. The principal delayed effect in the lung of breathing these particles is induction of alveolar cancers. These animal studies have prompted speculation about mechanisms of cancer induction in the respiratory system in man. For alpha irradiation, as well as for exposure to dusts and chemical agents in man, alveolar and bronchiolo-alveolar tissues appear to be very much more resistant to cancer induction than the bronchial epithelial cells. Some possible explanations for the special sensitivity of the bronchial epithelium in man are (1) the bronchial epithelium is more rapidly renewed by cell division than are the alveolar epithelium or connective tissue elements and radiosensitivity is related to this quality; (2) most occupational bronchial cancers have been observed in cigarette smokers who had chronic bronchitis and who had been exposed to irritants in smoke; (3) human populations are commonly and repeatedly exposed to viral infections of the respiratory epithelium which cause rapid loss of epithelial cells, requiring rapid proliferation of stem cells to repopulate the mucosa; and (4) other inhaled pollens, dusts, and gases in the general environment may contribute to bronchial irritation. Regardless of the reasons for this sensitivity, the tissue at special risk in man from inhaled transuranic elements is probably the bronchial epithelium, especially in the more proximal regions of the bronchial tree.

80–0312. National Cancer Institute; National Institute of Environmental Health Sciences; National Institute for Occupational Safety and Health. **Estimates of the Fraction of Cancer in the United States Related to Occupational Factors.** National Cancer Institute, National Institute of Environmental Health Sciences, National Institute for Occupational Safety and Health September 15, 1978, 49 pp.

The fraction of cancer in the United States that is related to occupational factors is estimated after considering recent evidence and taking into account the full consequences of past and present occupational exposures. Current theories suggest that most cancers have multiple causes. Alcohol itself appears to be a minor cause of cancer, but, when combined with cigarette smoking, leads to risks 15 times higher than those experienced by nonsmoking, nondrinking persons. A large proportion of cancers which appear to be attributable to smoking could also be attributable to asbestos, radiation, and other occupational factors. Cancer incidence is strongly dependent on age and duration of exposure; many industrial studies therefore detect only those cancers that develop early. Past exposure to asbestos is expected to result in about 2 million excess cancer deaths in the next 30 years (about 13 to 18 percent of the total cancer mortality expected for that period). Projections of future consequences of past exposure to established carcinogens suggest that occupationally related cancers may comprise as much as 20 percent or more of total cancer mortality in the future. Although occupational exposure to some of the more well-known carcinogens has been reduced, other occupational carcinogens remain unregulated, and a number of occupations are characterized by excess cancer risks which have not yet been attributed to any specific agent. Two alternate approaches to estimating the possible contribution of occupational exposure to human cancer incidence are presented. The first analyzes total cancer incidence (mortality) trends in the U.S. population, subtracting smoking-related cancers from the total to arrive at occupationally-related cancers. However, not all smoking-related cancers (i.e., lung, pancreas, bladder) are attributable to smoking. The fraction of lung cancer not attributable to smoking is increasing, and total cancers, possibly industrially related, have been increasing more rapidly in recent years than in the two decades from 1950 to 1970. A second approach is to compare cancer incidence in men and women: a higher incidence in men would tend to support the concept of industrial risk. Patterns and trends in total U.S. cancer incidence and mortality are consistent with the hypothesis that occupationally related cancers comprise a substantial and increasing fraction of total cancer incidence. The conclusion that a substantial fraction of all cancers (20 to 40 percent) in the United States is occupationally related is not inconsistent with the conclusion that a substantial fraction of cancers is also associated with other factors, such as cigarette smoking and diet.

80–0313. Nikolov, I.; Stoianov, I.; Stoichev, I.: Draganov, I.; Petkova-Bocharova, T.; Chernozemski, I. **Tyutyunopushene, Profesiya i Zlokachestveni Tumori Vsred Selsko Naselenie na Vrachanski Okr'g. [Tobacco Smoking, Profession and Malignant Tumors in the Rural Population of Vratca District.]** *Onkologiia* 14(2): 61-66, 1977, Russian.

The influence of tobacco smoking and occupation on the development of malignant tumors was studied in 27 villages of the Vratca district. A total of 2,321 persons of both sexes, residents of the villages Pudriya and Golyamo Pestene, and 1,083 patients with malignant tumors registered during the period 1965-74 were questioned. The percentage of male smokers was 42.3 ± 3.4 and of female smokers 0.5 ± 0.4. It was found that tobacco smoking in men increases the risk of lung cancer (4.2 times), bladder cancer (2.9 times), labial cancer (3.6 times), and gastric cancer (2.4 times), and has no effect on the risk of developing cancer of the renal pelvis and ureter. In farm workers an increase is found in the risk of skin cancer (5.1 times), cancer of the renal pelvis and ureter (3.3 times), bladder cancer (3.3 times), gastric cancer (2.1 times), and labial cancer (2.1 times). (Auth. Abs.)

80–0314. Rehnlund, S. O. **Industriell Exposition och Lungcancer. [Industrial Exposure and Lung Cancer.]** *Lakartidningen* 74(37): 3070-3071, 1977, Swedish.

The environmental impact of the smelting plant of Ronnskar, Sweden, is being studied. This report is a survey of some recent results. The study involved all known cases of lung cancer among the personnel since 1928. The proportional lung cancer mortality was 9.0 percent as compared to the corresponding figure of 2.3 percent for all deaths within the male population (average 1969-70) of the county of Vasterbotten. The personnel were mainly exposed to arsenic, but also to sulfur dioxide, fluorides, copper, cadmium, antimony, mercury, nickel, selenium, and tellurium. The latency period from the onset of exposure to the appearance of the first lung cancer symptoms was on the average 32.8 years. Smokers and ex-smokers had the same latency period, 32.2 years, while 9 nonsmoking individuals (of 67) showed a latency period of 37 years. The older the individual on employment, the lower the latency period. In this study, 90 percent of the cancer cases occurred among smokers or ex-smokers, which is in agreement with earlier experience. Among the first 5,000 individuals employed, only 8.7 percent had never smoked, 72.6 percent were smokers, and 18.7 percent were heavy smokers (more than 20 cigarettes per day).

80–0315. Richardson, M. F. **The Problem of Multiple Primary Tumors in Patients With Oral Malignant Disease.** *Otolaryngologic Clinics of North America* 12(1): 155-160, February 1979.

This report considers the occurrence of multiple cancers in patients with oral malignant tumors and reviews relevant studies. In several studies, increased risk of developing second cancers was found in patients with smoking and drinking habits. Studies have shown that the more a patient smoked prior to the primary neoplasm, the greater the risk of developing a secondary neoplasm, even if smoking was stopped after the first diagnosis was made. Others, however, have shown that there is a 6 times greater risk of tumor formation for patients who continued to smoke, implying that smoking cessation can alter the risks of developing a secondary primary neoplasm. Second primary tumor diagnostic criteria, incidence, interval, site, treatment, and results are briefly described.

80–0316. Schuettmann, W. **Die Kombinierte Wirkung von Ionisierender Strahlung und Rauchen bei der Verursachung des Lungenkrebses. [The Combined Effect of Ionizing Radiation and Smoking on the Causation of Bronchial Carcinoma.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 150(3): 243-249, 1978, German.

This is a review of American and foreign epidemiological studies of lung cancer in smoking miners exposed to ionizing radiation from Radon and its decay products. Both earlier American and more recent (1976) Czechoslovakian studies of uranium miners agree that radiation is a decisive risk factor with smoking having an additive effect. This combined effect of radiation and smoking was investigated in smokers outside the workplace. Three radiation sources were considered: the natural environment (terrestrial and cosmic radiation), coal-fueled power stations, and tobacco smoke. Radium-226 and two decay products, the alpha emitter ^{210} Po and the beta emitter ^{210} Pb are found in phosphate fertilizer. Absorbed from the soil by the tobacco plant, these two radionuclides are subsequently located in cigarette smoke. Their combination with chemical carcinogens presents an additional risk to smokers.

80–0317. Sellars, S. L. **Epidemiology of Oral Cancer.** *Otolaryngologic Clinics of North America* 12(1): 45-55, February 1979.

The epidemiology of oral malignant disease, including geographic, ethnic, and socioeconomic variations, is considered. Oral cancer incidence in Western countries has declined considerably over the past 40 years. This decline has been attributed to changes in tobacco smoking and chewing habits, oral hygiene improvements, better nutrition, and effective syphilis treatment. There is a higher incidence in Southeast, Central, and East Asia; Africa; and the West Indies. Men account for 70 to 80 percent of the cases which occur in those over 50 years of age. Etiologic factors include syphilis, trauma, infection, alcohol and tobacco consumption (cigar, cigarette, pipe), and dietary deficiencies. Cheroot or chutta smoking, bidi smoking, and pan chewing are also associated with oral lesions and carcinomas.

80–0318. Tola, S.; Koskela, R.-S.; Hernberg, S.; Jarvinen, E. **Lung Cancer Mortality Among Iron Foundry Workers.** *Journal of Occupational Medicine* 21(11): 753-760, November 1979.

The present study was designed to test earlier findings of increased risk of lung cancer among iron foundry workers and to define the cause of the risk. The subjects were 3,425 workers with at least 1 year's employment in an iron foundry sometime between 1918 and 1972. There were no major differences in the smoking habits of current foundry workers representing different occupational groups. The proportion of smokers among all the workers was 57 percent and ranged from 51 percent among floor molders to 68 percent among furnacemen. The number (51) of cases of lung cancer which occurred prior to December 31, 1976 was significantly higher than that expected, i.e., 35.3 based on proportional mortality. Molders and casters had the highest excess risk in a case—control study among the cohort. The worker groups with high exposure to polycyclic aromatic hydrocarbons experienced a higher lung cancer risk than that of other groups, but the difference was not statistically significant. Although the proportion of smokers was highest among furnacemen, their risk of lung cancer was not increased. Calculation of a rank correlation between the occupations according to smoking habit and to relative frequency of lung cancer among the cases resulted in a negative coefficient (-0.45). Smoking was not considered a confounding factor. (Auth. Abs. Mod.)

80–0319. Weisburger, J. H. **Environmental Cancer: On the Causes of the Main Human Cancers.** *Texas Reports on Biology and Medicine* 37: 1-18, 1978.

A review of the causes of the principal human cancers is presented. Cancer resulting from occupational exposure is distinguished from cancers due to lifestyle, such as smoking and diet. The incidence of occupational cancers is relatively low. Most human cancers such as those of the respiratory tract, of the digestive system, and of endocrine-sensitive organs such as breast, ovary, endometrium, and prostate, are related to lifestyle. Lung cancer, which is primarily responsible for the overall increase in cancer incidence, can be traced to cigarette smoking; the more people smoke, the higher their risk for lung cancer. This risk is enhanced by other environmental factors, such as air pollution, asbestos exposure, and alcohol consumption. The role of dietary factors in the etiology of cancer of the intestines, breast, and prostate is discussed and recommendations for prevention are made.

80–0320. Weiss, W. **Lung Cancer Mortality and Urban Air Pollution.** *American Journal of Public Health* 68(8): 773-775, August 1978.

The geographic variations in lung cancer mortality rates in white males in Philadelphia were studied in relation to the earliest obtainable information on the geographic variation in air pollution. Death certificate data from the Philadelphia Department of Public Health for the years 1968-72 were examined. Philadelphia was divided into 10 health districts. Mortality rates were calculated as the average annual number of deaths per 100,000 and limited to age 30 and over, since almost all cases of lung cancer deaths occurred in this age group. The geographic variation was highly significant ($p<0.0005$). Medium and high mortality rate areas had heavy traffic, high building density, or several industries (i.e., chemical, metal, sewage, oil refineries). Concentration isopleths, drawn on a city map based on 1960 air pollution data, showed that the most heavily polluted areas in 1960 were associated with the highest lung cancer mortality rates a decade later. No information on smoking habits was available. While data were consistent with the hypothesis that air pollution is a factor in causing lung cancer, causal relationship is not justified because other important factors (i.e., smoking and occupational exposure) have not been taken into account.

80–0321. Weiss, W.; Moser, R. L.; Auerbach, O. **Lung Cancer in Chloromethyl Ether Workers.** *American Review of Respiratory Disease* 120(5): 1031-1037, November 1979.

During the period from 1948 to 1971, some workers in a chemical plant were exposed to chloromethyl ether in varying degrees and for varying durations. A historical cohort study of 1,446 male production employees from 1960 through 1975 showed both an increased risk of lung cancer as compared to that in the general population among 465 exposed workers and a dose—response relationship. Significantly increased risk occurred only among men with moderate and heavy chemical exposure. Smoking habit could account for some of the cases of lung cancer that developed in men with light chemical exposure and for cases among unexposed workers, but the cases in moderately and heavily exposed men occurred in increasing proportions of men who had never smoked. Microscopic sections of most of the lung cancers were reviewed, and the results suggested that small-cell carcinoma was a specific response to inhalation of chloromethyl ethers. (Auth. Abs. Mod.)

See also, **80–0284, 80–0285, 80–0292, 80–0394**

NON—NEOPLASTIC RESPIRATORY DISEASES

80–0322. Anderson, H. R. **Chronic Lung Disease in the Papua New Guinea Highlands.** *Thorax* 34(5): 647-653, October 1979.

In the Eastern Highlands of Papua New Guinea 46 men and 24 women with chronic lung disease underwent clinical and lung function investigations. In all cases the sole or predominant abnormality was irreversible airways obstruction, probably from chronic bronchitis with variable amounts of accompanying emphysema. There were close similarities to chronic obstructive lung disease in European populations in terms of symptoms, airways obstruction, transfer factor, and radiographic emphysema and inflammatory changes. Bronchiectasis and local fibrosis were present in a few subjects, but previous reports that pulmonary and pleural fibrosis are features of the disease were not confirmed. Possibly environmental and genetic factors may increase the associated blood gas disturbances leading to pulmonary hypertension. Unlike chronic obstructive lung disease in European populations, tobacco smoking is not an important etiological factor. Of the male subjects with forced expiratory volume in 1 second (FEV_1) <50 percent predicted, smokers comprised 76 percent of the group, ex-smokers 17 percent, and persons who had never smoked 7 percent. Among the males with $FEV_1 >50$ percent predicted, 88 percent were smokers and 12 percent ex-smokers. Although there is no direct evidence, the most likely causes of chronic lung disease are domestic wood smoke and acute chest infections. (Auth. Abs. Mod.)

80–0323. Blanco, G. G. **Effets de la Fumee du Tabac sur le Surfactant Pulmonaire. [Effects of Tobacco Smoke on Pulmonary Surfactant.]** *Broncho-Pneumologie* 28(4): 290-296, 1978, French.

Three phospholipids (lecithin, lysolecithin, and sphingomyelin) were extracted from bronchial secretions spontaneously expectorated by a group of young smokers and quantitatively determined. These subjects had no known respiratory insufficiencies. Some subjects were allowed to smoke cigarettes for a 12-hour period. Others abstained for 12 hours. The expectorants collected during that period were analyzed by thin layer chromatography or by gas chromatography. While smoking, expectorant lecithin content decreased significantly, but while abstaining from smoking it increased. The results indicated that in smokers who have long-time habits, pulmonary surfactant cannot be regenerated in sufficient quantity during smoking abstinence. A decrease in lysolecithin and sphingomyelin had no direct correlation with smoking.

80–0324. Dauber, J. H.; Rossman, M. D.; Daniele, R. P. **Bronchoalveolar Cell Populations in Acute Sarcoidosis. Observations in Smoking and Nonsmoking Patients.** *Journal of Laboratory and Clinical Medicine* 94(6): 862-871, December 1979.

Bronchoalveolar cells were obtained by segmental lung lavage from 14 patients with sarcoidosis and studied to determine changes in composition associated with acute disease and smoking history. In nonsmoking patients, both the proportion and number of lymphocytes recovered were significantly increased compared to nonsmoking controls ($p<0.001$ in both cases). Furthermore, the number of T cells was increased (4.19 ± 0.72 vs. $0.896 \pm 0.107 \times 10^6$ cells/100 ml, $p<0.001$), and activated T cells were also increased (1.04 ± 0.478 vs. $0.148 \pm 0.032 \times 10^6$ cells/100 ml, $p<0.01$). Less striking differences in these values were found when smoking sarcoid patients were compared to smoking controls, but there were no significant differences in these values for smoking and nonsmoking patients ($p>0.05$). Thus acute sarcoidosis in both smoking and nonsmoking patients appears to be associated with a lymphocytosis of the bronchoalveolar space which results from increased numbers of T cells and activated T cells. The potential importance of this finding with regard to diagnosis and pathogenesis is discussed. (Auth. Abs.)

80–0325. Eriksson, S. **Emfysemets Biokemi, Nagra Nyare Ron. [The Biochemistry of Emphysema, Some Recent Findings.]** *Kvartalsskrift* 73(4): 111-120, 1978, Swedish.

This article presents the protease theory for the development of emphysema. Panacinar emphysema appears to be linked with alpha-antitrypsin deficiency. The effect of cigarette smoking on the onset of panacinar emphysema symptoms was studied in 250 Swedish patients with severe antitrypsin deficiency. In smokers, symptoms such as impaired breathing appeared around the age of 40, as compared with nonsmokers in whom symptoms appeared 15 to 20 years later.

80–0326. Fischbein, A.; Rohl, A. N.; Langer, A. M.; Selikoff, I. J. **Drywall Construction and Asbestos Exposure.** *American Industrial Hygiene Association Journal* 40(5): 402-407, May 1979.

A clinical field survey of 114 drywall taping workers, employed in the New York Metropolitan area, was conducted. Examination included a careful lifetime occupational history with recording of all previous occupations on a specially designed questionnaire. Interview also included past medical history, smoking history, and review of chest symptoms, such as chronic bronchitis, dyspnea, and cyanosis. Chronic bronchitis was recorded as present if the individual gave a history of phlegm production for more than 3 months per year during a period of 2 years or longer. Each individual had a comprehensive physical examination with special emphasis on examination of the chest. Standard chest roentgenograms were taken. There were 79 smokers (69 percent), 13 nonsmokers (11 percent), and 22 ex-smokers (19 percent) among the examined workers. Chronic bronchitis was reported by 25 individuals (22 percent), all of whom were heavy smokers. A history of dyspnea on exertion was given by 10 workers (9 percent); of these, nine were heavy smokers, and one was an ex-smoker. Chest examination revealed dry rales in 10 individuals (9 percent), 7 of whom were smokers and 3 of whom were ex-smokers. Six of these had a positive chest X-ray for parenchymal disease; none had any visible pleural abnormality. Rhonchi were found in 15 workers (13 percent), 14 of whom were smokers. Five of these showed parenchymal disease on X-ray, while two had pleural thickening. The prevalence of abnormal chest films increased with longer duration since onset of exposure.

80–0327. Gadek, J. E.; Fells, G. A.; Crystal, R. G. **Cigarette Smoking Induces Functional Antiprotease Deficiency in the Lower Respiratory Tract of Humans.** *Science* 206(4424): 1315-1316, December 1979.

Current concepts of the pathogenesis of emphysema suggest that it results from an imbalance of elastase and antielastase activity within the alveolar structures. Although emphysema that is associated with hereditary deficiency of serum α_1-antitrypsin conforms to this scheme, the major risk factor in the more common form of emphysema is cigarette smoking. A study was designed to evaluate the premise that cigarette smoking may be associated with an acquired, functional defect in lung α_1-antitrypsin. Two groups were studied. The first consisted of 10 healthy individuals (6 nonsmokers and 4 smokers) with no evidence of lung disease; in the second group were 19 individuals (8 nonsmokers and 11 smokers) with idiopathic pulmonary fibrosis. Determination of the antielastase activity of α_1-antitrypsin obtained from the lungs of smoking and nonsmoking individuals revealed a nearly twofold reduction in the functional activity of this elastase inhibitor in the lungs of cigarette smokers even though antigenic concentrations of α_1-antitrypsin were the same in healthy nonsmokers and smokers ($p>0.2$) and were similar in smoking and nonsmoking individuals with pulmonary fibrosis ($p>0.3$). These data suggest that cigarette smokers may lose some of the normal antielastase protective screen of the lower respiratory tract, making them more vulnerable to destructive lung disease. (Auth. Abs. Mod.)

80–0328. Gelb, A. F.; Lugliani, R.; Schiffman, P.; Klein, E.; Zamel, N. **Site of Airway Obstruction After Acute Cigarette Smoking and Mild Smoke Inhalation.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(3): 481-490, 1979.

The first part of this study was designed to localize the site of airway obstruction after smoking one cigarette in 16 nonsmoking, male, healthy, asymptomatic volunteer subjects aged 30 ± 5 years (mean ± 1 SD). All subjects had baseline normal physiologic studies. Measurements made 5 and 10 min after termination of the cigarette smoking included forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1), specific conductance (SG_{aw}), maximal flow at 50 ($Vmax_{50}$) and 25 percent ($Vmax_{25}$) of FVC after breathing air and after three inspiratory VC of an 80 percent helium-20 percent oxygen mixture. The volume of isoflow ($Viso_V$) and the increase of flow at 50 percent FVC after breathing the helium mixture ($Vmax_{50}$ He-air/air) were calculated. The only statistically significant differences ($p<0.05$) occurred in $Vmax_{50}$ air at 5 min with a fall from a baseline mean ± 1 SD of 4.8 ± 1.1 to 4.6 ± 1.00 $lsec^{-1}$ and a fall in specific conductance from 0.25 ± 0.06 to 0.22 ± 0.06 sec^{-1} cm H_2O^{-1} . In the second part of the study, similar measurements were made on six of the previously studied volunteers following an unexpected exposure to smoke from a large forest fire. While $Vmax_{50}$ and SG_{aw} were reduced, the $Vmax_{50}$ He-air/air was increased and $Viso_V$ and $Vmax_{25}$ were unchanged. .This strongly suggests that reversible obstruction of the large airways predominated in both circumstances. (Auth. Abs.)

80–0329. Gupta, P. K.; Frost, J. K.; Geddes, S.; Aracil, B.; Davidovski, F. **Morphological Identification of Alpha-1-Antitrypsin in Pulmonary Macrophages.** *Human Pathology* 10(3): 345-347, May 1979.

Sputum specimens were obtained from 50 smokers, 10 nonsmokers, and 20 patients with chronic obstructive pulmonary disease. The polar orangophilic structures found in the smokers' pulmonary macrophages were analyzed by histochemical and immunofluorescence techniques and found to contain alpha$_1$-antitrypsin.

80–0330. Haack, D. G. **Interactions of Risk Factors for COPD. (Letter).** *American Review of Respiratory Disease* 118(4): 798-799, October 1978.

In reply to a letter concerned with risk factors for chronic obstructive pulmonary disease, the correlation between age and pack years is reiterated. Further, it is cautioned that significant differences between groups must be carefully interpreted. If two groups are age-matched, a difference between the groups with regard to pack years will indicate a difference in smoke consumption. If the groups are not age-matched, a difference in pack years may be due to differences in age, smoke consumption, or both. See also 78-0480 and 80-0100.

80–0331. Hayes, D. A.; Pimmel, R. L.; Fullton, J. M.; Bromberg, P. A. **Detection of Respiratory Mechanical Dysfunction by Forced Random Noise Impedance Parameters.** *American Review of Respiratory Disease* 120(5): 1095-1100, November 1979.

Respiratory mechanical parameters were computed from forced random noise impedance data in normal nonsmoking adults (group 1), asymptomatic smokers (group 2), and patients with obstructive pulmonary disease (group 3). Mean values for all derived parameters were significantly different ($p<0.025$) for group 3. Mean values of resonant frequency, the ratio of low-frequency (5 to 9 Hz) resistance to high-frequency resistance (15 to 19 Hz), and conductance normalized by height were significantly different ($p<0.05$) between groups 1 and 2. This approach appears to yield respiratory mechanical parameters that are sensitive to mechanical alterations induced by early pulmonary disease. The rapid, noninvasive, and effort-independent nature of this approach make it especially useful for epidemiologic studies and for studies of "noncooperative" subjects. (Auth. Abs.)

80–0332. Jedrychowski, W. **A Consideration of Risk Factors and Development of Chronic Bronchitis in a Five-Year Follow-Up Study of an Industrial Population.** *Journal of Epidemiology and Community Health* 33(3): 210-214, September 1979.

A 5-year followup study (1968-1973) of an industrial population in a fertilizer plant showed that the prevalence and development of chest symptoms in men was related mainly to their smoking habits and, to a lesser degree, to age. The proportion of those with chronic bronchitis (CB) symptoms was slightly smaller in 1973 than in 1968, possibly due to an increase in ex-smokers from 6.1 to 16.2 percent during that period. In the group of nonsmokers (29 out of 197 in 1973), the prevalence of CB symptoms was half as large in 1973 as in

1968 (7 vs. 14 percent). Among current smokers (136 out of 197 in 1973), CB symptoms were only slightly less prevalent in 1973 than 1968 (27 vs. 29 percent). The prevalence of CB symptoms in ex-smokers remained unchanged (16 percent) over the same period. Mean forced expiratory volume in 1 second (FEV$_1$) values were related to age, and decrease in FEV$_1$ was related to age and smoking habits, decreasing faster in older than in younger men (9 vs. 4 percent) and in current smokers than in nonsmokers and ex-smokers (7 vs. 3 and 5 percent, respectively). The levels of airborne dust and fluorides at the working places were related to the prevalence of CB symptoms, to the persistence of those symptoms during the 5-year period, and to decrease of FEV$_1$ values during that time. The decrease in FEV$_1$ was especially noticeable among current smokers. The 5-year incidence study showed that particular occupational factors do indeed have a real effect, but their meaning in the development of the CB syndrome is less marked than was expected from the prevalence rates. (Auth. Abs. Mod.)

80–0333. Lam, S.; Abboud, R. T.; Chan-Yeung, M.; Rushton, J.-M. **Neutrophil Elastase and Pulmonary Function in Subjects with Intermediate Alpha$_1$-Antitrypsin Deficiency (MZ Phenotype).** *American Review of Respiratory Disease* 119(6): 941-951, June 1979.

The relationship between neutrophil elastase, smoking, and pulmonary function was studied in 32 subjects with intermediate alpha$_1$-antitrypsin deficiency (Pi MZ phenotype). Nineteen subjects were detected in population studies, and 13 were detected in a family study of patients with chronic airway obstruction. Of the 32 subjects, 10 less than 30 years of age and 12 more than 30 years of age had normal lung function. Subjects in the older group were either ex-smokers or nonsmokers, whereas the younger group included five smokers. The remaining 10 subjects, all more than 30 years of age and most of them current smokers, had abnormal lung function as determined by one or more of the following indices: maximal expiratory flow, specific conductance, closing capacity, slope of phase III of the single-breath N$_2$ test, lung elastic recoil, or upstream resistance. The neutrophil elastase content of all Pi MZ subjects as a group was not significantly different from that of a control group of 26 subjects of Pi M phenotype. However, in Pi MZ subjects more than 30 years of age, those with abnormal lung function had greater neutrophil elastase content than did those with normal lung function. The 12 MZ subjects more than 30 years of age with normal lung function had less neutrophil elastase content as compared to the 20 M control subjects more than 30 years of age. In Pi MZ subjects more than 30 years of age, neutrophil elastase content correlated significantly with tests of maximal expiratory flow, airway conductance, closing capacity, and slope of phase III, whereas smoking showed a correlation with the latter four measurements, but not with tests of maximal expiratory flow. Neutrophil elastase and smoking interacted as significant factors related to abnormal lung function in Pi MZ subjects more than 30 years of age. In MZ antitrypsin-deficient subjects neutrophil elastase content is a significant risk factor related to lung function abnormalities and neutrophil elastase and smoking interact in producing abnormal lung function in Pi MZ subjects. (Auth. Abs. Mod.)

80–0334. Lauwerys, R. R.; Roels, H. A.; Buchet, J.-P.; Bernard, A.; Stanescu, D. **Investigations on the Lung and Kidney Function in Workers Exposed to Cadmium.** *Environmental Health Perspectives* 28: 137-145, February 1979.

Findings from epidemiologic studies of Belgian workers exposed to cadmium are reported. Only minor impairments of lung function (mild form of obstructive lung disease) were found after long-term occupational exposure (<20 years) to moderate concentrations of cadmium (Cd) oxide dust and fume. This conclusion cannot, however, be extrapolated to acute or subacute inhalational exposure. The effect of cigarette smoking on pulmonary function was evident in a group of workers with an average exposure of 4 years as indicated by a significant reduction of maximal expiratory flow rate at 50 percent of the vital capacity ($p<0.05$) and an increase in the time required to expire 50 percent of the vital capacity ($p<0.05$) in smokers of both the exposed and control groups as compared to nonsmokers of the same groups. Thus, an occupational exposure of 4 years to a time-weighted average concentration of about $10 \mu g/m^3$ (total dust) or less than $4 \mu g/m^3$ (respirable dust) has probably no deleterious effect on lung function. The kidney seems more sensitive to the chronic effect of cadmium than the lung. The nephrotoxicity of cadmium is evidenced by a tubular dysfunction characterized by increased excretion of β_2-microglobulin giving rise to classical tubular proteinuria and by a glomerular dysfunction evidenced by increased excretion of high molecular weight proteins and increased levels of β_2-microglobulin and creatinine in plasma giving rise to a glomerular-type proteinuria. These renal changes were mainly found in workers whose cadmium concentration at the time of the survey exceeded 1 μg Cd/100 ml in blood and 10 μg Cd/g creatinine in urine. It should, however, be stressed that higher levels of Cd in blood and in urine are not necessarily associated with the presence of excessive proteinuria. In newly exposed workers, the Cd level in blood increases progressively to a plateau after several weeks. Cadmium level in urine fluctuates more. In workers exposed for several months to an airborne concentration exceeding 200 $\mu g/m^3$, Cd concentration in urine seems mainly influenced by recent exposure. (Auth. Abs. Mod.)

80–0335. Melissinos, C. G.; Webster, P.; Tien, Y. K.; Mead, J. **Time Dependence of Maximum Flow as an Index of Nonuniform Emptying.** *Journal of Applied Physiology* 47(5): 1043-1050, November 1979.

A new technique was developed that permits accurate superimposition of V_{max} data over small volume intervals. When quick-release interrupted partial curves with similar volume history but different volume of initiation were compared over their late common-volume segments, the volume-history effects could be differentiated from time dependence of the maximal expiratory flow (V_{max}). Such time dependence was found in 7 of 7 bronchitics, 3 of 5 smokers, and none of the 14 nonsmokers tested. All the bronchitics, three of the five smokers, and one of the two ex-smokers showed flows higher in the partial initiated at lower lung volume than in the partial initiated higher in the double maneuver. The difference was 16 to 26 percent in the smokers who had a flow difference and 15 to 50 percent in the patients with chronic bronchitis. The slope of the lower partial flow-volume curve was more pronounced than the one of the maximal expiratory flow-volume curve, or the partial initiated at higher volumes; this resulted in progressive tapering of the flow difference ending with superimposition of the two curves. This effect could be completely masked by the total lung capacity effect. The duration of the initial flow transient was longer in the patients, reaching 140 msec; it was considered to be completed when the sudden change in slope of the partial-partial flow-volume curve occurred. The differences in V_{max} persisted beyond the control transient for time intervals that far exceeded this or any published estimates of the duration of the airway transient (180 to 510 msec in the smokers and 400 to 820 msec in the patients. The volume expired during the initial flow transient was not calculated. Excluding this "airway" transient, the volume of air expired, calculated by integration of the flow in excess of the flow seen in maneuvers beginning at higher lung volumes, was 160 to 300 ml for smokers who had a flow difference and 200 to 400 for patients. The emptying during the forced expirations is not uniform even in mild disease states and the time dependence of V_{max} is a sensitive test of lung inhomogeneity. (Auth. Abs. Mod.)

80-0336. Mustafa, K. Y.; Lakha, A. S.; Milla, M. H.; Dahoma, U. **Byssinosis, Respiratory Symptoms and Spirometric Lung Function Tests in Tanzanian Sisal Workers.** *British Journal of Industrial Medicine* 35(2): 123-128, May 1978.

Byssinosis and other respiratory symptoms and acute and chronic changes in forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1) were investigated in 77 workers in sisal spinning and 83 workers in sisal brushing departments in six Tanzanian sisal factories. Although the prevalence of byssinosis in spinning departments was found to be low (5.2 percent), it was very high in brushing departments (48.2 percent). Workers in brushing were exposed to sisal dust for a significantly longer period (11.77±7.3 years) compared to workers exposed to sisal in spinning (2.85±2.56 years). No significant difference was observed in the smoking habits of the workers: 42 percent of those in the brushing department smoked compared with 37 percent of those in spinning. More than 90 percent of smokers in both departments smoked less than 10 cigarettes a day. Although the number of smokers in brushing was similar to that in spinning, smokers were more prone to byssinosis than were nonsmokers or ex-smokers after standardization for duration of exposure. It is concluded that a high prevalence of byssinosis associated with chronic and acute changes in FVC and FEV_1 occurs in the brushing departments of sisal factories, and that this is related to lengthy exposure, high dust level, and smoking. (Auth. Abs. Mod.)

80-0337. O'Connor, S. A.; Jones, D. P.; Collins, J. V.; Heath, R. B.; Campbell, M. J.; Leighton, M. H. **Changes in Pulmonary Function After Naturally Acquired Respiratory Infection in Normal Persons.** *American Review of Respiratory Disease* 120(5): 1087-1093, November 1979.

Changes in pulmonary function due to naturally occurring respiratory tract infection were examined in 26 normal healthy volunteers during a period of 6 months. In this group there were 20 nonsmokers and 6 smokers. Of the smokers, 3 were cigarette smokers (10, 12, and 16 pack-years), and 3 were pipe smokers who had smoked for at least 15 years (25, 50, and 75 g of tobacco/week, respectively). Forced expiratory maneuvers in each volunteer were recorded at 2-week intervals throughout the study and daily during illness. Significant impairment of peak expiratory flow rate, forced vital capacity (FVC), forced expiratory volume in 1 second, and maximal mid-expiratory flow rate at 50 percent of the vital capacity was observed during infection, whereas changes in the maximal expiratory flow rate at 75 percent of vital capacity were nonsignificant. A group t test was used to assess whether there were any significant differences between the changes in pulmonary function after infection in the group of smokers and nonsmokers. There was no significant difference for any of the 5 pulmonary function variables at the 5 percent level, although the peak expiratory flow rate was significant at the 10 percent level. From these results, it is concluded that large airways are certainly affected during uncomplicated respiratory infections in normal healthy persons, and from the changes observed in FVC it is sugggested that more widespread involvement of the small airways may occur. (Auth. Abs. Mod.)

80-0338. Pannier, R. **Estimation de la Capacite de Travail et Problemes Sociaux. [Evaluation of Working Capacity and Social Problems.]** *Acta Tuberculosea et Pneumologica Belgica* 68(2/3): 193-210, April-September 1977, French.

Respiratory function tests were performed in 65 men less than 65 years of age who were suffering from chronic bronchitis. The aim of the study was not only to determine the physiopathological criteria of working capacity (or incapacity) but also to take into account some purely individual behavioral factors such as the patient's attitude toward health. In order to scientifically determine a physiopathological basis for working capacity, the ratios of forced expiratory volume to vital capacity as well as to residual volume were selected as determining factors. It was shown that some patients with comparable spirometric data considered themselves to be able to work and were gainfully employed, while others did not work—and this was due to different behavioral attitudes. It was also demonstrated that smoking habits had a very real and quantifiable effect on the development of chronic bronchitis. The danger point seems to lie around a daily tobacco consumption equivalent to 15 cigarettes, especially in patients with an unfavorable forced expiratory volume to vital capacity ratio. The fact that there were heavy smokers among active as well as inactive patients probably corroborates the theory that apart from purely biological conditions, some intangible sets of values and attitudes also play a certain role in health maintenance.

80-0339. Pavia, D.; Bateman, J.R.M.; Sheahan, N. F.; Clarke, S. W. **Effect of Ipratropium Bromide on Mucociliary Clearance and Pulmonary Function in Reversible Airways Obstruction.** *Thorax* 34(4): 501-507, August 1979.

The effects of (1) regular use for 1 week and (2) a single dose of a synthetic anticholinergic (ipratropium bromide) on lung mucociliary clearance and on bronchodilation were ascertained in a controlled, double-blind, cross-over study in 12 patients (10 men, 2 women) with reversible airways obstruction (mean increase in forced expiratory volume in 1 second (FEV_1) after isoprenaline: 17 percent, range 10 to 50 percent). Six patients were ex-smokers and six were current smokers whose level of tobacco consumption remained constant throughout the study. Two puffs from a metered dose inhaler of either placebo (propellants only) or drug (40 μg) were administered four times a day for 1 week (regular use), and mucociliary clearance was measured, by radioaerosol tracer, at the end of each treatment period and after a control period in which no treatment was given. On the mornings of the measurements after the placebo and drug periods, one final dose (single dose) of ipratropium (40 μg) or placebo was given 2.5 hours before the start of the test. There was no statistically significant difference between the three mean mucociliary clearance curves (control, placebo, and drug) for the group; however, there was a significantly greater penetration of the tracer in the test after drug administration towards the periphery of the lung compared with the other two. This increased penetration was attributed to bronchodilatation caused by the drug. Ipratropium's bronchodilator action was shown by increased lung function indices (FEV_1, forced vital capacity, and peak expiratory flow rates) following administration of the drug. Ipratropium bromide did not appear to impair mucociliary clearance, and it acted as an effective bronchodilator. (Auth. Abs. Mod.)

80-0340. Plamenac, P.; Nikulin, A.; Pikula, B.; Vujanic, G. **Cytologic Changes of the Respiratory Tract as a Consequence of Air Pollution and Smoking.** *Acta Cytologica* 23(6): 449-453, November-December 1979.

A comparison was made of cytologic abnormalities in sputum specimens from inhabitants of an urban environment in Yugoslavia with a high level of air pollution (Sarajevo) and from inhabitants of a rural area (Podvelezje). Of the urban subjects, 51 were male (27 smokers and 24 nonsmokers) and 53 were female (28 smokers and 25 nonsmokers); of the rural subjects, 40 were male (29 smokers and 11 nonsmokers) and 44 were female (24 smokers and 20 nonsmokers). The cytologic specimens were evaluated in reference to eosinophils, respiratory spirals, atypia of bronchial cells, and squamous metaplasia. The abnormal findings were more frequent among urban inhabitants than among rural ones. In both groups the abnormalities were more pronounced in smokers than in nonsmokers and in men than in women. Atypical squamous metaplasia was observed in 56 percent of the male and 46 percent of the female smokers and in 29 percent of the male and 16 percent of the female nonsmokers from the urban environment. There were no significant differences in cytologic abnormalities between nonsmokers from the urban environment and smokers from the rural area. (Auth. Abs. Mod.)

80-0341. Smidt, U. **Problems and Results in the Simultaneous Assessment of the Role of Smoking and Occupational Dust Exposure for Chronic Bronchitis and Pulmonary Emphysema.** *Broncho-Pneumologie* 28(3): 177-185, May-June 1978.

Problems encountered in the simultaneous assessment of the role of smoking and occupational dust exposure in chronic bronchitis and pulmonary emphysema and the results of a joint epidemiologic study of 13,000 subjects in Germany are presented. Each institute examined dust-exposed and nonexposed subjects from various industries, including coal mines; foundries; ceramic, asbestos, and cement works. A questionnaire was used and included questions about cough, dyspnea, phlegm, previous illnesses, occupational and smoking histories, clinical signs, ECG, chest X-rays, sputum, and lung function. Subjects were also grouped according to age (<35, 36 to 55, >55), average smoking habits during the last 5 years (moderate, <10 cigarettes/day; heavy, >10 cigarettes/day), and occupational dust exposure (low, high). Chronic bronchitis was grouped into three categories: type 1 (light), type 2 (moderate), and type 3 (severe). Young and middle-aged heavy smokers with high dust exposure had higher prevalences of chronic bronchitis (CB) than heavy smokers with low dust exposure. Moderate smokers with high dust exposure had higher prevalences of CB than moderate smokers with low dust exposure. In the high age group, moderate or heavy smoking, or high dust exposure caused a higher CB prevalence.

80-0342. Tattersall, S. F.; Pereira, R. P.; Hunter, D.; Blundell, G.; Pride, N. B. **Lung Distensibility and Airway Function in Intermediate $Alpha_1$-Antitrypsin Deficiency (Pi MZ).** *Thorax* 34(5): 637-646, October 1979.

This study examined the role of intermediate $alpha_1$-antitrypsin deficiency in predisposing to abnormalities of lung distensibility and airway function in 20 heterozygotes (Pi MZ) who were individually matched with a control Pi M subject of similar age, height, and smoking habits drawn from the same male working population. Among the Pi MZ subjects were 4 nonsmokers, 1 ex-smoker, 4 light smokers (up to 10 cigarettes daily), and 11 heavy smokers (20 or more cigarettes daily). There were no significant differences between the heterozygotes and their controls in the results of spirometry, maximum expiratory flow-volume curves (breathing air), single breath nitrogen test, arterialized capillary blood oxygen pressure, or single breath carbon monoxide transfer. Additional studies were made in 12 of the pairs of Pi MZ and Pi M subjects. Comparison of maximum expiratory flow-volume curves breathing air and 80 percent helium-20 percent oxygen showed no differences between the Pi MZ and Pi M subjects. Although airway function was similar in the 2 groups, 4 of 12 Pi MZ subjects showed abnormalities of the pressure-volume curve of the lung (reduction in lung recoil pressure, abnormal shape factor, increase in functional residual capacity). Abnormalities of washout of a helium-sulphur hexafluoride gas mixture, of a type previously described as characteristic of emphysema, were found in two of the men with abnormal pressure-volume curves. When the full range of tests of pulmonary function was considered, it was apparent that the abnormalities found were predominantly in three of the four middle-aged, heavily smoking Pi MZ subjects studied, but the numbers were too small to permit statistical analysis of this trend. The results suggest that Pi MZ subjects have an increased susceptibility to alveolar abnormalities without increased abnormalities of airway function; this may explain the increased frequency of emphysema at necropsy despite many studies showing no predisposition to abnormal airway function in life. The functional changes observed would be unlikely to cause symptoms. The risk of disablement from chronic lung disease appears to be only slightly enhanced by intermediate alpha$_1$-antitrypsin deficiency. (Auth. Abs. Mod.)

See also, **80–0311, 80–0353**

CARDIOVASCULAR DISEASES

80–0343. Alvarez Alva, R. **Tabaquismo y Salud. [Tobacco Smoking and Health.]** *Gaceta Medica de Mexico* 112(5): 331-343, November 1976, Spanish.

A study was carried out in eight Latin American cities in order to estimate the prevalence of tobacco smoking. Using questionnaires resembling those which are in use in the United States, the interrogators determined the incidence of smoking (percentages) as La Plata-58, Sao Paulo-54, Caracas-49, Santiago-47, Mexico-45, Guatemala-36, and Lima-34. Among the studied population there were 2.5 times more male smokers than female smokers. This proportion contrasts with that of the United States where male vs. female smokers are 42 vs. 30 percent of the population. The study also gives data on the increase in mortality caused by tobacco smoking among different professions of different countries, e.g., the mortality increase in Great Britain among medical doctors is 28 percent. Decreased mortality is noted when cigarettes are used with another form of tobacco. The coefficient of mortality caused by pulmonary carcinoma is 125 per 100,000 whereas after 5 years of abstention from smoking it diminishes to 50 per 100,000.

80–0344. Bastian, P. **Coronary Heart Disease in Tribal Aborigines—The West Kimberley Survey.** *Australian and New Zealand Journal of Medicine* 9(3): 284-292, June 1979.

Two hundred and forty-eight full-blood tribal Aborigines from the West Kimberley region of Western Australia (WA) were surveyed for the prevalence of coronary heart disease (CHD), and compared with the known prevalence in whites in the country town of Busselton, WA. The prevalence was greater among the Aborigines, with 7 percent of the men, and 11 percent of the women found to have "probable" coronary heart disease. Seven percent of the population had electrocardiographic changes characteristic of frank ischemia. The major risk factors contributing to this high prevalence were hypertension, diabetes mellitus, and obesity. There appeared to be a negative correlation between current smoking and the presence of CHD. The proportion of smokers for both sexes progressively dropped from "non" to "suspect" to "probable" CHD groups. None of the women in the "probable" CHD group currently smoked. Thirty-seven percent of the Aborigines were hypertensive, and 17 percent had diabetes mellitus. (Auth. Abs. Mod.)

80–0345. Beaglehole, R.; Prior, I.A.M.; Eyles, E.; Sampson, V. **High Density Lipoprotein Cholesterol and Other Serum Lipids in New Zealand Maoris.** *New Zealand Medical Journal* 90(642): 139-142, August 22, 1979.

The pattern of fasting serum lipids, with emphasis on high density lipoprotein cholesterol (HDL-C), and the relationship of the lipids with each other and with other coronary heart disease (CHD) risk factors were examined in a population-based sample of New Zealand Maoris. Analysis of covariance was used, controlling for age, to test for differences in lipid levels between those with and those without CHD risk factors (diabetes, smoking, alcohol consumption, oral contraceptives). Current male smokers had a significantly lower mean total cholesterol (5.79 mmol/l) than nonsmokers (6.11 mmol/l), $p = 0.022$. Current male drinkers had a significantly higher mean HDL-C level and a higher median triglycerides level than nondrinkers. There were no differences between smokers and nonsmokers or drinkers and nondrinkers in lipid levels in women. There were no sex differences in the distribution of total cholesterol and cholesterol fractions, but triglycerides were higher in men. High density lipoprotein cholesterol levels were lower in Maoris than in other populations. High density lipoprotein cholesterol was negatively correlated with body mass index and with low density lipoprotein cholesterol, but was not associated with total cholesterol. The possible relationship between the low levels of HDL-C and the high risk of coronary heart disease in Maoris requires investigation. (Auth. Abs. Mod.)

80–0346. Benacerraf, A.; Castillo-Fenoy, A.; Goffinet, D.; Krantz, D. **L'Infarctus du Myocarde Avant 36 Ans. A Propos de 20 Cas. [Myocardial Infarction Before 36. A Study of 20 Cases.]** *Archives des Maladies du Coeur et des Vaisseaux* 71(7): 756-764, July 1978, French.

Risk factors, clinical characteristics of the infarction, ventriculography, and coronary arteriography have been studied in 16 men (age 20 to 35) and 4 women (age 23 to 32) with myocardial infarct. Ten patients (50 percent) had a family history of vascular disease, 17 patients (85 percent) smoked over 20 cigarettes/day, and 15 (75 percent) exhibited lipid abnormality. All female patients had been using oral contraceptives for 2 to 5 years, and all were smokers. There was an average of three risk factors per patient. In 14 out of 20 cases (70 percent), smoking was associated with lipid abnormality. In 14 cases, no warning signals such as anginal pain preceded the myocardial infarct. In three cases, the attack was precipitated by a strong physical effort. An exercise test administered to 16 patients showed an associated abnormality in 1 out of 4 cases. The rate x pressure product was normal (3,530±607 cm Hg/mm), suggesting a satisfactory functioning of the coronary artery network. A recurrence was observed in 4 patients, all smokers, 9 months to 12 years after the initial infarct. The ventriculography was normal in 35 percent of the cases. Coronary arteriography showed significant lesions in one or two trunks in 12 cases, and normal coronary artery network in 6 cases. The lesion index was 1.6. The smoking-lipid abnormality association was found in 3 out of 6 cases with unaltered coronary artery and in 10 out of 12 cases with coronary lesions. Mortality was zero during the first month after the attack. Two patients died 45 days and 2 years after the attack. The latter patient was a smoker.

80–0347. Burch, P.R.J. **Coronary Heart Disease and Smoking.** *Tabak Journal International* (4): 241, 244, August 1978.

Coronary heart disease now accounts for about one quarter of all deaths in England and Wales. Furthermore, cigarette smokers suffer a higher mortality from the disease than nonsmokers, although the relative risk connected with smoking decreases with increasing age. The question therefore arises: Does smoking actually cause coronary heart disease? Or does the association have a constitutional basis? Genetic factors predispose both to smoking and to coronary heart disease, and we need to determine whether these factors are associated. Mortality statistics for England and Wales over the period 1921 to 1973 have been studied in an attempt to resolve these questions. These data bear critically on the issue of smoking and coronary heart disease, and they show that little or no causal action is involved; the association of the habit with the disease has a genetic basis. (Auth. Abs.)

80–0348. Crittin, J.; Waters, D. D.; Theroux, P.; Mizgala, H. F. **Left Main Coronary Artery Stenosis in Young Patients.** *Chest* 76(5): 508-513, November 1979.

Among 1,041 patients less than 45 years old who underwent coronary arteriography from 1972 to 1977, left main coronary stenosis $>$ 50 percent was present in 31 men (3.4 percent) and in 10 women (7.2 percent, $p<0.05$). The degree of stenosis did not correlate with the duration of symptoms, the severity of angina, the presence of a previous myocardial infarction, nor with the number of risk factors. Hypertension was present in 5 of 10 women and in 3 of 31 men ($p<0.0025$). Cholesterol and/or triglycerides were increased in 82 percent of patients (32 of 39) and 85 percent (35 of 41) smoked more than 10 cigarettes per day within the 5 years prior to admission. The number of risk factors did not increase with the severity of left main stenosis but did increase with the number of associated stenotic coronary arteries. The clinical and angiographic features in young men did not seem to differ from those described in unselected populations; however, in young women, left main coronary stenosis was often an isolated lesion associated with a short duration of symptoms, a high prevalence of hypertension, no previous myocardial infarction, and a normal ventriculogram, suggesting the possibility that a different pathophysiologic mechanism might be involved. (Auth. Abs. Mod.)

80–0349. Danner, S. A.; de Beaumont, M.-J.; Dunning, A. J. **Cardiovascular Health in the Tenth Decade.** *British Medical Journal* 2(6138): 663, September 2, 1978.

In order to determine whether persons who live unusually long healthy lives have few coronary risk factors or whether their longevity is determined familially, 100 Dutch nonagenarians (50 men and 50 women) were studied. The investigation consisted of an interview, physical examination, ECG, chest X-ray, and blood tests. It was found that hypertension and diabetes mellitus had been diagnosed in 19 percent and 8 percent of the subjects, respectively. Only 2 percent had smoked more than an occasional cigarette; in general, the women had never smoked and the men smoked only a few cigars a day. Current total cholesterol concentration was very low, especially in men, and high density lipoprotein was normal. When the life span of the nonagenarians' parents was compared to that of the parents of a control group, no significant difference was found ($p>0.10$). It is concluded that in many respects the coronary risk profile of these apparently healthy nonagenarians represents the mirror image of that of the contemporary coronary-prone middle-aged adult.

80–0350. Davis, J. W.; Davis, R. F. **Acute Effect of Tobacco Cigarette Smoking on the Platelet Aggregate Ratio.** *American Journal of the Medical Sciences* 278(2): 139-143, September-October 1979.

The purpose of this study was to determine whether cigarette smoking acutely increased circulating platelet aggregates as detected by lowering of the platelet aggregate ratio. Eighteen (11 male and 7 female) volunteers ranging from 16 to 47 years of age rested during a 20-minute control period and smoked two unfiltered tobacco cigarettes during another 20-minute period. Among the volunteers, 13 were nonsmokers, 3 were cigarette smokers, and 2 were pipe smokers. The smokers did not smoke for at least 8 hours before each experimental period began. Platelet aggregate ratios (mean ± SD) before and after the control period were 0.86 ± 0.13 and 0.82 ± 0.12, respectively ($p > 0.05$), and before and after smoking were 0.84 ± 0.14 and 0.66 ± 0.18, respectively ($p < 0.01$). The mean plasma nonesterified fatty acid concentration was not significantly different before and after smoking, suggesting that the decrease in platelet aggregate ratio was not mediated through elevation of the plasma nonesterified fatty acid concentration. The results are consistent with the theory that platelet aggregates contribute to the increased incidence of myocardial infarction and sudden death from coronary artery disease observed among cigarette smokers. (Auth. Abs. Mod.)

80–0351. Emerson, P. A. **Preventing Thromboembolism After Myocardial Infarction: Effect of Low-Dose Heparin or Smoking.** *American Heart Journal* 96(1): 129-130, July 1978.

The effects of low-dose heparin and of smoking on the prevention of thromboembolism after myocardial infarction are evaluated. Studies have indicated that thromboembolic complications after myocardial infarction are most likely in the elderly, in those with heart failure, in those with significant dysrhythmia, and in nonsmokers rather than in smokers. Studies have reported decreased incidence of leg vein thrombosis in smokers after gynecological operations. One possible explanation is that smokers who had to stop smoking after admission to a coronary care unit start their postoperative or postmyocardial infarction mobilization earlier. It is concluded that low-dose heparin prophylaxis should be given to all myocardial infarction patients, particularly to those with an increased risk of thromboembolic complications.

80–0352. FDA Drug Bulletin. **Oral Contraceptive Labeling.** *FDA Drug Bulletin* 8(2): 12-13, March-April 1978.

A major warning that women who use oral contraceptives (OCs) should not smoke was added to the labeling in 1978. The warning was based on studies, reported in 1977, which established an increased risk of heart attack and other circulatory problems such as stroke for women who smoke while taking OCs. Healthy women who do not smoke but use OCs double the risk of suffering a heart attack, compared with nonusers of OCs. But OC users who also smoke, especially if they smoke 15 or more cigarettes per day, are three times more likely to die of a heart attack or other circulatory disease than OC users who do not smoke, and are ten times more likely to die of a heart attack or circulatory disease than women who use neither OCs or tobacco. The risk of heart attack for women taking OCs increases with the amount of smoking and with age, and is higher in women with other conditions that predispose them to heart attack such as high blood pressure, obesity, and diabetes.

80–0353. Hebbel, R. P.; Eaton, J. W.; Modler, S.; Jacob, H. S. **Extreme But Asymptomatic Carboxyhemoglobinemia and Chronic Lung Disease.** *Journal of the American Medical Association* 239(24): 2584-2586, June 16, 1978.

Carboxyhemoglobinemia is a well-known consequence of carbon monoxide exposure from smoking. However, only moderately elevated levels have been reported. This article reports the case of an asymptomatic man with severe chronic obstructive lung disease and carboxyhemoglobin levels repeatedly in excess of 30 percent (maximum, 38.0 percent) due to smoking cigarette-sized cigars and inhaling 20 of these daily. The mechanism by which such high levels were attained was primarily a combination of arterial hypoxia and a high carbon monoxide yield from tobacco. For a given level of carbon monoxide exposure, the hypoxic person will attain a higher carboxyhemoglobin level than will a person without hypoxia. (Auth. Abs. Mod.)

80–0354. Herzog, C. **Ovulationshemmer: Kardiovaskulare Komplikationen und zusatzliche Risikofaktoren. [Oral Contraceptives: Cardiovascular Complications and Additional Risk Factors.]** *Schweizerische Rundschau fur Medizin (PRAXIS)* 68(41): 1321-1329, October 9, 1979, German.

This is a review of case-controlled epidemiological studies of oral contraceptives and cardiovascular disease published in the U.S. and in West European journals from 1967 to 1979. The studies provide evidence of a statistical relationship between oral contraceptive usage and cardiovascular complications such as hypertension, myocardial infarction, and thromboembolic diseases. Relative risks, compiled from several retrospective and prospective studies, of cardiovascular disease and mortality in women using oral contraceptives are presented. Some studies attribute an important role not only to the estrogen component, but also to the progestogen component of the pill in altering blood vessel walls, blood coagulation, fibrinolysis, hemodynamics, and in developing hypertension. The synergistic effects of additional cardiovascular disease risk factors, such as smoking, are also considered to be very significant. Smoking is implicated in risk of arterial and venous thromboembolism. One study estimated the mortality risk of myocardial infarction to be 42 percent in pill users and smokers. Calculations of the anticipated mortality rate show that the combination of oral contraception and heavy smoking (over 15 cigarettes/day) could be more dangerous than the use of uncertain prophylactic methods in 15- to 29-year-old women.

80-0355. Huy, N. D.; Laliberte, D.; Samson, H.; Roy, P. E. **Tabagisme et Troubles Coronariens Dans la Region de Quebec. [Smoking and Coronary Disease in the Quebec Region.]** *L'Union Medicale du Canada* 106(8): 1110-1115, August 1977, French.

This research project investigates the relationship between smoking and coronary heart disease in the Quebec area. It was performed on a sample of 668 patients; 340 patients were suffering from coronary heart ailments, and 328 others were being treated for other illnesses. The latter group was designated as the control group. Results showed that 81 percent of the coronary disease group were smokers, while only 63 percent of the control group were smokers ($p<0.001$). Furthermore, the proportion of heavy (≥ 25 cigarettes/day) smokers aged 40 to 59 was 41 percent in the coronary group and only 20 percent in the control group. In the coronary group, 39 percent were heavy smokers and 45 percent were nonsmokers as compared to 19 percent and 21 percent, respectively, in the control group. Statistical analysis showed that these proportions were significantly different ($p<0.001$). An investigation of other factors such as sex and occupation is supporting evidence of a causal relationship between heart disease and heavy smoking. (Auth. Abs. Mod.)

80-0356. Kallio, V.; Hamalainen, H.; Hakkila, J.; Luurila, O. J. **Reduction in Sudden Deaths by a Multifactorial Intervention Programme After Acute Myocardial Infarction.** *Lancet* 2(8152): 1091-1094, November 24, 1979.

Three hundred and seventy-five consecutive patients below 65 years who had an acute myocardial infarction (AMI) took part in a randomized rehabilitation and secondary prevention trial (part of a WHO-coordinated project) designed to study the effects of a multifactorial intervention program (including regular medical examinations, health education, and a physical exercise program) on morbidity, mortality, and return to work. After 3 years' followup, the cumulative coronary mortality was significantly smaller in the intervention group than in the controls (18.6 percent vs. 29.4 percent, $p=0.02$). The number of cigarette smokers and cigarettes smoked were reduced by approximately 50 percent in both intervention and control groups; thus smoking habits did not account for the differences in mortality observed in this study. The difference was mainly due to a reduction of sudden deaths in the intervention group (5.8 percent vs. 14.4 percent, $p<0.01$). The reduction was greatest during the first 6 months after AMI. In the intervention group and in the controls, 18.1 vs. 11.2 percent ($p<0.10$) presented with nonfatal reinfarctions. The number of patients with new electrocardiographic findings at the end of the 3 years was, however, almost the same in both groups. The results suggest that organized aftercare during the first 6 months after AMI with special emphasis on optimum medical control and health education contributes significantly to a reduction in the number of sudden deaths. (Auth. Abs. Mod.)

80-0357. Koch, A.; Reuther, R.; Boos, R.; Reuther, E.; Moerl, H. **Risikofaktoren bei cerebralen Durchblutungsstorungen. [Risk Factors of Cerebral Blood Circulation Disorders.]** *Verhandlungen der Deutschen Gesellschaft fur Innere Medizin* 83: 1773-1776, April 1977, German.

Stroke risk factors were investigated in a group of 100 male acute stroke patients aged 40 to 69 years and in 143 controls. The patients and controls were subdivided into two age groups: 40-59 and 60-69 years. Cases of cardiac infarct and arterial occlusion were excluded. The stroke relative risk, expressed by the ratio of number of stroke patients to number of controls, increased from 1.0 for patients with systolic blood pressure <120 to 32.0 in the 40-59 years group and to 94.0 in the 60-69 years group for patients with systolic blood pressure >160. Similar increases in relative risk were determined in patients with increasing diastolic blood pressure, and increasing blood sugar, cholesterin and serum triglyceride levels. For all of these factors, the relative risk was higher in older patients than in younger ones. The stroke relative risk was 1.0 for nonsmokers and 11.2 for smokers of more than 20 cigarettes/day. In ex-smokers, the increased risk was not detectable after 1 year.

80-0358. Lancet. **Passive Smoking and Angina.** *Lancet* 2(8086): 413-414, August 19, 1978.

The effects of passive smoking on patients with heart disease are considered in this editorial. A previous study of 10 angina patients showed that passive smoking will significantly reduce the amount of exercise needed to induce angina. Patients exposed to three smokers (5 cigarettes each for 2 hours) in either a well-ventilated or in a nonventilated room had increased resting heart rate, blood pressure, and carboxyhemoglobin levels. These measurements were higher in the patients in the nonventilated room. It is recommended that patients with coronary heart disease stop smoking or avoid passive smoking. Further, hospitals should have well-defined smoking and nonsmoking areas to better protect the comfort and the health of patients. See also 78-1031.

80-0359. Logan, R. L.; Thomson, M.; Riemersma, R. A.; Oliver, M. F.; Olsson, A. G.; Rossner, S.; Callmer, E.; Walldius, G.; Kaijser, L.; Carlson, L. A.; Lockerbie, L.; Lutz, W. **Risk Factors for Ischaemic Heart-Disease in Normal Men Aged 40. Edinburgh—Stockholm Study.** *Lancet* 1(8071): 949-954, May 6, 1978.

A study was made of 107 healthy men in Edinburgh and 82 in Stockholm, all aged 40 and selected at random, to identify factors which might explain why, in men of this age, the mortality rates from ischemic heart disease are three times greater in Edinburgh men. Clinical measurements and electrocardiographic procedures were standardized, and steps taken to eliminate interlaboratory differences. Edinburgh men were shorter and fatter, had higher systolic and diastolic blood pressures, drank more alcohol, had more ECG abnormalities and lower exercise tolerance, and smoked more cigarettes (but the number of cigarette smokers in the two groups was similar). Of the Edinburgh men, 51 percent were smokers (compared to 40 percent of the Stockholm men) and 41 percent smoked more than 15 cigarettes/day compared to 22 percent of the men in Stockholm ($p<0.01$). Edinburgh men had higher levels of total serum-triglycerides and triglycerides in very low (VLDL), low (LDL), and high (HDL) density lipoproteins. Triglyceride clearance (assessed by an intravenous fat-tolerance test) was similar. Serum cholesterol and LDL-cholesterol concentrations were similar in both cities, but cholesterol in VLDL was higher, and cholesterol in HDL was lower in Edinburgh men. The relative linoleic-acid content of plasma triglycerides and of plasma cholesterol esters were lower in Edinburgh men, and their subcutaneous adipose tissue glycerides had a lower polyunsaturated/saturated fatty acid ratio and a strikingly lower relative linoleic acid content. Edinburgh men had a greater total plasma insulin response to a standardized glucose-tolerance test. The glucose response was the same in both cities. Plasma free fatty acids and free glycerol were reduced by a lesser extent in Edinburgh men in response to a glucose load. Although the high incidence of ischemic heart disease in Scotland may be due to many factors, some of which are well established, this study points out metabolic factors which may provide new information on the pathogenesis of ischemic heart disease. (Auth. Abs. Mod.)

80–0360. Mackay, A.; Brown, J. J.; Cumming, A.M.M.; Isles, C.; Lever, A. F.; Robertson, J.I.S. **Smoking and Renal Artery Stenosis.** *British Medical Journal* 2(6193): 770, September 29, 1979.

The relationship between smoking and renal artery stenosis was studied in 85 hypertensive patients (46 women, with a mean age of 41.5 years, and 39 men with a mean age of 43.9 years). All of the patients had arteriographic evidence of stenosis or occlusion of at least one renal artery and a diastolic blood pressure of 100 mm Hg or over. Hypertension was malignant in 22 patients (mean diastolic pressure 142 ± 14 mm Hg) and nonmalignant in 63 (mean diastolic pressure of 119 ± 14 mm Hg); 44 patients had stenosis of a main renal artery, and 18 had an occluded renal artery. The 63 nonmalignant hypertensive patients with renal artery stenosis were matched for age and sex with two control groups: a randomly selected group of 63 nonmalignant hypertensive patients (mean blood pressure of 122 ± 16 mm Hg), and 63 randomly selected ward patients with nonmalignant hypertension (mean diastolic pressure of 120 ± 14 mm Hg) who had no pyelographic evidence of a renal lesion. The smoking habits of the study and control groups were examined. Smoking was almost twice as common in patients with nonmalignant hypertension and renal artery stenosis as in the comparable controls. Of the 13 patients with renal artery occlusion, all were smokers, while 46 percent of the patients in one control group ($p<0.01$) and 38 percent of the patients in the other control group were smokers ($p<0.001$). Of 32 patients with atheromatous stenosis, 84 percent were smokers, compared with 47 percent of one control group and 50 percent of the other group who were smokers ($p<0.01$). There was a close association with smoking in patients with renal artery stenosis who had malignant hypertension (20 of 22 patients smoked). The prevalence of smoking in the two control groups was similar to other studies. This observation and the difference in smoking habits between the subgroups of renal artery stenosis patients and controls indicate that the observed excess of smoking in renal artery stenosis is probably genuine.

80–0361. Mackay, A.; Nias, B. C. **Strokes in the Young and Middle-Aged: Consequences to the Family and to Society.** *Journal of the Royal College of Physicians of London* 13(2): 106-112, April 1979.

From a total of 100 stroke patients age 65 or less, 90 patients who had had a cerebral hemorrhage or infarction were studied to examine factors which may predispose younger patients to stroke. Except for two teenage patients, patients were compared with controls matched for age and sex. Hypertension, obesity, family history, oral estrogens, smoking, and other conditions were considered. Of the 88 matched patients, 70 percent were smokers compared with 41 percent of the controls ($p<0.001$). There were more male than female smokers in both patient and control groups, but the percentage of female smokers in the patient group exceeded that of male smokers in the control group (59 percent vs. 48 percent). Smoking was also heavier among stroke patients than among controls; 68 percent vs. 44 percent smoked 20 or more cigarettes a day ($p<0.05$).

80–0362. Manthey, J.; Morgenstern, W.; Nuessel, E.; Opherk, D.; Stoeppler, M.; Wesch, H.; Kuebler, W. **Spurenelemente im Serum und in Erythrozyten bei Patienten mit koronarer Herzkrankheit. [Trace Elements in Serum and Erythrocytes of Patients with Coronary Heart Disease.]** *Verhandlungen der Deutschen Gesellschaft fur Innere Medizin* 83: 1767-1769, April 1977, German.

The relationship between trace element levels in blood and known coronary heart disease risk factors was investigated in 83 patients with important alterations of coronary vessels and in 31 controls. The trace elements zinc (Zn), magnesium (Mn), chromium (Cr), and copper (Cu) in serum and lead (Pb) and cadmium (Cd) in whole blood were determined by atomic absorption spectroscopy. Selenium (Se) and rubidium (Rb) trace levels in serum were determined by neutron activation analysis. A significant ($p<0.002$) correlation was found between Mn level and diastolic blood pressure and between Rb ($p<0.1$), Mn ($p<0.05$), and Cu ($p<0.05$) levels and cholesterol content in serum. The blood Cd level in nonsmokers was 0.4 ng/ml, while in heavy smokers (>20 cigarettes/day) it was 5 to 15 times higher. Ex-smokers also had an elevated Cd level for as long as 1 year after smoking withdrawal. The Mn and Cu levels were significantly ($p<0.05$) correlated with the degree of coronary heart disease computed as a Gensini score. A step-wise linear discriminatory analysis indicated that the serum Mn level could be used to screen patients for coronary heart disease.

80-0363. Merx, W.; Effert, S.; Wolfers, J. **Risikofaktoren bei Patienten mit akutem Myokardinfarkt—prognostische Bedeutung und langfristiger Trend. [Risk Factors in Patients with Acute Myocardial Infarction—Prognostic Significance and Long-Term Trend.]** *Verhandlungen der Deutschen Gesellschaft fur Innere Medizin* 83: 1725-1727, April 1977, German.

A followup study was made of 140 men and 30 women (average age 59.4 years) who had been hospitalized with acute myocardial infarction between January 1969 and December 1972. In the spring of 1976, the surviving 113 patients were reexamined for hypertension, hyperlipemia, smoking (more than 10 cigarettes/day), and diabetes mellitus. Of the survivors, 24 had experienced a second infarction. Among the 81 patients who subsequently died or suffered a reinfarction, no significant difference was detected during the acute phase between the number of those with either hypertension, smoking, or hyperlipemia, and those without these risk factors. The only significant difference was found for diabetics. At the followup examination, a drastic decrease in the proportion of cigarette smokers from 76 percent in the acute phase to 11 percent was noted. At the same time, the percentage of patients with a reinfarction was higher in the presence than in the absence of each of the four risk factors, although this difference was only significant for hyperlipemia. Patients with only one or no risk factor experienced a reinfarction significantly less frequently than those with several risk factors. It is concluded that the prognosis after a myocardial infarction is independent of the risk factors in the acute phase, with the possible exception of diabetes mellitus.

80-0364. Morrison, J. A.; Kelly, K.; Mellies, M.; deGroot, I.; Khoury, P.; Gartside, P. S.; Glueck, C. J. **Cigarette Smoking, Alcohol Intake, and Oral Contraceptives: Relationships to Lipids and Lipoproteins in Adolescent School-Children.** *Metabolism: Clinical and Experimental* 28(11): 1166-1170, November 1979.

The effects of cigarette smoking, alcohol intake, and oral contraceptives on plasma cholesterol, triglyceride, high density lipoprotein cholesterol (C-HDL), and low density lipoprotein cholesterol (C-LDL) were assessed in 965 12-19-year-old school children in the Cincinnati Lipid Research Clinic's Princeton school survey. Among those surveyed, 144 reported smoking cigarettes (53 smoked 10 or more cigarettes per day), 63 reported consumption of alcoholic beverages, and 13 reported taking oral contraceptives. After pair matching for age, sex, race, and total plasma cholesterol, adolescent children who smoked had mean C-HDL 6.1 mg/dl lower, and mean C-LDL 4.1 mg/dl higher, than nonsmokers ($p<0.01$). These findings for C-HDL were replicated by covariance analysis, adjusting for age, race, sex, alcohol intake, and triglyceride levels. Adolescents who drank alcohol had higher C-HDL and triglyceride levels and lower C-LDL than nondrinkers, but the differences were not significant. Adolescent females taking oral contraceptives had higher triglyceride, C-HDL, and C-LDL levels than matched controls, but the differences were not significant. If a portion of smoking's contribution to coronary heart disease risk is mediated through its inverse association with C-HDL, and if smoking habits initiated in adolescence continue into adulthood, this report provides additional physiologic data relevant to programs designed to prevent, reduce, or stop cigarette smoking in the adolescent years. (Auth. Abs. Mod.)

80-0365. Onat, A. **Ilk Miyokard Infarktusunu Atlatan Kisilerde Uzun Sureli Prognoz. [Long Term Prognosis of Survivors of Initial Myocardial Infarction.]** *Istanbul Universitesi Istanbul Tip Fakultesi Mecmuasi* 40(1): 50-62, 1977, Turkish.

A report based on the followup study of 66 cases of myocardial infarction (MI) is given. All cases had recovered from the initial MI within 1-1/2 months; followup periods extended up to 11 years in some cases, amounting to a total of 247 patient-years. The patient population was considered to be representative with regard to age and several risk factors. Average annual mortality was found to be 6 percent. Factors such as preexisting coronary heart disease, association of cardiomegaly or heart failure, and a history of smoking and coexisting arterial hypertension elevated mortality. Localization of infarction to the inferior wall is associated with a threefold greater survival rate compared to other locations. (Auth. Abs. Mod.)

80-0366. Ponteva, M.; Elomaa, I.; Backman, H.; Hansson, L.; Kilpio, J. **Blood Cadmium and Plasma Zinc Measurements in Acute Myocardial Infarction.** *European Journal of Cardiology* 9(5): 379-391, May 1979.

Blood cadmium and plasma zinc were measured in 47 patients with myocardial infarction (MI) and in 37 control subjects. There was no significant difference between MI patients and controls with respect to sex and smoking habits (47 percent of the patients vs. 43 percent of the controls were smokers). Increased blood cadmium values were found in 33 percent of the MI patients. The mean blood cadmium value of the MI patients was significantly ($p<0.001$) higher than that of the control subjects. The difference between the zinc values of the subjects was studied in 35 MI patients with raised serum enzyme activity. In MI patients, the mean plasma zinc content was significantly ($0.001<p<0.01$) lower than in healthy controls. The correlation between blood cadmium and smoking was low, possibly because those classified as smokers included ex-smokers who had not been smoking for 5 years prior to the infarction. Of the enzymes, the highest positive correlation was found between the ratio of blood cadmium to plasma zinc and the glutamic oxaloacetic transaminase activity. With respect to the ratio of blood cadmium to plasma zinc, 43 percent of MI patients had values in excess of the controls and 40 percent had values within the limits of the controls. Therefore, the rise in the ratio is of limited value for the diagnosis of myocardial infarction in an individual patient. (Auth. Abs. Mod.)

80–0367. Puska, P.; Tuomilehto, J.; Salonen, J.; Neittaanmaki, L.; Maki, J.; Virtamo, J.; Nissinen, A.; Koskela, K.; Takalo, T. **Changes in Coronary Risk Factors During Comprehensive Five-Year Community Programme to Control Cardiovascular Diseases (North Karelia Project).** *British Medical Journal* 2(6199): 1173-1178, November 10, 1979.

A comprehensive community program to control cardiovascular diseases (CVD) in North Karelia, Finland, was carried out between 1972 and 1977. The objective of the program was to reduce prevalence of smoking, serum cholesterol concentration, and raised blood-pressure values among the population of the area. The effect was evaluated by examining independent representative population samples in 1972 and 1977 in both the county of North Karelia and a matched control county. Over 10,000 subjects were studied each time, the participation rate being around 90 percent. The decrease that occurred in the risk factors, especially in men, was in general greater in North Karelia compared with the control county. There was a significant net reduction of 9.8 percent in smoking among men in North Karelia ($p<0.05$); a smaller reduction occurred among women. When a multiple logistic function was used for the three risk factors an overall mean net reduction of 17 percent among men and 12 percent among women was observed in the estimated risk for coronary heart disease in North Karelia. This community program effectively reduced the levels of the three main risk factors for CVD in the population, and thus mortality and morbidity from CVD should fall. (Auth. Abs. Mod.)

80–0368. Ramsay, L. E. **Intermittent Claudication in Hypertensive Men.** *Journal of the Royal College of Physicians of London* 13(2): 100-102, April 1979.

This study reports the incidence of intermittent claudication in hypertensive men attending hospital clinics and examines associated factors such as smoking, high blood pressure, stroke, and myocardial infarction. Each patient with claudication had a control matched for sex, age (same decade), hospital, and hospital number. Smoking habits were designated by four categories: current cigarette smokers, ex-smokers, pipe or cigar smokers, and those who had never smoked. Of 1,357 hypertensive men, 75 (5.5 percent) had probable or definite intermittent claudication. The incidence was clearly related to increasing age and to smoking. In patients over 40 years of age, cigarette smokers had a relative risk for claudication of 4.6 ($p<0.001$) and ex-smokers a relative risk of 4.5 ($p<0.01$) compared to those who had never smoked. Pipe or cigar smokers had a nonsignificant increase in risk. The patients with claudication and matched hypertensive controls were compared. The relationship with current and previous cigarette smoking was confirmed ($p<0.01$).

80–0369. Rebel-De Vries, H. A.; Bruschke, A.V.G. **Coronaria-Angiografische Bevindingen bij Vrouwen in de Vruchtbare Levensfase; het Verband Tussen Klinische Verschijnselen en Risicofactoren. [Findings Obtained by Coronary Arteriography in Women in the Reproductive Age Group With Ischemic Cardiac Disease; Correlation Between Clinical Symptoms and Risk Factors.]** *Nederlands Tijdschrift Voor Geneeskunde* 123(28): 1202-1206, July 14, 1979, Dutch.

In a period of 3 1/2 years, coronary arteriography was carried out in 126 women aged 45 years or less. In 61 premenopausal women, this examination was performed because of suspected ischemic heart disease. Hemodynamically significant coronary obstructions were encountered in 34 patients; this number accounts for only 3 percent of the total number of patients with coronary obstructions who were examined during the same period. The correlation between the angiographic abnormalities and the clinical picture in these women was the same as that in the total group of patients (men and women). The risk factors hypertension, hypercholesterolemia, and smoking were present more frequently than in the average female population of this age group. Of the 34 patients, 28 were smokers: 13 smoked more than 20 cigarettes/day. No association with the use of oral contraceptives could be demonstrated. (Auth. Abs.)

80–0370. Renaud, S.; Dumont, E.; Godsey, F.; Suplisson, A.; Thevenon, C. **Platelet Functions in Relation to Dietary Fats in Farmers From Two Regions of France.** *Thrombosis and Haemostasis* 40(3): 518-531, February 15, 1979.

Platelet aggregation (to thrombin, adenosine diphosphate (ADP), collagen, epinephrine) and clotting activity of platelet-rich plasma (PRP), platelet-poor plasma, and of washed platelets, were studied in relation to lipemia, glycemia, dietary nutriments, and platelet phospholipid composition, in order to determine whether long-term feeding of dietary fats affected platelet functions in humans. The study subjects were 44 healthy male farmers (aged 40-45) from 2 French regions, Var and Moselle. Farmers who smoked more than 20 cigarettes per day and farmers who were heavy drinkers (more than 100 g of alcohol) were eliminated. When the results from smokers were compared with those from nonsmokers, there were no significant differences in the parameters examined (coagulation times, aggregation, cholesterol, and systolic blood pressure). In the Moselle subjects, the platelet clotting activity of PRP and of washed platelets, the platelet aggregation to thrombin and ADP, were significantly ($p<0.001$) increased as compared to those from Var, but not the plasma cholesterol, which was identical in the two regions. In the Moselle region, the intake of total calories, total lipids, and saturated fats was higher than in the Var region. However, it was only with the saturated fat intake (mostly stearic acid) that the platelet clotting activity ($p<0.01$) and the platelet aggregation ($p<0.001$) were significantly correlated. The platelet clotting activity was also significantly ($p<0.001$) correlated with the fatty acid composition of the platelet phospholipid fractions phosphatidyl serine and phosphatidyl inositol.

80-0371. Robertson, F. W.; Cumming, A. M. **Genetic and Environmental Variation in Serum Lipoproteins in Relation to Coronary Heart Disease.** *Journal of Medical Genetics* 16(2): 85-100, April 1979.

Variation in the serum concentration of very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoprotein (LDL), and high density lipoprotein (HDL) was studied in: (1) 192 survivors of myocardial infarction under the age of 50 living in northeastern Scotland; (2) 250 relatives, mostly first-degree; and (3) 259 unrelated individuals, comprised mostly of spouses and their relatives. In regular smokers, the VLDL levels were substantially and significantly higher in both sexes, by about 30 percent in males and 25 percent in females. The proportional increase was equally evident in the concentration of either VLDL cholesterol or VLDL triglyceride. The LDL concentration was significantly higher in women smokers, but not so in men. For HDL cholesterol, though the differences were statistically insignificant in either sex, the smokers tended to have lower values, reflecting the familiar inverse relationship between VLDL and HDL cholesterol. Of male index cases, 87.7 percent were categorized as smokers, though most had given up the habit after their infarction. Of male controls and index case relatives combined, 72.8 percent were classed as smokers, and the difference was statistically significant ($0.01<p<0.02$). For the smaller sample of female index cases, the proportion of smokers was 88.2 percent compared with 46.8 percent in female controls and index relatives ($p<0.001$). The smoking habit was common in this population, and the average number of cigarettes smoked per day by regular smokers was 19.3 ± 0.03 in men and 16.0 ± 1.0 in women, whose mean ages when they started smoking were 16 and 18 years, respectively. The high correlation between VLDL and measures of fatness, for which heritability estimates were statistically insignificant, as well as the association between raised VLDL concentration and smoking, provided confirmatory evidence of the major importance of nongenetic causes in the variation of VLDL. The results are discussed in relation to the origin of the effects of smoking, variation in proneness to coronary disease, and the biological significance of differences in HDL concentration. (Auth. Abs. Mod.)

80-0372. Rosenfield, A. **Oral and Intrauterine Contraception: A 1978 Risk Assessment.** *American Journal of Obstetrics and Gynecology* 132(1): 92-106, September 1, 1978.

This report reviews the current literature on the various risks associated with the use of oral contraceptives and intrauterine contraceptive devices. Reports on oral contraceptives from large prospective studies are now beginning to supplement the detailed reports resulting from earlier case-control studies. These studies suggest that in Western societies there is an increase in the incidence of a variety of circulatory diseases, with an increased risk of death. Factors such as smoking are important when weighing the risk of complications from oral contraceptives. Annual mortality rates (per 100,000 women) from venous thromboembolism by pill use, smoking habits, and age, have shown an increased risk of thromboembolic phenomena among heavy smokers, particularly women over the age of 40. A study of myocardial infarction (MI) risk factors (hyperlipoproteinemia, hypertension, smoking more than 15 cigarettes per day, diabetes, and oral contraceptive use) indicated that individuals with two or more risk factors were more common among MI cases than among controls. The combined effect of the risk factors was found to be synergistic, and the presence of 3 or more risk factors was estimated to increase the risk of MI 128-fold. Studies which focused attention on the importance of cigarette smoking as a MI risk factor demonstrated a significant difference between control cases and MI cases when smoking and pill use were combined. The annual mortality rate in women (especially those over 40) with MI was directly influenced by these two factors. With intrauterine contraceptive devices it now has been fairly well documented that there is an increased risk of pelvic inflammatory disease. After reviewing and assessing the risks of both methods in some detail and comparing these risks to the benefits, the resultant conclusion is that the benefits continue to outweigh the risks for both methods, except for older women who are both oral contraceptive users and heavy smokers. (Auth. Abs. Mod.)

80-0373. Stamler, J. **Lifestyles, Major Risk Factors, Proof and Public Policy.** *Circulation* 58(1): 3-19, July 1978.

Major risk factors in coronary heart disease were reviewed, with particular emphasis on the role of nutrition. Preventive measures for coronary heart disease were also discussed. International epidemiologic data indicated that reducing or eliminating certain risk factors, such as a high cholesterol diet and cigarette smoking, can reduce the risk of premature coronary heart disease. Multivariate analyses, including the Framingham Study, showed significant relationships between coronary heart disease rates and serum cholesterol, blood pressure, and cigarette smoking. The relationship of serum cholesterol to risk, while simultaneously controlling for blood pressure and cigarette smoking, was shown to be independently significant in the Pooling Project. Data from animal and human studies indicated that high blood pressure and cigarette smoking were minimally significant for atherogenesis in the absence of nutritional and metabolic prerequisites.

The Multiple Risk Factor Intervention Trial suggested that it was possible to reduce the incidence and mortality from arteriosclerosis in men in the upper 10 percent of risk by lowering their serum cholesterol, blood pressure, and cigarette smoking. Since 1968, there has been a steady decline in coronary heart disease mortality rates in the United States for persons aged 35-74. Trends indicate that Americans are becoming more concerned about healthy diets and healthier lifestyles. Per capita consumption of cigarettes sharply decreased in 1967-1969, probably due to preventive measures by health, educational, and government agencies. Recommended coronary heart disease preventive measures include dietary changes (to prevent or control hyperlipidemia, obesity, hypertension, and diabetes), elimination of cigarette smoking, and drug control of hypertension.

80–0374. Stephan, E. **Kombination Schirmbild—Blutdruckmessung. Erfahrungen und Ergebnisse im Kanton Bern. [Radiophotography—Blood Pressure Measurement Combination. Experiences and Results in the Canton of Bern.]** *Schweizerische Medizinische Wochenschrift* 109(7): 234-243, February 17, 1979, German.

Results are presented from the 1977 screening of 17,112 persons 30 years of age and older for hypertension and its association with pathologic heart conditions and smoking. Blood pressure measurements and mass radiography were used in the early detection of hypertension in cases requiring control or therapy. A questionnaire distributed to participants at the time of screening obtained information concerning blood pressure history and smoking habit. The data showed that 14.6 percent of the screened population had blood pressure readings above the 160/95 mm Hg level defined by WHO as hypertension. Diastolic pressure over 95 mm Hg was found in 5.9 percent and over 110 mm Hg was found in 1.8 percent of the examined population. Among the examined hypertensives and treated patients, 41.7 percent vs. 33.8 percent, respectively, were smokers. Of the total examined population, 31.5 percent smoked. Of those screened 8.9 percent had heart pathology and were smokers and 3.4 percent had heart pathology and hypertension and were smokers. The possibility of the simultaneous detection of smoking and overweight as additional risk factors for hypertension is cited as one of the advantages of preventive screening.

80–0375. Streja, D.; Mymin, D. **Moderate Exercise and High-Density Lipoprotein-Cholesterol. Observations During a Cardiac Rehabilitation Program.** *Journal of the American Medical Association* 242(20): 2190-2192, November 16, 1979.

The effects of a 13-week moderate exercise program on fasting plasma insulin, lipids, and lipoprotein cholesterol concentrations were studied in 32 sedentary, middle-aged men with coronary artery disease. Twenty-eight subjects were nonsmokers. Among those, 24 had a history of smoking, which was discontinued at least 3 months before the study. One of the four men quit smoking during the study. The preponderant component of the exercise program was walking or slow jogging. There was no significant change in the systolic blood pressure and pulse rate product response to a standard exercise load. The high density lipoprotein cholesterol (HDL-C) level increased, and the fasting plasma insulin concentration decreased. There were no significant changes in plasma triglycerides or low-density lipoprotein cholesterol levels. In sedentary subjects with coronary artery disease, a modest increase in activity can result in an increase in the HDL-C level and a decrease in the plasma insulin concentration. These changes occurred in the absence of variations in diet, smoking habit, adiposity, or plasma triglyceride concentrations and did not require a cardiovascular training effect. (Auth. Abs.)

80–0376. Velasco Rami, J. A.; Tormo Alfonso, V. **Rehabilitacion de Pacientes con Infarto de Miocardio. Resultados al Ano de Seguimiento. [Rehabilitation of Myocardial Infarct Patients. Results of a One-Year Followup.]** *Revista Espanola de Cardiologia* 30(6, Part 2): 695-704, 1977, Spanish.

In order to compare the results obtained with the rehabilitation and secondary prevention techniques of myocardial infarcts (MI), a study was made of a total of 115 male patients who had suffered a MI 3 weeks earlier and who were grouped into an intervention group (65 cases) and a control group. During their stay in the hospital (3 weeks), all underwent the same exploration, treatment, and premature mobilization methods. After being discharged, the patients in the rehabilitation group attended the clinic in order to carry out supervised exercises. One year after having suffered the MI, all the patients underwent the same clinical, radiological, analytical, psychological, and ergometric explorations. Special attention was also paid to the work situation and changes in lifelong habits, with reference, in particular, to tobacco. In the ergometric control carried out 1 year after the MI, the patients in the rehabilitation group possessed a greater physical capacity than those in the control group (101.86 and 88.69 watts, respectively), and this difference was significant. There were no differences within each group in the physical capacity as far as the location of the infarct or the kind of work were concerned. In neither of the two groups was there any appraisable change in heart diameter in the year's observation. A higher percentage (80.3 percent) of the patients in the rehabilitation group returned to work than those in the control group (66.7 percent), and the frequency with which extramedical causes affected the nonreturn to work was also noticeable. A total of 81.9 percent of the patients in the rehabilitation group gave up smoking compared with 64.4 percent of the control group. This difference was significant and confirms the effectiveness of the repetition of advice on tobacco. With reference to the mortality, although this was greater in the control group (10 percent) than in the rehabilitation group (6.1 percent), the difference was not statistically significant. (Auth. Abs.)

best copy available

80–0377. Vessey, M. P.; McPherson, K.; Johnson, B. **Mortality Among Women Participating in the Oxford/Family Planning Association Contraceptive Study.** *Lancet* 2(8041): 731-733, October 8, 1977.

Forty-three deaths are known to have occurred among the 17,032 participants in the Oxford/Family Planning Association contraceptive study up to the end of April 1977. Nine deaths from cardiovascular causes have been observed among the women in the oral-contraceptive entry group (49,681 woman-years of observation) while no such deaths have been observed among the women who entered the study while using a diaphragm or an intrauterine device (39,146 woman-years of observation). In general, the women in the oral contraceptive group compare favorably with the women in the other groups in regard to the distribution of cardiovascular disease risk factors, except as to smoking. Although only a small proportion of the women smoked heavily (15 or more cigarettes/day), 17 percent of the oral contraceptive users were heavy smokers, compared to 7 and 12 percent, respectively, of the women using diaphragms or intrauterine devices. These findings are consistent with the results presented in the report from the Royal College of General Practitioners Oral Contraception Study. (Auth. Abs. Mod.)

80–0378. Wakeham, H.R.R. **Environmental Carbon Monoxide From Cigarette Smoking.** In: *Proceedings of the Sixth International Tobacco Scientific Congress*, Tokyo, November 14-20, 1976, The Japan Tobacco and Public Salt Corporation, 1977, pp. 93-101.

The current drive for bans on smoking in public places and in work places is based on the assertion that tobacco smoke in the atmosphere constitutes a health hazard to exposed nonsmokers. It is said that the hazard arises mainly from carbon monoxide generated during the burning of a cigarette. Cigarette smoking is an insignificant source of carbon monoxide in the atmosphere as compared to other natural and manmade sources. Even in tightly closed spaces with a large percentage of smokers, only rarely is it possible to build up concentrations which would exceed the established threshold limiting values for extended exposures. Examples including meeting rooms, houses, buses, and aircraft are discussed. The resulting carboxyhemoglobin levels in nonsmokers are considered. It is concluded that the carbon monoxide from environmental tobacco smoke is well below the amount needed to produce the maximum allowable limit of 4 percent carboxyhemoglobin in the blood as suggested by the World Health Organization. (Auth. Abs.)

80–0379. Wallace, R. B.; Tamir, I.; Heiss, G.; Rifkind, B. M.; Christensen, B.; Glueck, C. J. **Plasma Lipids, Lipoproteins, and Blood Pressure in Female Adolescents Using Oral Contraceptives.** *Journal of Pediatrics* 95(6): 1055-1059, December 1979.

Comparative profiles are reported for plasma lipids and lipoproteins, blood pressure, smoking, and selected socioeconomic variables for females less than 20 years of age using oral contraceptives (OC) and for pair-matched nonusers from the Lipid Research Clinics Prevalence Study. About 5 percent of adolescent females reported OC use; they had significantly higher levels of plasma total cholesterol, triglyceride, and high density lipoprotein cholesterol than did the nonusers. Significantly more nonusers of OC belonged to households of the lowest occupational status; no differences in OC usage by educational status of the head of household were discernible. Significantly more OC users (58 percent) than nonusers (23 percent) were classified as current smokers. (Auth. Abs.)

80–0380. Watson, D. S. **Health Education for Hypertensive Patients.** *Australian Family Physician* 8(3): 315-320, March 1979.

In order to determine whether a health education program on hypertension would improve a patient's chances of lowering blood pressure and maintaining health, 118 asymptomatic hypertensive patients were invited to participate in a clinical trial. The patients were placed in four groups: routine general practitioner (GP) treatment (controls), GP treatment plus health education program, GP treatment plus daily self-monitored blood pressure (BP), and GP treatment plus daily monitoring plus health education program. The program was prepared by a consortium of adult educators, health educators, dieticians, nurses, and doctors. Volunteer lay educators were extensively trained to run the four program classes. The classes lasted 60 to 90 minutes, and were limited to 10 to 12 people. Attendance was 54 percent for 4 meetings and 22 percent for 3 meetings. Questioned about their attitudes toward hypertension, patients named lack of fitness (95 percent), dietary factors (72 percent), stress (93 percent), excess weight (92 percent), heavy smoking (83 percent), light smoking (40 percent), and regular heavy drinking (84 percent) as important health factors. Of the 18 smokers who attended the health education program, 4 reduced their smoking and 5 stopped smoking. Of the 11 smokers in the daily monitoring and control groups, 1 stopped smoking and 1 reduced smoking. In the control group, 19 of 30 patients reduced their BP; in the health education program group, 26 of 32 patients reduced their BP; in the daily monitoring group, 17 of 26 patients reduced their BP; and in the daily monitoring plus health education program group, 23 of 30 patients reduced their BP.

See also, **80–0272, 80–0293**

PREGNANCY AND INFANT HEALTH

80–0381. Akkermann, S.; Grindel, B.; Seidenschnur, G. **Zur Epidemiologie der Fruhgeburtlichkeit Berufstatiger. [Epidemiology of Premature Birth in Working Women.]** *Zentralblatt fur Gynaekologie* 100(18): 1153-1161, 1978, German.

The risk factors for premature birth in working women vs. housewives are discussed as part of the research project 'Perinatology' of the East German Ministry of Health. The effects of the mother's age, number of previous births, education, full or part-time employment, and different social and environmental factors on the risk for premature birth were examined in various areas of the German Democratic Republic. Data compiled from 1975 to 1977 interviews paired mothers of premature and full-term children by age and parturition in order to compare the effects of maternal body weight and smoking habit on prematurity. In a group of 31 pairs of normal or overweight mothers, 10 mothers of premature children were smokers compared to only 5 mothers of full-term children. It was estimated that about one-half of the premature births in married working women could be attributed to nicotine abuse and/or relative underweight.

80–0382. Breart, G.; Hennequin, J.-F.; Crost-Deniel, M.; Rumeau-Rouquette, C. **Etude de l'Insuffisance Ponderale a la Naissance. A Partir d'un Echantillon Representatif de Nouveau-Nes. [A Study of Insufficient Weight at Birth. From a Representative Sample of Newborns.]** *Archives Francaises de Pediatrie* 34(7, Supplement 2): 221-232, August-September 1977, French.

In a representative sample of 11,330 newborns the distribution of birthweights was established in terms of gestational age. The comparison with Lubchenco's curves shows important differences since the 10th percentile of the American curve is below the 3rd percentile of the French sample for prematures. The use of a technique of multivariant analysis distinguishes groups with low and high risks of prematurity and hypotrophy. The distinction is more obvious for the first group than for the second. The study of the rate of early neonatal mortality in terms of weight and gestational age led to differentiation between the risk related to hypotrophy and that related to prematurity. Among sociocultural risk factors considered were marital status, paternal socio-professional category, maternal education, and smoking habits. There was no significant difference in prematurity incidence among nonsmokers, smokers who stopped smoking during pregnancy, and smokers who continued smoking during pregnancy. However, there was a significant difference in hypotrophy incidence ($p<0.01$) for smokers who stopped smoking during pregnancy. There was no significant difference in prematurity incidence for nonsmokers, smokers of less than 10 cigarettes/day, and smokers of 10 or more cigarettes/day. However, there was a significant ($p<0.001$) difference in hypotrophy incidence for smokers of less than 10 cigarettes/day. (Auth. Abs. Mod.)

80–0383. Gordon, Y. B.; Lewis, J. D.; Pendlebury, D. J.; Leighton, M.; Gold, J. **Is Measurement of Placental Function and Maternal Weight Worth While?** *Lancet* 1(8072): 1001-1003, May 13, 1978.

A prospective survey of 1,029 women who delivered during 1976 at St. Bartholomew's Hospital in London was undertaken to assess the predictive value of a variety of clinical, ultrasonic, and biochemical variables in the detection of high-risk pregnancies. Among the variables considered were levels of human placental lactogen (HPL), maternal weight, and smoking. The main objective of this study was to define the role of biochemical monitoring in fetal risk prediction. Three statistical descriptors were used to assess the clinical value of the risk factors: relative risk, predictive value, and sensitivity. High HPL values were associated with a low relative risk (0.2), whereas consistently depressed values were associated with increased risk (2.9). Therefore, low HPL values indicated higher risks, but the predictive value was only 26 percent. While nonsmokers had a relative risk of 0.6 ($p<0.01$), the relative risk increased to 1.4 for smokers of 1 to 15 cigarettes/day and to 2.0 for smokers of >15 cigarettes/day; the predictive values were 8, 12, and 18 percent, respectively. The best prenatal predictors of fetal risk were severe preeclampsia, low maternal HPL levels, low maternal weight at 32 weeks gestation, and heavy maternal smoking. Of the high-fetal-risk group, 62 percent had one or more of these features, but so did 33 percent of the controls.

80–0384. Lehtovirta, P. **Tupakointi ja Raskaus. [Smoking and Pregnancy.]** *Duodecim* 95(1): 5-7, 1979, Finnish.

The harmful effect of tobacco use during with pregnancy is in direct proportion to the number of cigarettes smoked per day. Nicotine acts as a vasoconstrictor on the uterine blood vessels and with carbon monoxide and cyanide causes hypoxia in the fetus with both direct and long-term effects. When the cyanide is converted to thiocyanate, vitamin B12 is consumed. The thiocyanate level of the blood of smoking mothers and their fetuses is elevated. Cyanide also acts directly as a cell toxin because it inhibits cytochrome oxidase and the carbonic acid anhydrase of erythrocytes. Smoking slows down the growth of the fetus and may also cause some retardation of the mental development. No direct teratogenic effects have been found. The incidence of heart diseases is elevated in fetuses of smokers. The frequency of miscarriages and hemorrhages doubles. Pregnant women who smoke have a lower frequency of toxemia and hypertension, possibly because smoking has caused miscarriage in some of the women who would otherwise suffer from such ailments.

80-0385. Meberg, A.; Haga, P.; Sande, H.; Foss, O. P. **Smoking During Pregnancy—Hematological Observations in the Newborn.** *Acta Paediatrica Scandinavica* 68(5): 731-734, September 1979.

Hematological values were measured in 28 newborn infants of mothers smoking 10-20 cigarettes daily during pregnancy, and in 25 infants of nonsmokers. Maternal serum thiocyanate concentration at delivery (mean±S.D.) was significantly elevated in smokers compared to nonsmokers (109.6 ± 29.9 μmol/l and 48.2 ± 16 μmol/l, respectively; $p<0.01$), and the thiocyanate concentration correlated positively to the maternal smoking level ($r=0.80$, $p<0.001$). Higher hematocrit levels ($p<0.05$) were found on the first day of life in infants of smoking mothers (60.8 ± 5 percent) compared to controls (57.5 ± 4.8 percent). The hematocrit levels correlated positively with the maternal smoking level ($r=0.318$, $p<0.05$) and the maternal serum thiocyanate concentrations at delivery ($r=0.389$, $p<0.01$). Cord serum values for erythropoietin, serum iron, transferrin, and ferritin showed no statistically significant difference between the two groups. A significant inverse correlation was found between the hematocrit value on the first day of life and the cord serum ferritin concentration ($r=-0.495$, $p<0.005$). The present results suggest that maternal smoking stimulates fetal erythropoiesis, probably through a hypoxic effect on the fetus, dose related to the maternal smoking level. Increased erythropoiesis may cause increased iron incorporation into erythrocytes at the expense of iron storage in the bone marrow and reticuloendothelial system. (Auth. Abs. Mod.)

80-0386. Roels, H. A.; Hubermont, G.; Buchet, J. P.; Lauwerys, R. R. **Placental Transfer of Lead, Mercury, Cadmium, and Carbon Monoxide in Women. III. Factors Influencing the Accumulation of Heavy Metals in the Placenta and the Relationship Between Metal Concentration in the Placenta and in Maternal and Cord Blood.** *Environmental Research* 16(1/3): 236-247, July 1978.

Concentrations of lead, mercury, and cadmium were determined in placentas from 474 European women and were compared with the levels found in maternal and newborn blood. The influence of some epidemiological factors (residence, smoking, drinking habit, age, occupation, previous pregnancies) on heavy metal accumulation in the placenta was also investigated. Of the pregnant women, 333 were nonsmokers and 109 were smokers (>3 cigarettes). The median values of the three heavy metals in the placentas were 7.5, 1.06, and 1.08 μg/100 gm (wet weight) for lead, mercury, and cadmium, respectively. In comparison with maternal blood, the placenta does not concentrate lead nor mercury but concentrates cadmium about 10-fold. Cadmium concentration in the placentas was significantly correlated with that in maternal blood ($r=+0.38$); for lead the correlation was lower although still statistically significant ($r=+0.22$); for mercury the level in the placentas was not significantly correlated with the metal concentration in maternal blood. Among the three heavy metals, only cadmium shows an increased accumulation in the placentas of smokers. No significant effect of current residence, maternal age, and occupation on the accumulation of the heavy metals in placenta was observed. (Auth. Abs. Mod.)

80-0387. Rush, D. **Cigarette Smoking During Pregnancy: The Relationship With Depressed Weight Gain and Birthweight. An Updated Report.** In: Kelly, S.; Hook, E. B.; Janerich, D. T.; Porter, I. H. (Editors). *Birth Defects: Risks and Consequences.* Conference Proceedings, Academic Press, New York, 1976, pp. 161-172.

An updated report of a study of 221 mothers and their singleton live-born infants who participated in a longitudinal study of nutritional supplementation in pregnancy is presented. Data for the following variables were available: prepregnant weight, weight at final prenatal clinic visit, length of gestation, birthweight of delivered infant, and number of cigarettes smoked per day (0, <5, 5-9, 10-14, 15+) at registration. The women had no known medical problems. Those who reported cigarette smoking at registration ($n=84$) had significantly lower subsequent mean weight gain than nonsmokers ($n=137$) (0.91 ± 0.42 lbs/week vs. 1.02 ± 0.41 lbs/week, $p<0.05$). There was a strong and highly significant decreasing gradient of weekly weight gain with the amount of reported smoking (0.13 fewer lbs/week gained per additional cigarette smoked, $p<0.005$). Infant birthweights were also significantly lower in smokers than in nonsmokers (3107 gm vs. 2834 gm, $p<0.001$). The gradient of decreasing birthweight for smokers was 15.7 fewer grams of weight at birth per additional cigarette smoked ($p<0.005$). Those who stopped smoking before delivery had infants of higher birthweight, but this difference was not significant ($p<0.10$). Gestation in smokers was 0.5 weeks less than in nonsmokers. Multiple regression analyses were performed. Decreased weight gain and caloric restriction which are associated with increased cigarette smoking in pregnancy are probable important mechanisms in the effect of cigarette smoking on depressed fetal growth.

80-0388. Schwartz, D. **Description de l'Approche Epidemiologique. Un Exemple: Le Role du Tabac. [Description of the Epidemiologic Approach. An Example: The Role of Tobacco.]** *Archives Francaises de Pediatrie* 35(Supplement 1): 15-18, May 1978, French.

The article discusses the use and value of epidemiologic methods used in the specific case of the effect of smoking during pregnancy on fetal diseases, mortality, birthweight, and maternal well-being. A great number of studies have demonstrated that there is a definite strong correlation between smoking during pregnancy and fetal viability as well as maternal well-being and even mortality. However, parameters such as maternal age, body weight and height, parity, the number of cigarettes smoked, smoking habits (e.g., inhalation) must be taken into account before meaningful conclusions can be made. Special refined statistical tools, such as multivariate analysis, are helpful in cutting through the maze of variables and makes it possible for the physician to warn pregnant women about the high risk of smoking during pregnancy.

OTHER DISEASES AND CONDITIONS

80–0389. Chernow, B.; Castell, D. O. **Diet and Heartburn.** *Journal of the American Medical Association* 241(21): 2307-2308, May 25, 1979.

This article discusses the role of various foods in the etiology of heartburn (gastroesophageal reflux), the potential for which is created by a decrease in the pressure of the lower esophageal sphincter (LES). Small changes in sphincter pressure can be observed after carbohydrate meals, a significant decrease can be observed after a meal high in fats, and an increase can be observed after a protein meal. Although caffeine has been shown to have only a minimal effect on LES pressure, coffee results in a significant increase in response. Alcohol, frequently incriminated as a heartburn producer, reduces LES pressure and decreases esophageal peristalsis. Other foods implicated in causing heartburn are fatty, spicy, and fried foods, some garden salad ingredients, and orange and tomato juice. Cigarette smoking may also contribute to esophageal reflux by decreasing LES pressure either through the action of nicotine or through the release of β-adrenergic agents.

80–0390. Christen, A. G.; Armstrong, W. R.; McDaniel, R. K. **Intraoral Leukoplakia, Abrasion, Periodontal Breakdown, and Tooth Loss in a Snuff Dipper.** *Journal of the American Dental Association* 98(4): 584-586, April 1979.

Gingival recession and advanced periodontal destruction were seen in a 36-year-old male who had a 13 year snuff-dipping habit. The mucolabial region had an extensive area of leukoplakia. One quid of snuff had been kept in place for 3 to 4 hours, swallowed, and replaced continually throughout the day. The histopathologic diagnosis was hyperkeratosis and severe chronic gingivitis. Snuff dipping and chewing seem to be increasing in the United States, particularly in the South. Dentists should be aware that these habits can lead to clinical leukoplakia, gingival recession, tooth abrasion, periodontal bone destruction, and possibly malignancy.

80–0391. Christianson, R. E. **Gross Differences Observed in the Placentas of Smokers and Nonsmokers.** *American Journal of Epidemiology* 110(2): 178-187, August 1979.

Gross placental characteristics of 7,651 white and black smokers and nonsmokers are compared in light of the previous finding that placentas of smokers are heavier for the weight of their fetuses than are those of nonsmokers. Subchorionic fibrin deposits and placental calcification are more prevalent in the placentas of smokers than in those of nonsmokers (whites: $p<0.0001$, $p<0.0001$; blacks: $p<0.01$, $p<0.0001$; respectively). Abnormal cord coloration, "maternal floor infarcts," and true infarcts are significantly higher for white smokers vs. nonsmokers ($p<0.025$, $p<0.05$, $p<0.05$), but are not significant (NS) for black smokers vs. nonsmokers. While mean placental weights and placental weight distributions for light and heavy smokers combined do not differ from those of nonsmokers, other placental measurements do differ. Smokers have thinner, rounder placentas than nonsmokers (whites: $p<0.001$, $p<0.01$; blacks: $p<0.01$, NS) and the distance from the edge of rupture of the membranes to the placental margin is reduced among smokers (whites, $p<0.025$; blacks, NS). These findings are discussed in relation to placental changes observed at high altitudes and complications of pregnancy that differ for smokers and nonsmokers. (Auth. Abs. Mod.)

80–0392. Eriksen, H. M.; Nordbo, H. **Extrinsic Discoloration of Teeth.** *Journal of Clinical Periodontology* 5(4): 229-236, November 1978.

The etiology and composition of extrinsic tooth discolorations are reviewed. The majority of tooth discolorations are extrinsic in nature and appear as brown integuments. Various clinical indices and photometric techniques have been used for the evaluation of extrinsic discolorations. Smoking, tea or coffee consumption, and increasing age are promoting factors, and such discolorations are frequently seen in connection with oral use of antibacterial plaque-inhibiting mouthrinses. In many studies retention of tar components on tooth surfaces in smokers is mentioned as a possible cause of tooth discoloration, but the presence of furfural and acetaldehyde in tobacco smoke indicates other mechanisms as well. Chemical alteration of the acquired pellicle appears to be the major reason for these brown integuments. (Auth. Abs. Mod.)

80–0393. Formicola, A. J.; Deasy, M. J.; Johnson, D. H.; Howe, E. E. **Tooth Staining Effects of an Alexidine Mouthwash.** *Journal of Periodontology* 50(4): 207-211, April 1979.

A double-blind clinical study was used to develop a staining index to use in assessing the degree of tooth staining by an alexidine mouthwash. The effects of a prior prophylaxis or the use of a fluoridated toothpaste on staining propensity were also determined. The study included 180 subjects aged 18 to 65 years, classified according to smoking and coffee and tea drinking habits. There were 98 nonsmokers, 34 light smokers (up to 1 pack/day), and 21 heavy smokers (1 or more pack/day). A stain area x intensity score was computed. Alexidine usage significantly increased the product score. Prior prophylaxis did not reduce the product score. Stain scores of nonsmokers were less than those of smokers. The interactions of coffee, tee, or smoking with alexidine were not significant.

80–0394. Khadim, M. I. **The Effects of Pan and Its Ingredients on Oral Mucosa.** *Journal of the Pakistan Medical Association* 27(6): 353-356, June 1977.

The effects of pan chewing on the oral mucosa of the chewer are presented. The betel leaf (pan) is usually chewed with one or more of the following: areca nut, lime, and the leaves of a special grade of tobacco. Carcinoma of the oral cavity and oropharynx is the most common malignancy found in Pakistan, accounting for about 20 percent of all malignancies recorded in the Jinnah Postgraduate Medical Center in Karachi. A high incidence of oropharyngeal cancer has also been reported for other parts of Asia. Because pan chewing is common in areas that have a high incidence of oropharyngeal cancer, it has been considered the major contributing factor. Clinical findings have shown that carcinoma of the buccal mucosa develops in the area in which a quid of chewing tobacco has been carried. Pan itself has a low risk for carcinoma, but the addition of tobacco, either in smoking or chewing form, is reported to increase the risk from 4 to 8-29 times. It has been suggested that lime salts eaten in the betel quids may act as carcinogens or cocarcinogens. Leukoplakia, an important precancerous condition, is frequently found in chronically irritated areas of the mouth. Significantly large numbers of persons who habitually chew pan plus tobacco, or betel nut alone, were reported to have oral lesions, including leukoplakia. Many of the chemical constituents of tobacco and its combustion products are irritating substances which can produce leukoplakic alterations of the oral mucosa. Leukoplakia was reported to be more prevalent among persons who used both forms of tobacco than among pan chewers or bidi smokers. Other oral lesions and their possible association with pan chewing plus tobacco were described.

80–0395. Medical Letter on Drugs and Therapeutics. **Toxic Reactions to Plant Products Sold in Health Food Stores.** *Medical Letter on Drugs and Therapeutics* 21(7): 29-32, April 6, 1979.

Among herbal teas commonly found in health food stores are some that have harmful effects. Some teas are cathartic, anticholinergic, psychotogenic, allergenic, and abortifacient. One contains poison. Indian tobacco, which is smoked or used in tea, contains lobeline, a central nervous system-stimulating alkaloid similar to nicotine. Its ingestion in large doses can cause sweating, vomiting, paralysis, depressed temperature, coma, and death. Two other teas with diuretic action should also be avoided because of their toxic effects. Shave grass or horsetail plants contain nicotine and thiaminase. In horses and other grazing animals, ingestion of these plants has caused excitement, loss of appetite and muscular control, diarrhea, labored respiration, convulsions, coma, and death. Thiamine deficiency with classical beri-beri symptoms has been reported in sheep after experimental administration of shave grass.

80–0396. Mohlin, B.; Ingervall, B.; Hedegard, B.; Thilander, B. **Tooth Loss, Prosthetics and Dental Treatment Habits in a Group of Swedish Men.** *Community Dentistry and Oral Epidemiology* 7(2): 101-106, April 1979.

Dental history, dental status, dental treatment habits, and prosthetic treatment needs were studied in 389 Swedish men aged 21-54 years. Among the factors considered in relation to dental status were place of birth, number of brothers and sisters, level of education, and smoking habit. The subjects included 60.7 percent nonsmokers and light smokers (0-4 cigarettes/day), 17.0 percent moderate smokers (5-14 cigarettes/day or pipe smokers), and 22.4 percent heavy smokers ($>$ 15 cigarettes/day). Age, place of birth, educational level, and smoking habit were among the factors which seemed to be related to dental status. A correlation was found between smoking and edentulousness, and between use of partial dentures and smoking (both $p<0.01$).

80–0397. Poellmann, L.; Dietrich, A. **Beobachtungen zur akuten nekrotisierend-ulzerosen Gingivitis bei jungen Mannern. [Observations on Acute Necrotizing Ulcerative Gingivitis in Young Men.]** *Deutsche Zahnaerztliche Zeitschrift* 34(2): 222-224, February 1979, German.

Acute necrotizing ulcerative gingivitis, fever, and swelling of the regional lymph nodes were examined in 72 young men aged 18 to 24. All of the individuals were smokers; approximately three-fourths of the group smoked more than 60 cigarettes per day. Roughly half of the individuals examined had extremely poor oral hygiene or underlying psychological or physical stress at the onset of the disease. Dentitio difficilis and alcohol abuse appeared to be less important factors. The use of a mouthwash and improved oral hygiene, proved to be effective therapy. Diseases and disorders of internal structures of the body could not be diagnosed in any of these individuals. (Auth. Abs. Mod.)

80–0398. Wagner, I. U. **Nikotinvergiftung im Kindesalter. [Nicotine Poisoning in Children.]** *Paediatrie und Paedologie* 14(2): 191-195, 1979, German.

Thirty-five cases of nicotine poisoning in children 7 to 24 months old were observed at the Childrens' Clinic of the University of Vienna from January 1961 to June 1978. In most cases, the children were brought to the clinic within 30 minutes to 1 hour after eating cigarettes or cigarette butts. Most of the patients ingested from one-fourth to one cigarette, three patients one to two cigarettes. The most common symptoms were vomiting and tachycardia, which in one case lasted over 14 hours. No symptoms were observed in 18 patients. Treatment consisted of gastric lavage and administration of activated charcoal. Relatively few clinical symptoms were observed because nicotine resorption in the stomach is slow.

See also, **80–0334**

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–0399. Abelson, H. I.; Fishburne, P. M.; Cisin, I. **National Survey on Drug Abuse: 1977. A Nationwide Study—Youth, Young Adults, and Older Adults. Volume I: Main Findings.** Princeton, New Jersey, Response Analysis Corporation, 1977, 235 pp.

This survey reports findings on the use of alcohol, inhalants and hallucinogens, marihuana, opiates, sedatives, stimulants, tobacco, tranquilizers (nonmedical use), and legal drugs. Attitudes and beliefs of youth and adult users concerning the addictiveness and the legal and commercial futures of these substances are described. The public was more likely to have had experience with marihuana than with any of the other psychoactive drugs studied. More than one-fourth (28.2 percent) of the youth (age 12-17) reported marihuana experience and about one-sixth (16.1 percent) reported current use. For adults (age 18 and over), the lifetime prevalence rate (24.5 percent) was somewhat lower than that of youth, and current use (8.2 percent) was half of that reported by 12-17 year olds. Marihuana experience is strongly related to age; the highest prevalence rate was found among young adults aged 18-25. Of this age group, 6 in 10 have used marihuana compared to fewer than 2 in 10 older adults (age 26 and over). Young adults also had the highest rate for current use: 1 in 4 was a current user compared to only 1 in 30 older adults. From 1976 to 1977, there was a significant increase in prevalence rates among both youth and young adults but not among older adults. The experience level (tried at least once) among youth rose 5.7 percent, and among young adults increased by 7.2 percent. During the same time period, there was a significant increase (3.7 percent) in current use among youth. No change in current use was indicated for young adults or older adults. Use of a drug "stronger" than marihuana (and/or hashish) was reported by about 1 in 4 young adults, 1 in 10 youth, and 1 in 25 older adults. The use of marihuana (and/or hashish) only was reported by 1 in 5 youth, 1 in 3 young adults, and just over 1 in 10 older adults. No illicit use of any drug was reported by more than 8 in 10 older adults, 7 in 10 youth, and only 4 in 10 young adults. About half of the youth and two-thirds of the adults have used cigarettes. Among youth, about 2 in 10 were current smokers compared to 4 in 10 adults. Reported levels of current smoking for both youth and adults have remained virtually unchanged since the 1976 survey.

80–0400. Adams, P. I. **Changes in Personal Smoking Habits Brought About by Changes in Cigarette Smoke Yield.** In: *Proceedings of the Sixth International Tobacco Scientific Congress*, Tokyo, November 14-20, 1976, The Japan Tobacco and Salt Public Corporation, 1977, pp. 102-108.

Regular twice weekly measurements of puff volumes, durations, frequencies, and numbers of puffs per cigarette were made during a 6-month smoking experiment. The study included 124 smokers who were grouped into 4 panels and who smoked 8 different types of flue-cured cigarettes. The types of cigarette had several levels of nicotine (0.7 to 1.6 mg) and total particulate matter (10 to 19 mg) in the smoke and one included tip ventilation. The smoker also completed questionnaires designed to reveal subjective opinions about cigarette quality. The objective of the experiment was to study whether changes in smoking habit occurred in response to, and to compensate for, changes in smoke yield. Although the smokers were not told when their types of cigarette were changed, the smoking data revealed small differences. It seems certain that smoking parameters do change when the cigarette yield is changed, but the pattern of change is not what would be expected from a simple theory of compensation by increase or decrease of puff volume and number of puffs. No significant difference in consumption between types of cigarettes was found despite the fact that the smokers were supplied with free cigarettes throughout the experiment. However, the usually observed difference in cigarette consumption of male and female smokers was shown in only one panel. The data from the smoking of the ventilated cigarettes was the most conclusive: it gave clear evidence of compensation for about half of the magnitude of the reduction in smoke yield, and some unfavorable subjective comment. (Auth. Abs.)

80–0401. Adams, W. G. **Survey of Tobacco and Alcohol Use Among Undergraduates.** *Medical Journal of Australia* 2(3): 160, August 11, 1979.

A survey of 643 undergraduates (425 males and 218 females) at the University of New South Wales revealed that 28 percent of males and 23 percent of females currently smoked. Smoking rate was not related to students' sex, age, year of study, or domestic circumstances, but did vary significantly across faculties, ranging from 12.8 percent of students in applied science to 38 percent of students in law. In medicine, 20.6 percent of the students smoked. Among current nonsmokers, there was a significantly higher proportion of females who had never smoked ($p<0.01$). Smoking rate among undergraduates appeared lower than among students' age-peers in the Australian population. Concerning alcohol consumption, 92 percent of males and 96 percent of females currently drank. Drinking rate was not related to students' sex, age, year of study, domestic circumstances, or faculty, though frequency of drinking and amount of alcohol consumed per drinking occasion were higher among males. There was also a significant relationship between smoking and drinking habits. The drinking rate was significantly higher among smokers than among nonsmokers ($p<0.01$), and smokers also drank more frequently than nonsmokers ($p<0.01$). There was, however, no significant relationship between smoking and the amount of alcohol consumed per drinking occasion. (Auth. Abs. Mod.)

80-0402. Bewley, B. R.; Johnson, M.R.D.; Banks, M. H. **Teachers' Smoking.** *Journal of Epidemiology and Community Health* 33(3): 219-222, September 1979.

The influence of teachers' smoking on the smoking habits of school children was examined in a prospective study of 6,000 school children and 1,300 teachers in England over the 1974-1975 period. Comparisons were made between the levels of smoking among staff and students. While the prevalence of smoking increased between 1974 and 1975 among both boys and girls (from 6 to 9 percent and 3 to 6 percent, respectively), male and female teachers' cigarette smoking rates remained constant (approximately 21 percent for each). However, while most female teachers smoked only cigarettes, 20 percent of the male teachers smoked cigars or pipes or a combination of these and cigarettes. Fewer teachers smoked in grammar schools than in middle, secondary, or comprehensive schools. An examination of the proportion of teachers and children smoking in each school in 1974 showed a significant relationship between boys' and male teachers' smoking ($p<0.02$). In 1975, smoking by boys was again more common in schools where a high prevalence of smoking among male teachers had been found in 1974. In addition, the ratio of smoking prevalence among girls in 1975 to that in 1974 was also significantly linked to the 1974 prevalence of smoking among male teachers ($p<0.05$).

80-0403. Biener, K. **Stress bei Journalisten. [Stress in Journalists.]** *Munchener Medizinische Wochenschrift* 121(13): 449-452, 1979, German.

The incidence of stress in 227 Swiss journalists (189 men and 38 women) was evaluated by means of a questionnaire. The average age of the men was 43 years and that of the women, 39 years. The subjects were divided into 2 groups, 43 years and younger (111 men and 19 women) and 44 years and older (78 men and 19 women). About 90 percent of the subjects questioned admitted suffering from stress at that time. Significantly more of the subjects under stress were smokers than those without stress (39 vs. 22 percent, respectively). Of the subjects studied, 46 percent of those under 43 years and 51 percent of the older subjects were smokers. There were three ex-smokers in each group. Stress was not a clear health risk factor in this study.

80-0404. Clark, R. R. **Reactions to Other People's Cigarette Smoking.** *International Journal of the Addictions* 13(8): 1237-1244, 1978.

Cigarette smoking in social interaction is examined through a study of reactions and perceptions people have to others' smoking in their social presence. A questionnaire was administered to 130 subjects, among whom 57 were smokers (at least one cigarette daily), 30 were ex-smokers (former regular smokers), and 43 were nonsmokers (never smoked). Ex-smokers and nonsmokers in particular tended to report a disrupting effect upon their interactions with others when others smoked (17 and 14 percent, respectively). Smokers tended to report unaffected interaction (74 percent) and the desire to smoke (51 percent). Almost nobody (<5 percent) reported a positive effect on interactions by others' smoking. This finding of smoking as interruptive is discussed in the context of previous research findings that smokers report an increased social comfort with their own smoking. (Auth. Abs. Mod.)

80-0405. Cooreman, J.; Burghard, G.; Perdrizet, S. **L'Adolescent et le Tabagisme. Responsabilite des Parents et des Professeurs. Resultats d'une Campagne de Sensibilisation. [Teenagers and Smoking. Responsibility of Parents and Teachers. Results of a Sensitizing Campaign.]** *Journal de Medecine de Strasbourg* 9(9): 483-488, October 1978, French.

A questionnaire concerning smoking habits was administered to about 29,000 school children, aged 13 to 19 years, in the public and private schools of the Bas-Rhin region in France. The purpose of the 1976 survey was to analyze the smoking habits of the adolescents in relation to those of their parents and to the school environment. Analysis indicated that 30.3 percent of all respondents admitted smoking an average of 8.2 cigarettes/day since they were 14.6 years old. The difference between boys and girls in the percentage of smokers (33.4 vs. 27.7), daily cigarette consumption (9.3 vs. 7.1), and the percentage of smoke inhalers (83.2 vs. 75.2) was statistically significant ($p<0.01$). The percentage of adolescent smokers with nonsmoking vs. smoking parents was 23.6 vs. 45.4. The corresponding daily cigarette consumption for the adolescents was 8.1 vs. 9.1 cigarettes/day. The percentage of smokers with mother only smoking was higher than among those with father only smoking (41.1 vs. 33.1). Adolescent smoking began before 15 years in 44 percent of the boys and in 37 percent of the girls with nonsmoking parents. With at least one parent who smoked, 50 percent of the boys and 41 percent of the girls were smokers before they were 15 years old. There were 39.2 percent smokers in boys' schools, 30.8 percent smokers in girls' schools, and 28.6 percent in coeducational schools. Among boarding vs. day school students 39 vs. 28.4 percent were smokers. The effects of a sensitizing campaign and/or smoking prohibition were studied in 15,756 students. The sensitization program involved antismoking health education, filmstrips, discussions and debates. Among students subjected to sensitization and prohibition there were 23.2 percent smokers compared with 38.0 percent smokers among those subjected to sensitization only. The percentage of adolescent smokers decreased from 30.6 to 27.6 when the teachers were sensitized, and from 29.9 to 25.7 when the teachers were forbidden to smoke in classes.

80–0406. Doubrawa, R. **Erkundungsstudie zu Rauchgewohnheiten, Personlichkeitsmerkmalen, Raucher- und Nichtraucherimage bei jungen Frauen mit psychosomatischen Storungen. [Exploratory Enquiry Into Smoking Habits, Personality Traits, Smoker and Non-Smoker Image in Young Women With Psychosomatic Disorders.]** *Oeffentliche Gesundheitswesen* 40(12): 820-827, 1978, German.

A disproportionately large number of habitual smokers, particularly stress smokers, can be found among young women with psychomatic disorders. In an exploratory enquiry, 111 women staying at a health resort were asked questions regarding smoking habit. and personality traits. Smoker and nonsmoker images were compared in nonsmokers, ex-smokers, stress smokers, and nonstress smokers. The female smokers, especially stress smokers, showed more anomalies according to the Freiburg Personality Inventory than the nonsmokers (among other characteristics, a greater degree of nervousness and a larger amount of neurotic tendencies). Also seen were indications that stress smokers felt less responsible for having acquired and upheld the smoking habit. In both groups of smokers, social factors had a prominent habit forming function. There were hardly any group-specific differences between the smoker and nonsmoker stereotypes. However, the nonsmoker image usually showed more positive traits. (Auth. Abs.)

80–0407. Eiser, J. R. **Discrepancy, Dissonance, and the "Dissonant" Smoker.** *International Journal of the Addictions* 13(8): 1295-1305, 1978.

This paper reexamines, in the light of social psychological research, the theoretical assumptions made in a classic study of smoking habits and attitudes in Britain. One assumption made is that antismoking messages which are very discrepant from the opinions held by smokers should be avoided since they may produce attitude change in the opposite direction to that intended. It is argued that there is little empirical basis for this assumption, and that the discrepancy of a communication from the initial attitudes of a target audience is not necessarily the most important determinant of its effectiveness. Also distinguished are "consonant" and "dissonant" smokers, the latter being those who say that they would like to give up smoking if they could do so easily. It is argued that the term "dissonant" is a misnomer when applied to smokers who see themselves as unable to choose not to smoke. Practical and theoretical implications are discussed. (Auth. Abs.)

80–0408. Evans, R. I.; Rozelle, R. M.; Mittelmark, M. B.; Hansen, W. B.; Bane, A. L.; Havis, J. **Deterring the Onset of Smoking in Children: Knowledge of Immediate Physiological Effects and Coping With Peer Pressure, Media Pressure, and Parent Modeling.** *Journal of Applied Social Psychology* 8(2): 126-135, 1978.

A sample of 750 male and female students entering seventh grade was drawn from a population of physical education classes in 10 junior high schools in Houston, Texas. Subjects were pretested to determine base rates of smoking. Overall, 31 percent of the subjects reported smoking at least one cigarette within the previous month. There were no significant differences among the 10 schools in the amount of base rate smoking. Most children believed that smoking endangered their long-term health. Nevertheless, three sources of social pressure were often found to have an overriding influence to begin smoking: peers, models of smoking parents, and media. Interventions were developed to train children to cope with these pressures. Also stressed were immediate rather than long-term physiological effects of smoking. A dependent measure was utilized that generated increased validity of self-reported smoking. Treatment consisted of four 10-min videotapes on smoking and health, peer pressure, and parental influence. Based on reported smoking behavior, the treatment group ($n=80$), the feedback only group ($n=93$), and the testing (monitoring) only group ($n=97$) displayed smoking onset percentages of 10.0, 8.6, and 9.7, respectively, at the time of the final posttest. The onset rate of the control group ($n=104$) was 18.3 percent. Because of the low expected frequency of onset of smoking in the experimental groups, the experimental groups were combined for analysis purposes. A comparison between combined experimental groups and the control group yielded a significant chi-square value of 4.51 ($p<0.05$). (Auth. Abs. Mod.)

80–0409. Guillerm, R.; Radziszewski, E. **Une Nouvelle Methode d'Analyse de l'Acte du Fumeur. [A New Method of Analyzing the Act of Smoking.]** *Annales du Tabac* 13(1): 101-110, 1975, French.

The new method for analyzing the act of smoking which is presented comprises a combination of three methods of investigation: measurement of the frequency, volume, and duration of puffs using two complementary techniques (metric-flow and pyrometric); simultaneous analysis of the ventilatory behavior of the smoker; and an evaluation of the amount of smoke inhaled (carbon monoxide, carboxyhemoglobin). The methodology was adapted to study the behavior of smokers when a low-irritant cigarette was substituted for their usual cigarette. Owing to the changes in the way of smoking which are observed in smokers, this method for analyzing the act of smoking is useful in evaluating the potential biological effects of a new cigarette. (Auth. Abs. Mod.)

80–0410. Health and Welfare Canada. **Smoking Habits of Canadians 1975.** Department of National Health and Welfare, Technical Report Series No. 7, Canada, December 1977, 39 pp.

This report presents information on the smoking habits of Canadians during the years 1974 and 1975. It shows that the percentage of regular smokers in the Canadian population continues to decline. In 1975, 38.7 percent of the population 20 years of age and over were regular cigarette smokers, compared to 39.6 percent in 1974 and 42.2 percent in 1970. The decline in the percentage of Canadian smokers has been largely due to substantial decreases in regular smoking by adult males during the past 10 years. Between 1974 and 1975 there was a further 1.9 percent decrease. In contrast, regular smoking among adult females remained relatively stable during the past decade, and in 1975 the percentage of female smokers remained unchanged at 32 percent. The percentage of regular smokers in the teenage population dropped 1.6 percent between 1974 and 1975 to 28.5 percent. This change was largely due to a decline in the percentage of male teenage smokers. An examination of the relationship between level of education and use of cigarettes indicated that the highest percentage of regular male smokers was found among those with a grade 8 or less education whereas the highest percentage of regular smokers among Canadian females was found among those with at least some secondary education. Male regular smokers appeared to be smoking fewer cigarettes between 1974 and 1975, particularly in the age group 20 to 44 years, while there were increases in the percentage of female smokers aged 20 to 24 who smoked more than 25 cigarettes daily. In 1975, Quebec continued to have the highest percentage of male smokers 15 years of age and over (50.4 percent), followed by the Atlantic Region (44.5 percent), Ontario (40.6 percent), the Prairie Region (39.5 percent) and British Columbia (39.2 percent). The highest percentage of regular smokers in the female population was found in Quebec (34.6 percent); the lowest occurred in the Prairie Region (29.9 percent). Persons living in rural areas and large urban centers (population 100,000) seemed less likely to be smokers than persons living in small towns and cities. According to the 1975 survey, 11.7 percent of the Canadian population 15 years of age and over indicated that they had stopped smoking. The majority of these successful quitters stopped smoking because of health reasons and used no aid to assist them in quitting.

80–0411. Hirvonen, L.; Kansakoski, I. **Psykiatrisen Sairaalan Potilaiden ja Henkilokunnan Tupakointi. [The Smoking Habits of the Patients and the Staff of a Psychiatric Hospital.]** *Suomen Laakarilehti* 34(18): 1473-1476, June 20, 1979, Finnish.

The large number of smokers among chronic psychiatric patients represents a serious hygienic problem which can be largely influenced by members of the hospital staff. The smoking habits of 338 patients and 142 staff members were studied by means of a questionnaire, interviews, and observations. Of the 180 male patients, 99 were 40-59 years of age and 47 were 60 or older. The corresponding figures for women were 73 and 60. All staff members were below 60 years of age, and 18 men and 57 women were below 40. Of the male patients, 63.3 percent were smokers. No statistically significant difference was found in smoking prevalence between younger and older male patients. While 37 percent of the women patients below 60 were smokers, only 12 percent of those above 60 were. No correlation was found between age and smoking among staff members. More psychotic male patients smoked (68 percent) than psychotic female patients (30 percent), but neither group had a significantly larger number of smokers than other patients of the same sex. Out of 23 staff women, 21 smoked only cigarettes. Of the patients, 37 percent smoked more than 20 cigarettes or 20 gm pipe tobacco per day. There was no statistical difference between sexes in this regard. Half of the patients thought that tobacco would not have any bad effect on their health, and a third of the patients thought that the effect was positive.

80–0412. Hull, C. L. **The Influence of Tobacco Smoking on Mental and Motor Efficiency. An Experimental Investigation.** Westport, Connecticut, Greenwood Press, 1975, 165 pp.

This book describes a study, originally published in 1924, of the effects of smoking on neuromuscular, sensory-motor, and mental functions. The study was carried out in 18 young men (9 nonsmokers and 9 smokers) over a period of 18 days and involved smoking a pipe of mild tobacco (without inhaling the smoke) for a 25-min period prior to undergoing a series of tests. The following results were observed among the regular smokers: (1) a marked increase in heart rate and hand tremor; (2) no measurable effect on the rate of tapping or on the rate or accuracy of eye-hand reaction; (3) a slight increase in resistance to muscular fatigue and in speed of reading reaction time; (4) a marked gain in the rate of complex mental addition, but no measurable effect on the accuracy of addition; and (5) a probable loss in auditory memory and in efficiency of rote learning immediately after smoking. It was noted that only three functions (heart rate, hand tremor, and rate of addition) showed an unmistakable influence of smoking.

80–0413. Ingram, R.; Goldstein, B. **Role of Expectancy Factors in Behavioral Self-Control Therapies: An Experimental Inquiry.** *Psychological Reports* 42: 535-542, April 1978.

Most research in the area of behavioral self-control has focused either on its efficacy or its more obvious components, e.g., self-monitoring and self-reward. Expectancy factors have been relatively ignored. Utilizing a smoking reduction paradigm, the present study investigated the possibility that positive expectancy may be related to success in behavioral self-control therapies. Two groups of volunteer students who wished to reduce their smoking behavior were taught Homme's coverant self-control procedure. One group was told that the technique was 80 percent effective, while the other was informed that it was 15 percent effective. Of the seven members of the group with high expectancy, five had attempted to quit smoking prior to participation in the present study. The mean number of prior attempts was 1.5. All seven subjects in the group with low expectancy reported at least one attempt to quit (mean number of 2.2). Four of the five control subjects had made a previous attempt, their average number being 1.6. The group with low expectancy saw themselves as being slightly heavier smokers than did either the group with high expectancy or the controls. The means for the three groups, on a scale of 7, were 4.2, 3.5, and 3.2, respectively. Subjects reported smoking an average of 17.1 cigarettes per day. The group averages were, for high expectancy 17.4, for low expectancy 17.1, and for the control group 16.6. Subjects' average baseline smoking rate turned out to be somewhat below their self-reported rate. The overall group mean was 13.4, with the high expectancy, low expectancy, and control groups showing averages of 10.5, 15.4, and 14.5 cigarettes smoked per day, respectively. Although analysis of variance indicated no significant differences between groups, a Newman-Keuls analysis indicated that, for the group with high expectancy, reduction either reached or approached significance over the control group's level during 3 of the 4 weeks of treatment. Data suggested that, although its exact role requires further research, expectation is an important variable in behavioral self-control. (Auth. Abs. Mod.)

80-0414. Johnston, L. D.; Bachman, J. G.; O'Malley, P. M. **Drugs and the Class of '78: Behaviors, Attitudes, and Recent National Trends.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institute on Drug Abuse, DHEW Publication No. (ADM)79-877, 1979, 355 pp.

This publication reports trends in prevalence of drug use in high school seniors in 1978. Differences by sex, geographical region, population subgroup, and population density are tabulated. Between 6 and 7 in every 10 seniors (64.1 percent) reported illicit drug use at some time in their lives. However, a substantial proportion used only marihuana (27.6 percent of the sample or 43 percent of all illicit users). Overall marihuana use was somewhat higher among males, and daily use of marihuana was substantially higher among males (14.2 percent vs. 7.1 percent for females in 1978). Both the college-bound and the noncollege-bound seniors have been showing parallel trends in overall illicit drug use over the last 2 years. Both showed a rising proportion using marihuana only, and a steady (or only slightly increasing) proportion using illicit drugs other than marihuana. Annual marihuana use was reported by 47 percent of the college-bound and 52 percent of the noncollege-bound. Marihuana use was reported by 57 percent in the large cities but by only 43 percent in the nonmetropolitan areas. Use of either of the two major licit drugs, alcohol and cigarettes, was still more widespread than use of any of the illicit drugs. Nearly all students had tried alcohol (93 percent) and the great majority (72 percent) had used it in the past month. Some 75 percent reported having tried cigarettes at some time, and 37 percent had smoked at least some in the past month. Cigarettes were used daily by more of the respondents (28 percent) than any of the other drug classes. Of these respondents, 18.8 percent said they smoked a half-a-pack or more per day. There was practically no sex difference in the prevalence of smoking a half-a-pack or more daily (18.9 percent for males vs. 18.0 percent for females), although among regular smokers males appeared to consume a higher quantity of cigarettes. The greatest difference between the college plans groups involved daily smoking. Only 11 percent of the college-bound smoked a half-a-pack or more daily, compared with 26 percent of the noncollege-bound. The largest regional differences occurred for regular cigarette smokers. In the Northeast, 24 percent said they smoked half-a-pack or more per day compared with 20 percent in the North Central, 17 percent in the South, and only 12 percent in the West. While the proportion smoking half-a-pack of cigarettes or more per day remained constant at 20 percent for males from 1975 to 1977, between 1975 and 1977 the rate of cigarette smoking for females increased from 16 percent to 19 percent, virtually eliminating the previous sex difference. Over the past year, however, regular smoking declined slightly in parallel for both sexes.

80-0415. Jones, J. W.; Bogat, G. A. **Air Pollution and Human Aggression.** *Psychological Reports* 43(3, Part 1): 721-722, December 1978.

The effects of secondary cigarette smoke on human aggression was studied in 24 male and 24 female nonsmokers, ranging in age from 18 to 25 years. Subjects were first either angered or not angered and then were given a chance to vent their aggression. While doing so, one-half of the subjects were exposed to secondary cigarette smoke and the other half to clean ambient air. Results showed that, as predicted, subjects exposed to secondary cigarette smoke were more aggressive than were their clean air counterparts ($p<0.04$). Exposure to secondary cigarette smoke also increased aggression in angered subjects compared to their counterparts exposed to clean air.

80-0416. Klaukka, T.; Kalimo, E.; Nyman, K.; Byckling, T. **Savukkeiden Poltto Sosiaaliturvan Tutkimuslaitoksen Haastattelututkimusten Mukaan. [Smoking Prevalence According to the Interview Studies of the Research Institute of the Social Security System.]** *Sosiaalilaaketieteellinen Aikakauslehti* (3-4): 215, 1977, Finnish.

Two interview studies including questions regarding smoking prevalence were performed in 1968 and in 1979 by the Research Institute of the Social Security System in Finland. Of the entire population (over 15 years old) 27 percent were smokers: 38 percent of the men and 17 percent of the women.

Smoking was most common in the 25-34 age group, in which 43 percent of the men, 28 percent of the women, and 35 percent of the entire group were smokers. Smoking was more common in the cities (30 percent) than in the rural areas (24 percent). Urban men aged 25-34 smoked most frequently. Of male vs. female smokers, 55 vs. 25 percent smoked more than 15 cigarettes per day. In the capital area smoking was more common than elsewhere in the country. Of the 1976 population over 15-years-old, 32 percent were smokers: 29 percent of the women and 36 percent of the men (less than the national average for men) smoked. The corresponding figure in 1968 was 47 percent. This reduction in smoking applied to men of all age groups. Women aged 25-34 in the capital area showed an increase in smoking prevalence from 34 percent in 1968 to 37 percent in 1976. The percentage of women smokers under 25 years of age declined from 40 in 1968 to 36 in 1976.

80-0417. Knight, B. J.; Osborn, S. G.; West, D. J. **Early Marriage and Criminal Tendency in Males.** *British Journal of Criminology* 17(4): 348-360, October 1977.

The association of early marriage and criminal tendencies was examined in delinquent males and in a group of nondelinquent controls as part of the long-term Cambridge Study in Delinquent Development. Subjects were drawn from a London working class school population, and were followed from the age of 8 to 22 or 23. Their delinquent and marital status was compared on their 21st birthday. It was found that males who married young did not come predominantly from the delinquent sector of the population, but that an early start to heterosexual experience was associated with both delinquency and early marriage. Men who married before the age of 21 were found to be more aggressive than men who did not. Interviews of 21-year-old men showed that the social habits characteristic of delinquents at age 18 became less prominent in the whole sample. Men who married early were more often heavy smokers. At age 18, 80.2 percent of the delinquents, and 59.4 percent of the nondelinquents said that they were regular smokers; at age 21, 78.8 percent of the delinquents reported that they were regular smokers, compared with 58.1 percent of the nondelinquents. More of the married men were regular smokers. Getting married did not appear to reduce delinquency, but it did reduce some of the social habits associated with delinquency, particularly drinking, sexual promiscuity, and illegal use of drugs. However, heavy smoking, aggressive behavior, hostility to police, and liability to unemployment, were not reduced.

80-0418. Lakartidningen. **Rokvaneundersokning i Storstockholm. [A Smoking Prevalence Study of the Stockholm Capital Area.]** *Lakartidningen* 76(26-27): 2500, 1979, Swedish.

The results of a 1976-1978 study of smoking habits in the Stockholm area are presented. During that period the proportion of smokers among men decreased from 59 to 49 percent and increased among women from 51 to 54 percent. While women in Stockholm smoked more than the national average for women, the increase was proportional to the national average for this period. The percentage of daily smokers among men decreased from 48 percent in 1976 to 38 percent in 1978, corresponding to the national trend. The percentage of daily smokers among women was stable: 42 percent in Stockholm and 34 percent in the entire country during this time period. Among males in Stockholm the determination to give up smoking had increased. In 1976, 30 percent wanted to stop smoking, but did not think they would be able to, while 19 percent were more optimistic. Of those who wanted to stop smoking in 1977, 31 percent thought they would be able to, while 24 percent were doubtful. Among women, however, the percentage of pessimists was 35 in 1977 as compared to 25 in 1976, while the percentage of optimists had gone down from 25 to 20 percent.

80-0419. Marklund, U. **Smoking in Compulsory Schools. Interviews and Improvisations.** Institute of Education, University of Goteborg, Sweden, May 1979, 56 pp.

A total of 80 Swedish children in the 3rd, 5th, and 7th grades were interviewed, individually or in groups, about their smoking habits and their views on smoking to determine why children smoke and how they may be influenced not to smoke. Of the children who smoked, most did not begin until 5th or 6th grade. Until then they had a strong negative attitude to smoking, based mainly on their experiences with passive smoking. However, by 6th grade, 40 percent of the students had tried smoking, and 10 percent smoked regularly. Social and peer pressure seemed to be primarily responsible for children beginning to smoke at this age. At the 7th grade level, smoking took on a more adult pattern and became no longer important in itself, but was continued because there was nothing more interesting to do. Since the study showed that children rarely talk about smoking among themselves, it is important to discuss this problem openly with them to increase their awareness as to why people smoke. To achieve this awareness and better understanding of smoking behavior, role playing was utilized in children from 4th to 8th grade and is recommended as a valuable educational tool.

80-0420. Moss, A. J. **Changes in Cigarette Smoking and Current Smoking Practices Among Adults: United States, 1978.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics. *Advance Data from Vital and Health Statistics* , Publication No. 52, September 19, 1979, 15 pp.

This report presents the latest data from the Health Interview Survey (HIS) on smoking status and behavior of the adult U.S. population for the 6-month period July 17, 1978, through January 14, 1979. The data show that the proportion of cigarette smokers in the population has declined from 36.9 percent in 1970 to 33.3 percent in 1978. This decline occurred primarily among males (from 43.5 percent in 1970 to 37.4 percent in 1978); the proportion of female smokers remained about the same (31.1 percent in 1970 and 30.4 percent in

1978). While the prevalence of smoking is declining, the average number of cigarettes consumed daily per smoker is increasing. In 1970, 23.3 percent of adult smokers smoked 25 cigarettes or more a day, compared to 27.9 percent in 1978. In addition, 32.9 percent of all adults smoked less than 15 cigarettes a day in 1970 compared to 29.4 percent in 1978. Persons smoking fewer than 15 cigarettes per day were more often female, black, and either under 25 years of age or 65 years and over. The 1978 HIS data also show that approximately 60 percent of current smokers have tried at least once to stop smoking, and 60.5 percent of these stopped for 1 week or more during their last attempt. Proportionately more young smokers (42 percent) attempted to quit smoking than others and had appreciably greater success. Middle-aged smokers (45-64 years of age) were least likely to try to quit and also least likely to succeed. Over one-fourth of adults who currently smoke use lower tar cigarettes (less than 15 mg of tar), with a higher proportion of women doing so than men (35 vs. 23 percent).

80-0421. Murphy, H.B.M. **Historic Changes in the Sex Ratios for Different Disorders.** *Social Science and Medicine* 12(3B): 143-149, July 1978.

Shifts in the sex ratios for peptic ulcer, diabetes, lung cancer, and coronary heart disease are examined. Most have no simple, agreed, and solidly founded explanation. However, the explanations which best fit each shift are often the ideal self-image of a sex which appears to have led to either an excess vulnerability or a failure to make use of available remedies. It is suggested that such ideal or stereotyped self-images require more attention than occupied roles when attempting to assess the influence of social factors on illness. For instance, sex differences in lung cancer mortality are generally attributed to differences in tobacco consumption. However, when one views the subject historically and uses 1910 to 1970 British data on tobacco consumption and lung cancer rates, there are discrepancies which do not seem explainable in terms of the time lag between peak smoking period and period of greatest risk. While the male cancer rate appears to have mounted more rapidly than the increase in tobacco consumption, the slope of the curve for the female cancer rate is less steep than that for female tobacco consumption, and no correction for time lag makes them fit any better. The two most obvious explanations for such a difference would be that a certain threshold of individual consumption needs to be passed before cancer mutation is provoked, or that women inhale less than men. The second of these fits the existing facts better. In Britain, women tend to light a cigarette particularly when under tension, whereas men light up mainly when they are understimulated or bored. Moreover, in women the quantity of cigarettes smoked correlates with the score on a neuroticism scale whereas that is not true in men. However, American men do show correlation between quantity of cigarettes smoked and neuroticism. Apparently, the sex difference in vulnerability to lung cancer may have something to do with the functions which tobacco serves for each sex, and that might in turn be related to some biological difference, since we know that nicotine can have both a stimulant and a sedative effect, depending on circumstances and dosage. (Auth. Abs. Mod.)

80-0422. Ockene, J. K. **A Study of the Psycho-Social Factors Involved in Changing Smoking Behavior: Risk Factor Alteration in a Coronary Heart Disease Prevention Program.** Doctoral Dissertation, Boston College, Department of Education, Boston, University Microfilms 79-22078, 1979, 286 pp.

The sample in this study is comprised of 169 male smokers aged 35-57 who were identified as individuals with a high risk of developing coronary heart disease, but who were free of signs or symptoms of this disease upon entry into the study. All of the participants were enrolled in the Multiple Risk Factor Intervention Trial, a program funded by the United States Department of Health, Education, and Welfare. Participants were placed in groups composed of 10 to 12 men. Approximately 50 percent in each group were smokers. Each group met with a nutritionist, physician, and psychologist 90 min/week for 10 weeks. Health information and possible coronary heart disease risk reduction were discussed. The participants were categorized as Continuing Successes, Recidivists, or Nonstoppers. Continuing Successes were defined as individuals who were smoking at least five cigarettes a day at baseline, stopped smoking at some point during the 4-month intensive intervention phase, and reported maintenance of their zero cigarette level at regular 4-month monitoring points for up to 2 years from program entry. Recidivists were defined as individuals who were smoking at least five cigarettes a day at baseline, stopped smoking for at least 24 hours at some point during the 4-month intensive intervention phase, and reported being a smoker at a 4-month monitoring point. Nonstoppers were subjects who smoked at least five cigarettes a day at baseline, and never stopped smoking for at least 24 hours at any point during the 4-month intensive intervention phase. Continuing Successes, when compared to Recidivists and Nonstoppers, were measured as experiencing the least amount of stress during the 2 years of the study, having the most personal security, experiencing the most success with cessation attempts prior to the program, attending the most group sessions, stopping smoking at an early session, having their significant others attending the same number of group sessions as those of the Recidivists, having the most positive smoking environment, experiencing the least amount of negative effect, smoking the lowest number of cigarettes when smoking heaviest and at entry into the study, and having the greatest degree of change between the aforementioned times. Contrary to these findings, Nonstoppers measured the least positive on each of these variables when compared to the Continuing Successes and Recidivists. In each of the comparisons, there was a significant difference between Continuing Successes and Nonstoppers. Recidivists were significantly different from Nonstoppers and very close to Continuing Successes in their expectation of success, the number of group sessions they attended, the number of group sessions attended by their significant others, and their previous attempts at cessation. The Recidivists were significantly different from the Continuing Successes on the amount of stress measured by (1) weighted scores at the first and second annual visits, (2) ease and success of prior cessation attempts, (3) amount of negative effect experienced as measured by weighted scores at the first and second annual visits, (4) personal security, (5) number of cigarettes smoked at baseline (but not when smoking heaviest), and (6) amount of change between the time when smoking heaviest and entry into the program. Findings indicated that variations in the following factors were able to significantly discriminate among the three cigarette smoking behavior change groups: stress, personal security, social support as measured quantitatively by the number of group sessions attended by significant others and by the smoking environment, degree of negative effect experienced, and number of cigarettes smoked at baseline and when smoking heaviest. Also found to be a significant discriminant was experience of control as measured by expectation of success, the number of group sessions attended, the session of cessation, past experience of control, and degree of externalization.

80–0423. Office of Population Censuses and Surveys. **Cigarette Smoking.** Office of Population Censuses and Surveys Monitor, General Household Survey 1978, Government Statistical Service, Reference GHS 79/2, London, August 7, 1979, 8 pp.

This report presents information on cigarette smoking habits in Great Britain collected in the General Household Survey during 1978, together with comparative data from the 1972-1976 period. The decline in the prevalence of smoking, which has been observed since 1972, was more evident among men (from 52 to 45 percent) than among women (from 41 to 37 percent) and appears to be mainly due to a decrease in the proportion of those who were "light" smokers (fewer than 20 cigarettes a day). The trend toward increased consumption per smoker, which was evident among both sexes between 1972 and 1976, appeared to have been halted in 1978, particularly in men. The proportion of smokers fell among all age groups between 1972 and 1978, but the greatest decline took place among men between 16 and 24 years of age and those over 60. Both prevalence and consumption per smoker continued to be lower than average among the young and the elderly of both sexes. The decline in the proportion of smokers was observed among men and women in all socioeconomic groups, particularly among professionals. Average weekly cigarette consumption increased among all socioeconomic groups between 1972 and 1976, but the increase continued only among unskilled workers between 1976 and 1978.

80–0424. Petit, M. **L'Adolescent et le Tabac. [Tobacco and the Adolescent.]** *Gazette Medicale de France* 84(19): 1993-1996, May 13, 1977, French.

Behavior in relation to smoking in the adolescent may be analyzed from a psychodynamic standpoint. A review of the different stages of personality development aids in understanding certain pressures which may be satisfied by the use of tobacco. This satisfaction reveals conflicts which the adolescent had to overcome in early childhood. A second level of analysis involves not so much the special oral pleasure of tobacco, but rather its significance in a group context where morals and taboo are more liberal and where role models are usually hypersexed or seductive (e.g., cowboys, vamps, playboys). At this stage, the subject-group dialogue makes it more difficult to approach the problem, while its study encourages reflection on the realities of an antismoking campaign. (Auth. Abs. Mod.)

80–0425. Polsky, R. H.; Chance, M.R.A. **An Ethological Perspective on Social Behavior in Long Stay Hospitalized Psychiatric Patients.** *Journal of Nervous and Mental Disease* 167(11): 658-668, November 1979.

Ethological methods were employed to gather normative data on social behavior in long stay male inpatients in the ward environment. Most were diagnosed schizophrenic. Social behavior was categorized into four main types: altruistic (n=8), assertive (n=19), cigarette (n=4), and verbal (n=17). Behaviors in the cigarette category consisted of those which surrounded the exchange of cigarettes (exchange of money for a cigarette; passing of matches, paper, or cigarettes between patients; rendering of a light; nonverbal signal for a cigarette (held hand out)). Patients (n=24) were divided into three groups on the basis of the amount of social interaction (top, middle, and bottom thirds of the interactional scale). With the use of these divisions, results indicated (a) marked variability between thirds in the amount of social behavior exhibited; (b) differences in the kinds of social behavior utilized by each third—the top was predominantly verbal whereas the middle and bottom were predominantly nonverbal; (c) differences in the modes of interaction between thirds—verbal behavior characterized interactions between the top and top, and top and bottom, whereas assertive behavior characterized interactions between the top and middle, middle and middle, middle and bottom, and bottom and bottom; and (d) correlative relationships between various social and nonsocial parameters, the most noteworthy including the positive relationship between head up, eyes open, and high rate of social interaction, and the positive relationships between altruistic, cigarette, and verbal behavior. Discussion focused on issues of variability between patients, stability of behaviors over time, behaviors indicative of bonding and social attentiveness, profiles of behavior characteristic of each third of the interactional scale, and the ethological constructs of dominance and attention structure. (Auth. Abs. Mod.)

80–0426. Rimpela, M. **Nuorten Terveystavat. [The Health Behavior of the Finnish Youth.]** *Suomen Laakarilehti* 34(11): 849-855, 1979, Finnish.

The results of a governmental study of the health behavior of Finnish youth in 1977 are presented. Individuals born in

1958, 1960, 1962, and 1964 (3,209) were interviewed in July 1977. A second interview was performed half a year later. The participation percentages were 88 and 79 percent respectively. The percentages of daily smokers among the boys and girls respectively were as follows: 18-year-olds, 40 vs. 32; 16-year-olds, 29 vs. 27; 14-year-olds, 11 vs. 14; and 12-year-olds, 1 percent (both boys and girls). Between February and September 1977, 3 percent of the girls and 1 percent of the boys born in 1964 had started smoking; 1/5 of the girls and 1/3 of the boys had stopped. Of the individuals born in 1962, 7 percent of the girls and 8 percent of the boys had started; 13 percent of the girls and 16 percent of the boys had stopped. Of those born in 1960, 8 percent of the girls and 11 percent of the boys had started, 13 percent of the girls and 10 percent of the boys had stopped smoking. Of those born in 1958, 9 percent of the girls and 10 percent of the boys had started, and 20 percent of the girls and 8 percent of the boys had stopped smoking. Smoking among young people was affected by the social status of their parents, the academic success of the individuals, the smoking habits in their environment, the financial situation of the individual, as well as the structure of the family.

80-0427. Robles, R.; Martinez, R.; Moscoso, M. **Drug Use Among Public and Private Secondary School Students in Puerto Rico.** *International Journal of the Addictions* 14(2): 243-258, 1979.

This paper reports results of the first wave of a two-wave panel survey carried out on a representative sample of 18,712 Puerto Rican public and private secondary school students in the 1974-1975 academic year. Twelve percent of the students reported the use of one or more illicit drugs. The illicit drug most frequently used was marihuana. More than three times as many Puerto Rican adolescents have used alcohol and tobacco than any of the illegal drugs (41 vs. 12 percent, respectively). Of those surveyed, 5.5 percent reported only cigarette smoking, 13.9 percent used cigarettes and alcohol, 1.1 percent used cigarettes and drugs, and 7.4 percent used cigarettes, alcohol, and drugs. Private school students of both sexes reported more use of illicit and licit drugs than their public school peers, suggesting that socioeconomic factors may be influencing Puerto Rican adolescent drug behavior. Differences between Puerto Rican adolescents vary significantly depending on sex. While more males than females used illicit drugs (36 vs. 19 percent), and more males used both cigarettes and alcohol (38 vs. 29 percent), more females than males used cigarettes only (12 vs. 10 percent). It is suggested that Puerto Rican sociocultural conditions may have an important effect upon male-female drug use patterns. One of the most significant findings of the study is the high frequency of drug use among Puerto Rican adolescents who ever lived in other countries. (Auth. Abs. Mod.)

80-0428. Taylor, C. **Doctors Use Multiphasic Screening Plus Personalized Patient Education.** *Australian Family Physician* 8(3): 296-297, 299-301, 303-304, March 1979.

The application of a system of prospective medicine is described as a method whereby personalized health education is based on each patient's health evaluation data. A group of 509 clergy and their wives (mostly nonsmokers) participated in a health evaluation and education program sponsored by the First Australian Institute of Total Health Limited. Of the subjects, 31 percent had an elevated health risk assessment age; 54 percent had high serum cholesterol levels; 20 percent had elevated blood pressure; 16 percent were obese; and 24 percent led very sedentary lives. During the second year after the initial evaluation, a followup of 156 of the participants showed 64 percent had reduced their health risk assessment by an average of 3.4 years, 24 percent had increased theirs by about 2.2 years, and 11 percent had experienced no change. The patient's health risk assessment age was calculated from the following data: a Health Habits and Relationships Questionnaire, which included questions about the patient's smoking habits; vital lung capacity measurements; ECG; blood pressure measurements; and serum cholesterol levels. Guidelines are suggested for the application of multiphasic screening combined with patient education to a general practice. The patients should have medical health appraisals and counseling, and be encouraged to stop smoking, limit alcohol consumption, exercise properly, and manage stress effectively. To illustrate the potential impact of diet, exercise, and smoking cessation, it is suggested that the health age be recalculated for the patient, using ideal values (e.g., no cigarettes smoked) for these variables.

80-0429. Valeriote, C.; Tong, J. E.; Durding, B. **Ethanol, Tobacco and Laterality Effects on Simple and Complex Motor Performance.** *Journal of Studies on Alcohol* 40(9): 823-830, September 1979.

The effects of alcohol and tobacco on three simple motor tasks (slow knob turning, finger and foot tapping) and on one complex task (pursuit rotor) were studied in 24 men aged 19-27 years. All subjects were moderate smokers, smoking from 15 to 25 cigarettes daily, and moderate social drinkers, drinking alcoholic beverages at least once a week. In 6 test sessions, each subject was tested under 12 conditions derived from a counterbalanced repeated-measures 2 (laterality) x 2 (alcohol) x 3 (tobacco) design. To separate the effects of nicotine administration from nicotine deprivation, three tobacco conditions were used: deprived (4 hours), no cigarettes; deprived (4 hours), two cigarettes; and not deprived, no cigarettes. The two alcohol conditions were a placebo beverage and an alcoholic beverage aimed to give a peak blood alcohol concentration (BAC) of 0.06 percent. In one of the tobacco conditions, two commercial filter cigarettes 7 cm long were smoked. Each cigarette contained 1.3 mg of nicotine. The cigarettes were smoked at the rate of 1 puff per 30 sec to a mark 25 mm from the filter. Since no satisfactory placebo cigarettes are available, no tobacco was used in the no-cigarette condition. The alcoholic beverage consisted of Liquor Control Board of Ontario 35 percent alcohol at a dose of 2.5 ml per kg of body weight. Orange juice was added to equal 1.5 times the volume of alcohol. The placebo beverage consisted of orange juice (with 4 ml of alcohol floated on top) equivalent in volume to the alcoholic beverage for a given subject. Performance on the knob turning task was not significantly affected. Alcohol significantly lengthened intertap intervals for finger ($p<0.001$) and for foot ($p<0.003$) tapping. Alcohol also reduced the time on target on both circular and triangular plates ($p<0.001$) for the pursuit rotor task. Tobacco had no main or interactive effects on these motor tasks. However, a significant ($p<0.02$) interaction was present for tobacco and laterality for the circular plate only.

80-0430. Williams, C. L.; Eng, A.; Botvin, G. J.; Hill, P.; Wynder, E. L. **Validation of Students' Self-Reported Cigarette Smoking Status With Plasma Cotinine Levels.** *American Journal of Public Health* 69(12): 1272-1274, December 1979.

Plasma cotinine levels were measured in 137 students (64 males and 73 females, ranging in age from 14 to 17 years) as an independent validation of self-reported cigarette smoking status. Of the 20 students who reported daily cigarette smoking, 95 percent had detectable cotinine levels. In contrast, only 2 percent of the 98 students who reported that they never smoke cigarettes had detectable levels of plasma cotinine. Of the 19 students who reported occasional smoking, 21 percent had detectable cotinine levels. These 4 students smoked an average of 16 cigarettes a week, while the other 15 occasional smokers with nondetectable cotinine levels smoked only an average of 1 cigarette per week. The female to male smoking ratio was almost 2:1 among the daily smokers and almost 3:1 among the students who smoked occasionally. Results suggest that adolescents can report accurately on their smoking status if sufficient assurance of confidentiality is stressed. (Auth. Abs. Mod.)

See also, **80-0253, 80-0307, 80-0436**

SMOKING CESSATION METHODS

80-0431. Brengelmann, J. C. **La Therapie Comportementale des Etats de Dependance. [Behavior Therapy of Dependent States.]** *Psychologie Medicale* 8(1): 41-50, 53-60, 1976, French.

Behavior theory was used to treat tobacco, food, alcohol, and drug addicts. The tobacco group consisted of 256 male and 99 female cigarette smokers. The subjects were divided into control and other subgroups and were treated using the following methods: medication (lobeline or placebo), psychotherapy (electric, negative, sensitization), aversion, or self-control. The best results were obtained with the self-control technique. In this subgroup, the number of cigarettes smoked daily decreased by 36 percent after relapse, whereas there was a 10 percent decrease in the controls and a 15 to 29 percent decrease in the other subgroups. A group of 40 untreated subjects and 41 subjects who relapsed after the first treatment was subjected to a self-control procedure combined with contracting (betting) and a nonreturnable deposit of 100 marks. After 8 weeks of therapy at the rate of 2 sessions per week, two-thirds of the subjects decreased their cigarette consumption from 28 to 0, and over 90 percent decreased it to less than 10 cigarettes/day. A group of 249 smokers was also treated (by mail) with self-control combined with contracting and a nonreturnable deposit. The duration of the mail treatment was 12 weeks. Over 60 percent of the subjects ceased smoking and over 80 percent maintained their daily cigarette consumption at less than 10. Fourteen percent abandoned treatment. After 15 months, 44 percent of the subjects treated by mail ceased smoking completely.

80-0432. Cruise, J. S.; Fisher, F.; Cruise, R. J. **An Evaluation of a Smoking Withdrawal Clinic.** *Journal of the Medical Association of Georgia* 68(9): 819-822, September 1979.

The effectiveness of smoking withdrawal clinics operated by the Atlanta Lung Association between 1976 and 1977 was evaluated. The statistical analysis was based on data from 5 consecutive clinics ranging in size from 34 to 86 smokers each, with a total of 325 participants. The clinics consisted of six 1-hour sessions, and utilized the Seventh Day Adventist Church's 5-Day Plan to Stop Smoking. In addition, the director made regular post-clinic followup contacts with the participants. Age, sex, and marital status of the subjects were the variables considered in the analysis. Results of the clinics showed that (1) 63 percent of the participants stopped smoking at the end of the clinic, (2) 13 percent started smoking again within 3 months, (3) an additional 7 percent started smoking again within 6 months, and (4) 34 percent stopped smoking for 1 year or longer. The majority of individuals who started smoking again were in the 30 to 39 age group, followed by the 20 to 29 age group, and most of them were married. Most of the 120 persons who did not stop smoking were married, and 70 percent of the men tended to be in the older age group (40-59). Lack of motivation was the reason given by 62 percent of the individuals who had started smoking again. Discriminant analysis was used to determine if the four groups of people who stopped smoking for different lengths of time differed on the basis of the variables in this study. The most important variable was found to be age: the older a person was, the longer he was able to abstain from smoking. Marital status was the second most important variable: married, divorced, and widowed people were more likely to abstain from smoking for longer periods than single people. The need for better methods of increasing a smoker's motivation to quit was stressed.

80-0433. Fisher, M. L. **Helping Acutely Ill Patients Put Out the Fire.** *American Journal of Nursing* 79(6): 1104-1105, June 1979.

A program designed by a clinical nursing instructor is described which uses behavior modification techniques to help hospitalized postmyocardial infarct patients stop smoking. The patient plays a major role in determining the direction of his own behavior change and, together with the nurse, develops a plan specifically designed for him. Each time he smokes, the patient marks a daily tally sheet describing the time, occasion, and feelings associated with smoking in order to increase his awareness of his actions. The patient is also asked to prepare a list of reasons why he wants to quit. This enables the nurse to determine those factors which are likely to provide the strongest motivation for the patient. To illustrate the technique, a case report is presented of a 56-year-old executive who regularly smokes more than two packs of cigarettes a day.

80-0434. Hamilton, S. B.; Bornstein, P. H. **Broad-Spectrum Behavioral Approach to Smoking Cessation: Effects of Social Support and Paraprofessional Training on the Maintenance of Treatment Effects.** *Journal of Consulting and Clinical Psychology* 47(3): 598-600, 1979.

Two treatment programs were conducted to determine the efficacy of a multicomponent smoking intervention and various maintenance procedures. Prior to Program 1, 60 smokers were randomly assigned to one of three experimental conditions—a smoking abstinence program (SAP), a smoking abstinence program plus social support (SAP-SS), a smoking abstinence program plus social support and paraprofessional training (SAP-SS-PT)—or to one of two control conditions—effort control (EC) or a waiting list (WL). Individuals assigned to EC and WL conditions became treatment participants (EC-SAP-SS, WL-SAP-SS) during Program 2 and received training from SAP-SS-PT subjects. Results indicated that social support and paraprofessional training tended to facilitate maintenance, although 6-month followup data revealed considerable relapse across treatment conditions. (Auth. Abs.)

80-0435. Hynd, G. W.; Stratton, T. T.; Severson, H. H. **Smoking Treatment Strategies, Expectancy Outcomes, and Credibility in Attention-Placebo Control Conditions.** *Journal of Clinical Psychology* 34(1): 182-186, January 1978.

This study assessed the credibility of the rapid smoking procedure, covert sensitization, a combined approach, satiation, and a relaxation technique in a group of 38 self-reported cigarette smokers. The results suggested that while sex is not a factor in credibility ratings, different treatment strategies do elicit varying degrees of confidence in terms of positive treatment expectancies. Posttest comparisons revealed that satiation was least preferred, while the relaxation procedure was rated the highest in credibility. The other aversive treatment strategies did not differ significantly from either the relaxation or satiation procedures. These findings are discussed as they relate to credibility as a nonspecific treatment variable that smoking researchers may effectively manipulate through a rating procedure in creating a more sound experimental design in attention-placebo control conditions. (Auth. Abs.)

80-0436. Johns, P. A. **A Study of the Smoking Behavior of Participants in the Stop Smoking Program of the American Cancer Society.** Doctoral Dissertation, University of Georgia, School of Education, Athens, University Microfilms 79-15490, 1978, 146 pp.

The purpose of this investigation was to examine the effectiveness of the American Cancer Society's Stop Smoking Program in select districts of Georgia in order to identify potential predictors of successful completion of the program. In addition, the effect of participation in a Stop Smoking Program upon smoking behavior was examined. The experimental sample ($n=205$) was composed of participants in the Stop Smoking Program. Individuals who expressed interest in attending a program, but who had not yet done so were assigned to the control group ($n=31$). A confidential questionnaire and the Smoker's Self-Testing Kit (SSTK) were completed during the course of the Stop Smoking Program. Smoking behavior was assessed at 1 month, 3 months ($n=184$), 6 months ($n=151$), and 1 year ($n=115$) after the end of the program. In order to determine if participation in the program affected smoking cessation, related data were analyzed using the Chi square statistic and, where appropriate, phi coefficients to assess the magnitude of the effect between variables. Analysis of the data showed that there was a low association ($p<0.001$) between smoking behavior and participation in a Stop Smoking Program ($\varphi=.311$ at program end; $\varphi=.248$ 30 days later) when the entire experimental sample was compared to the control group. (Auth. Abs. Mod.)

80-0437. Lando, H. A. **A Manual for a Broad-Spectrum Behavioral Approach to Cigarette Smoking.** American Psychological Association, JSAS Manuscript No. 1371, Washington, D.C., 1976, 25 pp.

This manual provides a detailed description of specific treatment techniques isolated in more than 6 years of research with chronic smokers. A combination of aversion and maintenance procedures produced an abstinence rate of 76 percent at a 6-month followup, one of the most successful results obtained to date. A brief background and rationale for the techniques employed are provided. The aversive component of treatment involved 1 week of intensive smoking in which participants were required to at least double their usual smoking consumption. Participants attended six laboratory sessions during the week of intensive smoking. The maintenance program included contractual management (forfeiture of money for each cigarette smoked, self-reward/self-punishment contingent upon smoking), booster sessions, and follow-up contact and support. A stimulus-control procedure was studied as the first element in a comprehensive three-phase program of preparation, intensive smoking, and maintenance. Participants were required to smoke at set intervals for a 2-week period prior to undergoing intensive smoking. The purpose of this stimulus-control technique was twofold: (a) to bring smoking under the influence of an external cue (time) and (b) to lessen the pleasure derived from smoking and to induce reactance by requiring participants to smoke on schedule rather than at their own volition. An appendix includes a background and medical history questionnaire, a medical consent form, contracts, and a list of suggestions distributed to all participants. (Auth. Abs. Mod.)

80–0438. Menapace, R. H. **The Effects of Fear Arousal and Subjective Probability of Coping Success on Attitudes, Behavioral Intention, and Behavior.** Doctoral Dissertation, Temple University, Philadelphia, University Microfilms 77-13,573, 1977, 76 pp.

Eighty-seven cigarette smokers served as subjects in a 3 x 3 factorial design with after-only measures on the dependent variables. Three levels of fear (high, moderate, and low) defined one independent variable, while three levels of probability of smoking cessation technique effectiveness (high, moderate, and low probability) defined the second independent variable. Smoker eligibility was defined as 10 cigarettes per day for at least 1 year. Experimental subjects were either shown one of two 11-min films (high or moderate fear condition) or asked to fill out a family history of smoking behavior (low fear condition). Following the films or survey, all subjects read communications linking lung cancer to cigarette smoking and including recommendations to quit. The multiple correlation for fear, number of cigarettes smoked, and success probability was .517. A 3 x 3 analysis of variance for intention to quit smoking yielded the predicted fear arousal effect ($p<0.003$).

See also, **80–0413**

80–0439. Rodgers, M. P. **The Effect of Social Support on the Modification of Smoking Behavior.** Doctoral Dissertation, University of Michigan, Ann Arbor, University Microfilms 77-18,105, 1977, 289 pp.

This study included 57 smokers who were assigned to one of three treatment conditions: group support (GS), mail support (MS), and no support (NS). The participants were smoking a mean number of 27 cigarettes/day at the program's start, had been smoking an average of 14 years, and had tried to quit smoking an average of 4.6 times. All subjects were told that the program consisted of two 2-hour workshops on self-management training for smoking termination. The GS subjects were also told that they would have six additional small group meetings. Each subject identified negative and positive reinforcers and wrote a contract specifying (1) what reinforcements and stimulus control methods they would apply and (2) how many cigarettes they would reduce each week before quitting entirely. GS treatment was significantly better than NS treatment at 1, 2, and 3 months ($p<0.05$). Although GS was better than MS at all time periods, there was no significant difference. MS was better than NS at 1 and 2 months ($p<0.05$).

80–0440. Sutton, S. R. **Interpreting Relapse Curves.** *Journal of Consulting and Clinical Psychology* 47(1): 96-98, February 1979.

It has been argued that the shape of the relapse curves for smoking, alcohol, and heroin supports a learning conception of relapse in terms of decay of extinction. The present article shows, however, that the relapse curve cannot suggest or support a theory of relapse. Like all survival curves, it reflects the operation of one or both of two general processes—selection of the surviving individuals and changes in individuals' probabilities of survival—neither of which is specific to relapse. However, by comparing individuals who relapse soon after treatment with those who relapse later or remain abstinent, one may be able to identify the factors influencing probability of survival and to develop a theory of relapse. (Auth. Abs.)

TOBACCO ADDITIVES

80–0441. Wakeham, H. H. **Future Directions in Flavor Controls.** *Tobacco Reporter* 105(10): 43, 68, 70, October 1978.

Flavor additives have long been used in the manufacture of tobacco products, even before cigarettes were accepted as the way to use tobacco. But the traditional cigarette tobacco smoke flavor suffers badly from high filtration and air dilution in the new low delivery products. Public acceptance of such cigarettes requires that somehow the semivolatile flavorful components in smoke be increased relative to the other components. Since many of the important flavor compounds are volatile or semivolatile substances, ways had to be found to fix them in the cigarette so that they would not be released or lost until the cigarette was smoked. Early efforts have concentrated on controlling menthol, and several methods have been used. In one of the earliest methods, menthol was introduced via filter charcoal from which it is released when smoke components are absorbed as the cigarette is puffed. A second approach with menthol has been to prepare carbonate esters of menthol and then polymerize them. The result is a stable compound which decomposes at elevated temperatures. Such a material applied to the tobacco will release menthol when the cigarette is smoked. Still another way to fix flavorants such as menthol is to use inclusion complexes containing the flavor compound. A further extension of this principle is the use of microencapsulation where the flavorant is enclosed in tiny polymer-gum beads which rupture when heated, releasing the flavorant. Although many variants of these general methods for fixing flavorants in the cigarettes have been described in the patent literature, only the charcoal filter method has been commercially successful, probably due to the cost of the other methods.

TOBACCO MANUFACTURING AND PROCESSING

80–0442. Coq, F.; Vidal, S. **Formation of the Porous Structure of Cigarette Filters.** *Beitraege zur Tabakforschung* 8(4): 225-232. December 1975.

An original method based on permeability measurement is applied to studying the formation of the porous interstitial structure of cigarette filters. Using this method, it is shown that the porous structure of a filter is formed in the course of the filling process, which is accomplished by compression of the filter material without significantly modifying the specific structure of this material. In the filling process, the elements of the filter material are assembled in the filter by overlapping. A given filter material is characterized by the specific volume it takes up when its elements are assembled at the compression limit without any crushing. A filter material is, moreover, characterized by the manner in which its elements are assembled during the formation of the porous interstitial structure of a filter. The permeametric method allows the definition of this characteristic. The results can be applied to the choice of filter material and to the determination of the optimum filling rate, permitting the rapid development of a cigarette filter with fixed characteristics. (Auth. Abs.)

80–0443. Tso, T. C.; Gori, G. B. **A Novel Approach in Tobacco Production as Food Source and Smoke Material—Year 1976 and Year 2000.** In: *Proceedings of the Sixth International Tobacco Scientific Congress*, Tokyo, November 14-20, 1976, The Japan Tobacco and Public Salt Corporation, 1977, pp. 81-86.

The tobacco plant is a potential source of drugs, chemicals, and pesticides. One promising area in which tobacco production can be made more economical entails utilization of the protein fraction, especially Fraction I Protein from tobacco as a source of food. Fraction I Protein is composed of amino acids similar to those found in cow or human milk, and the yield of this protein in tobacco may be as high as 400 lbs per acre. The remaining tobacco materials can be reconstituted as a smoking product of desired chemical and physical properties. Toward the above goals, a theoretical model which meets the requirements of leaf desirability is established. Novel cultural, curing, and postharvest handling methods to achieve this model are discussed. Basic information and detailed data are presented. It is feasible that in the year 2000 tobacco can be used both as a desirable smoking material and as a source of food and other valuable products. (Auth. Abs.)

TOBACCO ECONOMICS

80–0444. Gregory, F. H. **Gearing for Thailand's Smoking Requirements.** *Tabak Journal International* (6): 392-393, December 1978.

The Thailand Tobacco Monopoly (TTM) has the sole right to manufacture cigarettes in a country where imported cigarettes are banned and where cigar and pipe smoking is negligible. These circumstances have led to increasing profitability, and the TTM now provides a revenue for government departments that is higher than any other state enterprise. Recently a new factory has been opened and an expensive new brand of cigarettes introduced onto the market. These moves are in accordance with the management's policy of first increasing production to meet increasing demand and, second, improving the quality of their products. Although they are using greater amounts of locally grown tobacco, it is realized that foreign tobacco is an essential ingredient if the quality of their cigarettes is to be maintained or improved. The present management, therefore, expects that they will need to import tobacco for years to come. (Auth. Abs.)

80–0445. Maier, F. **Die Tabakproduktion in der EG 1976 und 1977. [Tobacco Production in the European Economic Community 1976-1977.]** *Der Deutsche Tabakbau* 57(23): 301, December 1, 1977, German.

In 1976, net increases in total tobacco acreage and in total tobacco crop yield are noted for four principal tobacco producing countries. The total acreage increase occurred predominately in France and Italy. Projected figures for 1977 show an acreage decrease of approximately 3,000 hectares and a crop yield decrease of 8,000 tons. These figures reflect anticipated decreases in Italy and France, respectively. In 1977, Italian production of Beneventano and Orientalis tobacco varieties decreased and production of Geudertheimer, Forchheimer Havanna IIc, and Paraguay varieties increased.

80–0446. Poehl, H.-G. **Cigarette Consumption in the Federal Republic of Germany—Slight Recovery in 1976.** *Tabak Journal International* (2): 85, 88, 90, April 1977.

The German cigarette industry had a satisfactory year in 1976. With 128.04 billion cigarettes which, expressed in retail prices, correspond to a turnover of about 15 billion DM, the industry achieved not only its best sales result ever, but with 147.3 billion cigarettes also an all-time production record. Sales of filter cigarettes again showed above average growth. Of the 128.04 billion cigarettes sold, 110.57 billion were filter cigarettes. The share of filter cigarettes thus increased from 86 to 86.36 percent of total sales. Although sales of cigarettes of low nicotine content and those without nicotine increased by about 800 million from 34.79 to 35.60 billion cigarettes, the share in the overall market fell slightly from 28.07 to 27.80 percent. Cigarette smokers paid 5.5 percent more in tobacco tax during 1976, and the total tax revenue from the consumer tax on cigarettes reached 9.4 billion DM in 1976 which constituted 7.11 percent of all the taxes collected by the Federal Republic. The sales increase of about 4 billion cigarettes or 3.2 percent compared with 1975 is all the more remarkable when one considers the continuing attacks on smoking. (Auth. Abs. Mod.)

80–0447. Tobacco Reporter. **On Interconnecting Interests: Rembrandt Buys More Mines.** *Tobacco Reporter* 103(12): 23-26, December 1976.

Tobacco companies in recent years have increasingly diversified, investing in mines, insurance, food products, and shipping. As a result, tobacco firms are receiving a growing percentage of their income from nontobacco sources. In the case of the Rembrandt group, nontobacco income increased from zero in 1971 to 13 percent in 1976; for Brown and Williamson, from 6 to 20 percent; and for British-American, from 7 to 26 percent during the same period.

See also, **80–0443**

BILLS AND LEGISLATION

80–0448. Federal Trade Commission. **Report to Congress Pursuant to the Public Health Cigarette Smoking Act for the Year 1978.** Federal Trade Commission, Washington, D.C., December 24, 1979, 32 pp.

This report includes information concerning 1978 domestic cigarette sales and advertising; cigarette labeling effectiveness; current practices, methods, themes, and appeals of cigarette advertising and promotion; nonsmokers' rights; regulatory activity; and legislative recommendations. Two appendices contain domestic sales and advertising statistics, and an FTC staff analysis, "A Study of Public Attitudes Toward Cigarette Smoking and the Tobacco Industry in 1978," by the Roper Organization. There have been significant gains in reducing smoking prevalence among almost all segments of the population. The percentage of all adults who smoke regularly was 33 percent in 1978, the lowest point in over 30 years. The percentage of adult men who regularly smoke declined from 53 to 38 percent between 1955 and 1978, and while the percentage of adult women who regularly smoke had increased from 25 to 32 percent between 1955 and 1965, it decreased to 30 percent by 1978. Nevertheless, in one category, women aged 18 to 25, smoking prevalence is increasing. Perhaps the most important reduction in smoking prevalence has been the drop in teenage smoking over the past 5 years. The data suggested that teenage smoking habits are major determinants of lifelong cigarette consumption. The percentage of smokers of both sexes dropped from 15.6 percent of all teenagers aged 12 to 18 in 1974, to 11.7 percent in 1979. However, the percentage of teenage girls 17 to 18 years old who smoke has risen 0.3 percent since 1974, to 26.2 percent in 1979. Current themes in cigarette advertising fell into the following categories: attempts to minimize the health hazards of smoking; appeals to taste, pleasure, and satisfaction; association of smoking with positive personality characteristics such as sophistication, intelligence, rugged individualism, and youthful, attractive appearance; association with success in such endeavors as romance, adventure, business, and social relationships; association with outdoor and athletic activities; and association with fresh air and clean environment.

GENERAL

80-0449. Albertson, P. **Therapeutic Potential for Marijuana and its Derivatives.** *Medical Tribune and Medical News* 20(13): 16-21, March 28, 1979.

Marihuana has a long therapeutic history. One of marihuana's major constituents, delta-9-tetrahydrocannabinol (THC), and the synthetic cannabinoids have been of great interest to scientists studying intraocular pressure (IOP), asthma, and depression. Focus has since turned to cardiovascular effects. An average total dose range of 1.5 to 3.0 mg THC given to IOP study subjects resulted in an increased heart rate in 6 of 10 subjects, with an increase range of 95 to 142 beats/min while on the higher dose. There was no bradycardia and no statistically significant change in blood pressure. Two patients who were given the higher dose, however, experienced a marked decrease in blood pressure in the range of 80/40 mmHg with presyncopal symptoms. In a study of patients with angina, marihuana use was linked to developing angina at a lower exercise level than after smoking a cigarette. One long-term effect of marihuana smoking in young men who smoked 5 marihuana cigarettes/week for 5 years was significantly impaired resistance to air flow compared to that in cigarette smoking controls. Marihuana is helpful as a bronchodilator in some asthma subjects; however, marihuana also has irritating side effects such as moderate to severe bronchoconstriction, cough, and chest discomfort in other asthma subjects. Beneficial effects of THC were observed while testing for use as an antidepressant. A double-blind study in cancer patients receiving chemotherapy compared the effects of 0.1 mg/kg t.i.d. orally of THC with placebo. The principal benefits included improved appetite, lack of expected weight loss, and more control of nausea and vomiting.

80-0450. Bjartveit, K. **Tobakksloven i Norge—Erfaringer fra Gjennomforingen. [The Tobacco Law of Norway—Experiences from the Realization.]** *Nordisk Medicin* 91: 224-226, 1976, Norwegian.

The prohibition of tobacco advertisement and the requirement that packages of tobacco products be provided with health hazard warning labels, as well as declarations of contents, are included in the Norwegian Tobacco Law of July 1975. It was expected that the effect would mainly be seen as a decrease in the number of new smokers. Smoking surveys have been performed regularly since 1973. Among men of all age groups a statistically significant decrease in the number of daily smokers was found between June and December 1975. The percentage of smokers dropped from 52 percent in June to 49 percent in September and 48 percent in December. Among the cigarette smokers the average consumption in June was 14.4 cigarettes per day; in September and December 14.1 and 13.2, respectively. Among women the percentage of daily smokers remained constant at 33 percent with an average of 10.6 cigarettes smoked per day. There had, however, previously been a steady increase in the number of daily smokers among women.

80-0451. Buchbinder, B.; Helzer, S. G.; Offensend, F. L. **Preliminary Report on Evaluating Alternatives for Reducing Upholstered Furniture Fire Losses.** U.S. Department of Commerce, National Bureau of Standards, Publication No. NBSIR 77-1381, November 1977, 30 pp.

This paper presents preliminary results from a pilot project designed to test the utility of applying decision analysis to fire hazard problems. To test the methodology, an analysis is being performed to determine the effectiveness and economic consequences of alternative intervention strategies for reducing upholstered furniture fire losses in residences. A probabilistic model has been developed to assess quantitatively the expected fire losses under each alternative. This paper describes the analysis on one alternative: the proposed upholstered furniture standard currently under consideration by the Consumer Product Safety Commission. The loss model for this alternative is described in some detail. Preliminary results on costs, losses, and cost plus loss to society and the present value of these quantities are presented. For cigarette, open flame, electrical, and other ignition sources the current numbers of smoldering ignitions were 88,117; 2,613; 2,447; and 8,709, respectively. Corresponding current ignition source probabilities were 0.70, 0.13, 0.05, and 0.12. (Auth. Abs. Mod.)

80-0452. Cady, B. **Smokers Should Pay the Medical Costs of Smoking.** *Resident and Staff Physician* 25(4): 44, April 1979.

It is estimated that 370 million dollars (10 percent of all health care costs) are spent each year in Massachusetts alone for illnesses caused by tobacco. Two methods are suggested by which these medical costs could be apportioned to the actual users of tobacco (one-third of the population), rather than be distributed among all of the 5.8 million inhabitants of the State as they presently are: (1) a "health tax" could be added to each package of cigarettes sold; and (2) health and life insurance costs could be substantially increased for smokers and proportionately reduced for nonsmokers.

80-0453. California Fire Incident Reporting System. **Annual Report. California Fire Incident Reporting System. 1977.** Office of the California State Fire Marshal, Sacramento, 1978, 94 pp.

This fourth annual report of the California Fire Incident Reporting System summarizes data submitted for calendar year 1977. Between 1976 and 1977, there was an overall reduction of 19,463 fires, primarily due to 12,130 fewer wildland and brush fire incidents. The following information is presented in 23 tables: the number, percentage, and type of fire, by time of day and day of week; the number and percentage of annual fires, according to property classification and type of fire; property classification and property complex, management, type, and number of stories; extent of fire damage and number and costs of fires per range of property loss; number and costs per range of content loss; fire area and level of origin; source and form of heat causing ignition; type and form of material first ignited; the act or omission causing ignition; fire casualty reports; and fireworks. Two appendixes explain the property classifications used in the tabulations and the table column headings. Smoking materials, such as cigarettes, cigars, and pipes, caused 1,758 of the 35,767 fires in passenger road vehicles and 594 of the 1,428 fires in hotels, motels, or inns. Overall, 18,547 of a total of 194,585 fires were started by smoking materials.

80–0454. Curran, T. L. **Physicians Who Smoke. (Letter).** *New England Journal of Medicine* 298(18): 1037, May 4, 1978.

Physicians, as a group, are aware of the high risk to the fetus of the smoking mother and many of them have set a good example for their patients by giving up smoking themselves. A case is reported of a woman in her ninth month of pregnancy who refused medication prescribed by the respondent for otitis media because of possible harm to the fetus. Although informed that her smoking habit of one to two packs a day was more detrimental to the fetus than the medication, she did not stop smoking, probably because her obstetrician smoked two packs a day and was not likely to advise his patients about the dangers of smoking. Physicians who smoke are admonished to "heal themselves."

80–0455. Dufour, A. **Cent Ans de Lutte Antitabagique. [Hundred Years of Anti-Smoking Campaign.]** *Histoire des Sciences Medicales* 12(2): 136-142, 1978, French.

A history of tobacco consumption in France from 1560 to the present is presented. Sporadic resistance to the use of tobacco was noted in France in 1600, when King Henri IV prohibited its use, and in 1602 when the Paris Police Department prohibited its sale and use in taverns. Parallel efforts to suppress tobacco use were observed in several European countries such as Russia, Turkey, and Iran. Only after cigarettes were introduced in 1843 and the first smoking-related accidents occurred, did a serious antismoking campaign supported by scientific findings begin in France. A one-man antismoking crusade begun in 1838 is credited with the legal prohibition of smoking in many public places and the doubling of the tobacco tax. Few of these measures were actually enforced, however, and cigarette consumption in France increased from 1 billion in 1900 to 83 billion in 1975 with a corresponding increase in tobacco-related disease incidence. Resurgence of antismoking activity in France was based on the 1971 London publication, "Smoking and Health Now," a report of American and British research from 1956 to 1970. A series of recommendations on tobacco smoking in France presented in 1972 were unanimously approved by the Academy of Medicine and presented to the Government. The Government acted only when Giscard d'Estaing, a nonsmoker, was elected as President and Mrs. Simone Veil was appointed as Minister of Health. The antismoking campaign launched by the Minister was aided by other ministers, the Academy, and numerous associations, and resulted in promulgation of the basic "Law on Smoking Prevention" of July 9, 1976, which was implemented by the decrees of September 12, 1977, and November 17, 1977. These decrees prohibit smoking in schools, hospitals, food storage and sale premises, public road transport vehicles, and elevators, and make separation of smokers mandatory in trains, airplanes, and commercial navigation vessels. Further, the decrees regulate press advertisement.

80–0456. Elliott, J. **Many Questions, Fewer Answers, About Pot.** *Journal of the American Medical Association* 243(1): 15, 18, January 4, 1980.

Currently 43 million Americans, roughly 30 percent of the adult population, are known to have tried marihuana at least once. There are 16 million who use it regularly and, most alarming, 4 million are under age 17. Today research is focusing on three areas of particular concern: the effects of the drug on the lungs, on reproductive function, and on the brain. The strongest consensus on the biologic effects of marihuana probably concerns lung damage. Because marihuana smoke does contain a wide range of hydrocarbons, and apparently more carcinogens than tobacco smoke, it can be expected to cause some damage to the lungs and upper airways. Results of some studies indicate the smoke is significantly more toxic than tobacco smoke. Recent animal studies describing effects of marihuana on the lung, the brain and ovulation and pregnancy outcome in monkeys, are described.

80–0457. Eriksson, C.-G.; Tibblin, G. **Hur Gick det for 30-talets Idrottsman? [What Happened to the Athletes of the 1930's?]** *Lakartidningen* 76(15): 1409-1412, April 11, 1979, Swedish.

This report is based on data from a study that began in 1963 of 885 randomly selected men aged 50. Data are presented from a 1973-74 followup study of 718 men which considered the effect of competitive sports on health and health habits. Of the men, 48 percent were regarded as athletes (participating once a year in a sports competition for 5 consecutive years). Of the entire group, 56 percent were smokers, as compared to 62 percent of the football players, 65 percent of the wrestlers/boxers, and 50 percent of the joggers/skiers. High frequency of smoking in a group was associated with a high frequency of alcohol problems. Excess weight, smoking, and alcohol problems were common among former wrestlers/boxers and football players.

80-0458. Fazey, C. **Psychoactive Drug Use and Abuse: A Guide to Sources of Information for Social Scientists.** Preston, Lancashire, England, Preston Polytechnic, 1979, 52 pp.

This booklet is a guide for social scientists interested in publications on the use and abuse of psychoactive drugs including alcohol and tobacco. The guide contains information on strategies for literature searching and a bibliography of primary, secondary, and tertiary sources. Also included is information on sources of research support in the field of drug use and abuse.

80-0459. Fletcher, C. M. **Do People Smoke for Nicotine? (Letter).** *British Medical Journal* 1(6120): 1143, April 29, 1978.

An association between reduced nicotine delivery of cigarettes and increased cigarette consumption between 1950 and 1975 has been reported (*British Medical Journal* 1(6117): 922, April 8, 1978). The respondent points out that the annual consumption per adult smoker over a 25-year period describes the effect of a variety of changes in a population of smokers that itself is changing. Average consumption per smoker has increased because many men who were lighter smokers have stopped, and women who are now smoking tend to smoke more. Research indicates that large abrupt reductions of nicotine are followed by increased consumption, but not in the case of smaller changes such as from 1.4 to 1.0 mg of nicotine per cigarette. See also 78-1392.

80-0460. Gopalachari, N. C. **Carcinogenicity of Tobacco Smoke and Measures to Produce "Safer" Tobacco.** *Tobacco News* 1(4): 1-3, October 1977.

The role of tobacco smoke in carcinogenesis having been established, tobacco scientists have attempted to develop measures to reduce the tumorigenic activity of tobacco smoke. Among suggested measures to achieve these objectives are reduction of particulate matter, reduction of tumorigenic agents, and reduction of ciliotoxic agents through (1) genetic selection of tobacco having low concentrations of nicotine, proteins, carbohydrates, and resins; (2) treatment of tobacco for extraction of undesirable components; (3) tobacco additives; (4) blending; (5) control of the amount of tobacco by varying the proportion of raw tobacco, stems, and homogenized tobacco; (6) varying the cut of shreds; (7) porosity of paper to ensure better oxidation; and (8) using filters for the reduction of harmful smoke components.

80-0461. Hamza, J. **A Szermaradvanyok Dohanyzasegeszsegugyi Vonatkozasai. [Health Related Aspects of Residues in Smoking.]** *Dohanyipar* (2): 46-50, March-April 1978, Hungarian.

Pesticide residues and their decomposition products on tobacco raise new health-related problems of smoking, as during pyrolysis they may enter the mainstream smoke. In estimating the health effects of pesticide residues the following points must be considered: (1) the daily consumption of cigarettes, (2) the transfer rate of residues from tobacco to smoke, and (3) the quantity of residues absorbed by the body. The literature concerning these three factors is reviewed.

80-0462. Hersey, P. **Smoking and Natural Immunity to Tumours. (Letter).** *Medical Journal of Australia* 2(5): 233, September 8, 1979.

A possible explanation of the association of smoking with a higher incidence of certain malignant diseases is the possible adverse effect of smoking on the immune system. Several studies with mice chronically exposed to cigarette smoke showed suppression of primary and secondary antigen response in lung tissue-associated lymphoid cells. Chronic cigarette smokers have been found to have elevated leukocyte counts, lower levels of certain natural antibodies, and lower antibody response after vaccination with influenza virus compared with nonsmokers. In another study, natural killer cell activity against cultured tumor cells was lower in smokers than in nonsmokers. Closer examination of the effects of this environmental hazard on the immune system in relation to neoplasia appears warranted.

80-0463. Hutt, P. B. **Unresolved Issues in the Conflict Between Individual Freedom and Government Control of Food Safety.** *Ecotoxicology and Environmental Safety* 2(3/4): 447-469, December 1978.

A partial list of common food components found to be carcinogenic in test animals includes benz(*a*)anthracene, benzo(*a*)pyrene, cadmium, caffeine, dibenz(*a, b*)anthracene, polyaromatic hydrocarbons, pyrrolizidine alkaloids, and tannic acid. The centerpiece of any future discussion of disease prevention through regulation of food safety must be a very straightforward and realistic appraisal of the practicality of prevention techniques in light of the constitutional and ethical limitations imposed by our society and the unwillingness of many citizens to voluntarily change their"vices." Among these are alcohol and caffeine consumption and smoking. The annual cancer risk for smokers is 1 per 800, based on epidemiological data. For those enclosed in a room with a smoker, the annual cancer risk is 1 per 100,000, based on equivalent concentration of benzo(*a*)pyrene in cigarette smoke. Focusing on the issue of "free choice" will be helpful in targeting (1) those areas where prevention techniques can be effective because they do not involve significant diminution of individual choice (e.g., reducing water and air pollution), and (2) those areas where they will be of limited effectiveness because they depend upon individuals voluntarily changing their life-styles when they do not wish to do so (e.g., reducing caloric intake rather than consuming artificially sweetened food).

80-0464. Miller, G. H.; Schumaker, J.; Gori, G. B.; Lynch, C. J. **Less Hazardous Cigarette Debated. (Letters).** *Journal of the American Medical Association* 242(10): 1029-1031, September 7, 1979.

A response to an article on less hazardous cigarettes (*Journal of the American Medical Association* 240(12): 1255-1259, September 15, 1978) cites various discrepancies. Among these are (1) invalid critical value computations, (2) improper selection of tar and nicotine values, (3) incorrect proportional impact assessment of toxic substances, (4) inattention to carboxyhemoglobin research data, and (5) inappropriate assumptions of additive safety. The authors of the original article refute each of these criticisms. See also 79-0369.

80-0465. Neumann, H.-J. **Determining the Concept of Condensation Products Caused by Tobacco Smoking.** *Tabak Journal International* (6): 385-386, December 1978.

It is correct to define as "condensate" those vaporous organic reaction products which arise in addition to combustion products (i.e., carbon dioxide and water) when cigarettes are smoked, and which can condense when cooling to form an aerosol of a liquid phase in a gaseous phase. This condensate is clearly defined in DIN 10240 as "dry smoke condensate." Reference to this composite substance as "tar" must be rejected, as it is incorrect and can lead to inaccurate assumptions regarding occurrence and composition. (Auth. Abs.)

80-0466. Nicolle, P. **Un Precurseur de la Lutte Contre le Tabagisme. [A Precursor of Smoking Control.]** *Histoire des Sciences Medicales* 12(2): 143-144, 1978, French.

An 1869 brochure warned against tobacco abuse, citing its interference with bronchi and nervous system functions such as memory and intelligence. To control the smoking habit, use of low-nicotine tobacco varieties, education of the general public by means of conferences, and prohibition of the sale of tobacco to children and juveniles were proposed. Family doctors were advised to use their influence with youth in order to persuade them to renounce their smoking habit. Also, the hope that the Society Against Tobacco Abuses, founded in Paris in July 1869, would show satisfactory results was expressed.

80-0467. Parliament of the Commonwealth of Australia. **Drug Problems in Australia: An Intoxicated Society?** Report from the Senate Standing Committee on Social Welfare, 1977, 233 pp.

This report on drug use in Australia contains information concerning models of control and national strategy, alcohol, tobacco, analgesics, cannabis, amphetamines and barbiturates, and education. Data from studies of consumption, sales, and user age are compiled. Existing legislation and recommended modifications are also discussed. For tobacco, the Committee recommends that (1) the Commonwealth Government declare as policy its intention to decrease the consumption of tobacco at the rate of 2 percent a year for the 5 financial years commencing with 1978-79, (2) excise policy be one tool used to reduce the consumption of tobacco, (3) the Commonwealth Government end its annual financial contribution to the Tobacco Industry Trust Account, (4) State Governments cease their contributions to tobacco-growing research, (5) the Commonwealth Government gradually move toward ending all specific and general subsidies to the tobacco industry within the next 10 years while ensuring adequate structural readjustment arrangements for tobacco producers, and (6) the Commonwealth Government ensure that the burden of any readjustment falls first on imported leaf. For possession of marihuana for personal use as already defined in most States, the Committee recommends that (1) the offense not be legally defined as a crime, (2) the penalty be solely pecuniary and at a fixed amount approximate to that now being imposed by the courts in most States (i.e., $100 to $150), (3) the court appearance be either optional for the defendant or mandatory in the event of nonpayment of penalty, (4) police be directed not to fingerprint or photograph defendants, (5) no record of conviction kept by the courts or the police shall be used in subsequent proceedings or in relation to any application by the offender for employment, and (6) a conviction should not, of itself, disqualify a person for employment.

80-0468. Pool, J. L. **Youth Perspectives: Smoking and Health. (Letter).** *Chest* 76(5): 500, November 1979.

Concern that there are 4,500 new smokers starting each day and that 1.7 million girls and 1.6 million boys are teenage smokers prompted a National Interagency Council on Smoking and Health sponsored conference in San Francisco on April 26-27. Successful education techniques such as teenage counseling by teenage counselors and demonstration of adverse health effects (pulse rate, carbon monoxide levels) were discussed. Cigarette advertising, not peer pressure, was described as the influential factor encouraging the smoking habit. The problem of new female smokers was addressed as the percentage of 18- and 19-year-old smokers increased from 19 to 26 between 1968 and 1979. In contrast, the percentage of 17-18-year-old boys who smoked dropped from 30.2 to 19.3 between 1968 and 1979. The importance of the physician's role was mentioned.

80-0469. Schievelbein, H. **Wissenschaft nicht mehr gefragt? Gedanken zu einem Antiraucher-Kongress. [The Scientists Are Not Consulted Anymore? Reflections in Connection With an Anti-Smoking Conference.]** *Munchener Medizinische Wochenschrift* 121(34): 1058-1059, August 24, 1979, German.

Comments on the "Fourth World Conference on Smoking and Health" held from June 18-21, 1979, in Stockholm are presented. The assessment of the WHO Director General on the need for action rather than discussion and the alleged trend of the Conference officials' promotion of political action rather than medical information are deplored. This trend is illustrated by examples from conference proceedings on the Third World, passive smoking, medical and social costs, and smoking withdrawal. In the cases of passive smoking and cost, the conclusions of a U.S. study and two other studies are criticized as scientifically unsupported. An unsuccessful million dollar smoking withdrawal program launched by an important U.S. organization is cited to support the opinion that investment in an information and education program for children would be more productive. Also, it is stated that forcing the tobacco industry to produce less harmful cigarettes would be a promising preventive measure. It is believed that only scientific discussion can lead to progress in solution of the problem of smoking and health.

80–0470. U.S. Consumer Product Safety Commission. **Report on the Fire/Burn Problem in the United States. Volume I. Analysis. Volume II. Selected Data Tables.** U.S. Consumer Product Safety Commission, Bureau of Epidemiology, March 1978, Vol. I: 189 pp. Vol. II: 242 pp.

The fire/burn injury and death problem in the United States has been analyzed in this report using data from the National Household Fire Survey (1974), the National Health Interview Survey (1971-1974), the National Electronic Injury Surveillance System (1976), Mortality Statistics (collected by the National Center for Health Statistics, 1971-1975), and the Consumer Product Safety Commission Death Certificate Project (1974 and 1975). There were an estimated 5.6 million fire incidents in the United States during the period from April 1, 1973 to April 15, 1974. An estimated 4.5 million of these fires occurred in residences. The dollar loss attributable to residential fires during the period was estimated to be $1.8 billion. Appliances, in general, were specified as ignition sources in 3.4 million of the residential fires. When fires associated with consumer products other than appliances were considered, the most frequently mentioned ignition sources were smoking materials; an estimated 569,000 fires were caused by cigarettes, cigars, pipes, matches, and lighters. Accidental burn injuries were relatively rare and accounted for less than 3 percent of all injuries sustained by the civilian, noninstitutionalized population of the United States. Burn injuries frequently occurred in the home and were sustained primarily by lower income groups, males, young children, young adults, and the less educated. Accidental burn deaths were also relatively rare. They accounted for less than 10 percent of the total accidental deaths. The leading cause of accidental burn deaths was a fire in the home. Males, residents of the South, races other than white, and the very young and the elderly were the most likely victims of burn deaths. Product-involved accidental burn deaths were even less frequent in occurrence, yet they accounted for approximately 20 percent of the accidental product-involved deaths. The majority of product-involved burn deaths were fire related, with the cause of death being a fire in the home. Nonappliance ignition sources were the ignition sources most often resulting in death. (Auth. Abs. Mod.)

80–0471. U.S. Department of Health, Education, and Welfare. **Preventing Disease/Promoting Health. Objectives for the Nation.** U.S. Department of Health, Education, and Welfare, U.S. Government Printing Office, Document No. 1979-644-770, August 1979, 125 pp.

This publication presents a series of objectives which call for specific achievements during the 1980's that will result in measurable reductions in the rate of premature death, diseases, and disabling conditions that can be prevented. The objectives were developed by participants in the HEW sponsored conference held in Atlanta on June 13-14, 1979. Experts from around the country discussed the prospects for progress in 15 key areas, where there is evidence that appropriate risk-reducing actions can improve health. Topics discussed included health services (hypertension control, family planning, pregnancy and infant health, immunization, and sexually transmitted diseases), health protection (toxic agent control, occupational health and safety, injury control, fluoridation of water supplies, and surveillance and control of infectious diseases), and changes in lifestyle (smoking, alcohol and drug abuse, nutrition, physical fitness, and stress management). The following report outline was used for each topic: (1) the nature and extent of the problem (health implications, status and trends); (2) potential preventive measures and their relative strength; (3) specific objectives to be attained by 1990; (4) principal assumptions upon which the objectives are based; and (5) data sets necessary to track progress towards the objectives. Specific objectives for antismoking measures for 1990 include reducing the proportion of adult men and women who smoke from the current 33 percent to less than 25 percent; reducing the proportion of 12- to 18-year-olds who smoke from a current 12 percent to below 6 percent; establishing laws in all States to encourage nonsmoking in public, work, and eating places; improved health warning labeling for cigarette packages and advertisements; reducing the sales-weighted average tar content of cigarettes, currently 17 mg, to below 10 mg; and reducing the concentration of known carcinogens and carbon monoxide, hydrogen cyanide, nitrogen oxides, and acrolein gases in proportion to the reduction in tar content. Smoking research needs include definitions of smokers' preferences and needs to assist in cessation efforts; the most cost-effective cessation programs; the relatively long-term risks of lower tar-nicotine-gas cigarettes; the role of various social and marketing factors on smoking onset; and the most effective and easily disseminated methods of smoking prevention in youth, the most effective interveners, and the critical intervention age, as well as the optimal duration of the programs.

AGRICULTURE

80–0472. Boeger, M.; Drabek, J.; Pissiotas, G. **Method for the Cultivation of Plants Employing N-Alkyl-N-Phenylsulfenyl-N′-Phenyl-Formamidine Derivatives.** United States Patent No. 4,040,811, August 9, 1977, 6 pp.

This patent describes a method for controlling the growth of plants (e.g., the growth of suckers in tobacco) by applying N-alkyl-N-phenylsulfenyl-N′-phenyl-formamidine derivatives to the plant or the soil. Application to the plants is preferred and for this purpose the use of solutions or aqueous dispersions of the active compounds is the most appropriate. The actual amount of active substance applied will need to be adjusted in accordance with the variety of tobacco plant cultivated and the time of application, as well as in accordance with ambient circumstances such as prevailing weather conditions. The compounds are preferably applied at the rate of 0.1 to 10 kg of active substance per hectare.

80–0473. Cargill, R. L. **Tobacco Sucker Control.** United States Patent No. 4,095,971, June 20, 1978, 6 pp.

This patent describes a method for the chemical control and inhibition of primary and secondary axillary buds in tobacco plants by the systemic application of carbetamide to the plants. The herbicide is applied in liquid, solid, or semisolid compositions in amounts from 0.25 to 20 lbs of active ingredient per acre to the tobacco leaves and to the soil. Carbetamide is less toxic to mammalian species than the previously used common systemic sucker control agents and thus is safer for use by unskilled agricultural workers. Further, in the absence of significant toxicity, residues of the sucker control agents on the tobacco would provide a significantly lower health and environmental hazard to the processors and ultimate users of tobacco.

80–0474. Comber, R. **Treatment of Tobacco.** United States Patent No. 4,127,136, November 28, 1978, 4 pp.

This patent describes a method of hastening the process of curing and drying tobacco leaves by applying a wilt-accelerating chemical agent to mature growing tobacco plants. This allows the leaves to wilt over a period of 2 to 50 days and reduces the water content of the leaves by at least one-fourth, preferably by 40 to 90 percent, which corresponds to a 36 to 80 percent reduction in the total weight of the leaves. The leaves are then harvested and processed for use as smoking, chewing, or snuff products. The wilt-accelerating agent may be applied directly to the leaves by spraying, or to the roots or stalks of the plants. Suitable wilt-accelerating agents include both organic and inorganic substances, including sodium chlorate and bipyridylium compounds as well as acids and bases such as phosphoric, nitric, and perchloric acids.

80–0475. Cooke, A. R.; Starke, G. R. **Method for Inhibiting the Growth of Tobacco Suckers.** United States Patent No. 4,077,795, March 7, 1978, 15 pp.

This patent describes a method for controlling the growth of suckers in tobacco plants by spraying the plants with a solution containing a 2,6-dinitroaniline derivative alone or combined with a surfactant. These compounds are used at the rate of about 1 to 3 lbs active material per acre of tobacco in 50 to 200 gallons of water, preferably in formulation with a wetting agent, particularly a nonionic or an anionic agent. Such a wetting agent significantly enhances the tobacco sucker controlling activity of the dinitroaniline derivative compositions while possessing no such controlling activity by itself. The amount of surfactant used in the admixture is not critical, although ordinarily it should not be used in amounts greater than about 2 percent by volume of the final spray solution. Preferably, the surfactant will comprise 0.1-1 percent of the final spray volume.

80–0476. Fujikawa, K.; Takahashi, R.; Yokomichi, I.; Kimura, F.; Kaji, T.; Sakashita, N. **Method of Tobacco Sucker Inhibition.** United States Patent No. 4,029,490, June 14, 1977, 8 pp.

The growth of tobacco plant suckers is inhibited by the application of a benzylthiolcarbamate. This compound may be added to the stems and leaves at the topping stage at a concentration range of 1,000 to 5,000 ppm. Compounds of this group were found to have 100 percent sucker inhibition with small or no chemical injury to the plant.

80–0477. Kish, C. J. **Tobacco Sucker Control Agent and Method.** United States Patent No. 4,077,796, March 7, 1978, 6pp.

A method is described for controlling the growth of suckers on topped tobacco plants by a single application of an aqueous homogenized mixture of a saturated fatty alcohol and a salt of 1,2-dihydro-pyridazine-3,6-dione. The saturated fatty alcohols found to be useful in the mixture include those in the range C_6 to C_{18}, although C_8 (octanol) and C_{10} (decanol) alcohols are preferred. The solution contains from 5 to 25 percent by weight of the pyridazine salt and from 10 to 40 percent by weight of the saturated fatty alcohol.

80–0478. Meisinger, R. H. **Benzophenones and Benzhydrols.** United States Patent No. 4,120,687, October 17, 1978, 5 pp.

Sucker growth in tobacco plants can be controlled by applying a benzophenone or a benzhydrol to the growing plants. These compounds are applied to the plants during the topping stage, and are usually applied during the period from 1 to 7 days before to 1 to 7 days after topping. Typical rates of application are from 0.5 to 20 pounds per acre.

80–0479. Nishiyama, R.; Takahashi, R.; Sakashita, N. **Method for Controlling Growth of Tobacco Suckers.** United States Patent No. 3,989,502, November 2, 1976, 5 pp.

Tobacco suckers can be controlled by applying a composition containing as the active ingredient at least one phosphorothionoamidate at concentrations ranging from 1,000 to 7,000 ppm. The composition is applied from 2 weeks before to 2 weeks after the optimum topping period. In several experiments the percentage of sucker control was close to 100 percent, and there was little or no phytotoxicity.

80–0480. Wilcox, M. **N-Benzyl-2,6-Dinitro-3-Amino-4-Trifluoromethylanilines as Plant Growth Regulants.** United States Patent No. 4,116,667, September 26, 1978, 7 pp.

N-benzyl-2,6-dinitro-3-amino-4-trifluoromethylanilines are useful as herbicides and plant growth regulating agents (sucker control). In one experiment, when Hicks tobacco plants began to exhibit suckers, the plants were treated with (1) 50 ml of a 50 percent acetone 0.1 percent triton X-114 solution, (2) an octylphenoxypolyethoxyethanol, and (3) 750 ppm of N-(2′-methylbenzyl)-N-ethyl-2,6-dinitro-3-amino-4-trifluoromethylaniline (compound A) or N-(2′-chloro-6′-fluorobenzyl) -N- ethyl-2, 6-dinitro -3- amino -4- trifluoromethylaniline (compound B). Twenty-five days later, sucker size was rated. Thirty-three days later, the suckers were removed, counted, and weighed. The sucker size with compound A, compound B, or control was 1.8 (0 to 2.5 cm), 1.0 (0 to 2.5 cm), and 4.0 (>10.2 cm), respectively. The corresponding number of suckers (for five plants) and their total weight was 7, 74 gm; 0, 0 gm; and 14, 820 gm.

80–0481. Yu, P. K. **Diphenyl Ethers for Tobacco Sucker Control.** United States Patent No. 4,124,370, November 7, 1978, 6 pp.

Sucker growth in tobacco plants can be controlled by applying a diphenyl sulfide or a diphenyl ether to the growing plants. Compounds useful in inhibiting tobacco sucker growth have the formula $\Phi(Y)_n X\Phi(Z)_m$, where X is an oxygen or a sulfur atom; Y and Z are halogen, alkyl, trifluoromethyl, alkoxy, hydroxy, nitro, cyano, carboxy, carbalkoxy, carbamoyl, or alkylthio groups; and m and n are 0, 1, 2, or 3. These compounds are applied to the tobacco plants during the topping stage, and are usually applied during the period 1 to 3 days before to 1 to 3 days after topping, and preferably on the same day as topping. The compounds can be applied in any amount which will give the desired degree of control of sucker growth, without adversely affecting the growth of the tobacco plants. Typical applications are from 0.5 to 20 lbs per acre, and preferably 1 to 10 lbs per acre.

TOBACCO PROCESSING AND MANUFACTURING

80–0482. Aikman, L. N. **Tobacco Smoke Filter Unit.** United States Patent No. 4,038,994, August 2, 1977, 9 pp.

A cigarette holder is described which contains a filtering element in a cavity within the barrel of the holder. The filter element is provided with a flange that extends from the element and divides the cavity into an upstream filtering chamber and a downstream storage and filter chamber. Conformations in the flange permit communication from the upstream to the downstream side of the cavity. Smoke enters the filter cavity through a restricted opening where it is made to pass at high velocity. The smoke is then permitted to expand and cool so that the tars and nicotine in the smoke are condensed. The chamber and the filtering element are arranged so that the smoke swirls to impart a kinetic energy to the condensate which results in an inability to follow the conformations past the flange. This results in the condensate settling on the surfaces of the filter element where it is condensed. This device is useful as a withdrawal kit in that it can be used to reduce tar and nicotine intake with time and can also be used as a convenient, easily cleaned, and effective tar and nicotine filter. (Auth. Abs. Mod.)

80–0483. Almqvist, S.-O.; Enzell, C. R.; Hjern, L. O.; Artho, A.; Koch, R. **Filter for Tobacco Smoke.** United States Patent No. 4,033,362, July 5, 1977, 3 pp.

A new type of filter for tobacco smoke is described which is very effective for selective removal of low molecular, often noxious components in tobacco smoke, such as hydrogen, methane, hydrogen cyanide, carbon monoxide, acetaldehyde, and acrolein from the mainstream smoke. The filter is characterized by a diffusion portion, in which the smoke stream is separated from the outer air by means of a permeable membrane of fibrous or nonfibrous polymer material such as polyethylene, polypropylene or silicone rubber, which is permeable to low molecular smoke components. When cigarettes with and without the filter were tested, the following concentration reductions in the smoke of cigarettes with a filter were observed: nicotine 20 percent, tar 30 percent, acetaldehyde 66 percent, acrolein 66 percent, carbon monoxide 60 percent, acetone 60 percent, phenol 3 percent, 2,4-dimethylphenol 0 percent, and indolene 3 percent.

80-0484. Arledter, H. F.; Marek, J. **Method of Making a Laminated Tobacco Foil.** United States Patent No. 3,870,054, March 11, 1975, 8 pp.

A tobacco foil is made from at least two plies, each comprised of a film of predominantly reconstituted tobacco particles. An intermediate layer of cellulose, comminuted tobacco ribs, or porous inorganic materials is provided between the films. The intermediate layer may contain one or more active substances such as tobacco extract, burn control agents, ascorbic acid, magnesium perborate, odorants, or flavorants. The films are precipitated from an aqueous suspension, combined, and then dehydrated by pressing and thermal drying. Precipitation is carried out on a double sheet formerly composed of two screens which are approximately opposite one another in the sheet-forming region. This foil affects cigarette properties such as weight, drawing resistance, and number of draws, as well as content of moist and dry tar, nicotine, benzo(*a*)pyrene, and phenol. For instance, in smoke from filterless cigarettes with conventional vs. novel foils, nicotine and phenol contents were, respectively, 0.46 vs. 0.20 mg and 62.5 vs. 31γ/cigarette. (Auth. Abs. Mod.)

80-0485. Baker, J. C. **Cigarette Filter.** United States Patent No. 3,964,493, June 22, 1976, 16 pp.

A filtered cigarette having a forward portion, a strainer, and a filter is described. The filter is made of molded plastic with one or a number of surfaces whose interconnected channels help to provide long and twisting paths for the air-smoke mixture. The constant changing of directions in the x, y, and z coordinates creates a centrifugal force which propels the solids and liquids contained in the smoke outward, reducing the tar and nicotine levels in the smoke. The O-, H-, and T-shaped passages are particularly appropriate for this purpose. At the same time, the channels are relatively open and do not hinder the "draw" of the cigarette. In one embodiment, the passageway of the filter is around the periphery of a hollow cylindrical core, covered by paper. This passageway is comprised of numerous circumferential axially spaced grooves interconnected by extending connector channels. Islands or abutments at the discharge ends of certain air-smoke channels abruptly change the direction of flow of air-smoke mixture and separate it into distinct streams which are eventually recombined. The co-mingling and turbulence as the two streams are recombined may promote the reaction of carbon monoxide in the smoke with oxygen in the air mixture creating additional carbon dioxide and reducing carbon monoxide content. (Auth. Abs. Mod.)

80-0486. Baker, J. C. **Cigarette Filter.** United States Patent No. 3,958,579, May 25, 1976, 10 pp.

A filtered cigarette includes a forward portion, a strainer, and a filter made of molded plastic. The filter has one or a plurality of surfaces with interconnected channels which form long and complex air-smoke mixture paths. This filter is efficient at high velocities when centrifugal action compensates by depositing more liquids and solids and nicotine or alkaloids along filter walls. The large and unusually long wall area provides a trap for sulfur dioxides from matches and a trap for hydrocarbon gases from the burning of butane lighters and cigarette paper.

80-0487. Berger, R. M. **Tobacco Smoke Filter.** United States Patent No. 4,022,222, May 10, 1977, 7 pp.

Tobacco smoke filters are described which have an elongated hollow inner member and an intermediate member overwrapped with a conventional plug wrap. The intermediate member is preferably composed of a filtering material different from that of the inner member in order to provide selective filtration through the two layers. The inner and intermediate members are preferably formed of cellulose acetate tow with the inner tow fibers being finer than those of the intermediate tow; but polyethylene, polypropylene, or nonwoven staple fibers may also be used. The intermediate member functions to remove the larger particles of undesirable material so that the interstitial spaces of the inner member are not clogged. The intermediate member provides a smooth outer surface for conventional plug wrap attachment eliminating the need for an internal glue line. (Auth. Abs. Mod.)

80-0488. Berger, R. M. **Method and Apparatus for Making Tobacco Smoke Filter.** United States Patent No. 4,064,791, December 27, 1977, 8 pp.

Tobacco smoke filters having (1) an elongated annular intermediate member overwrapped with a conventional plug wrap and (2) discrete inner plugs whose lengths are shorter than the intermediate member and whose smoke passage resistance is at least that of the intermediate member are described. The inner plugs are secured within the intermediate member against axial displacement. The inner member may be composed of a filtration material, as is the intermediate member, or the inner member may be nonpermeable to smoke. Portions of the intermediate member may be crimped to embed them in the inner member. The inner member may be centrally positioned within the intermediate member to form cavities at both filter ends or only one inner cavity. The smooth outer surface of the intermediate member enables the attachment of a conventional plug wrap without the need for an internal glue line. An inner rod may be cut to form a multiplicity of inner plug members which are then axially spaced by injecting jets of air between adjacent members. Data from plugs with an inner member composed of either a cellulose acetate tow or a polystyrene nonpermeable to smoke indicate that the former plug results in a 2.4 inch pressure drop and a 69 percent retention, whereas the latter plug results in a 1.0 inch pressure drop and a 78 percent retention. (Auth. Abs. Mod.)

80-0489. Berger, R. M. **Tobacco Smoke Filter.** United States Patent No. 3,994,306, November 30, 1976, 7 pp.

Tobacco smoke filters having a composite cylindrical inner member formed from two semicylindrical inner elements and overwrapped with a common plug wrap are described. The composite inner member has a discrete inner cavity formed in each semicylindrical element and a composite cavity formed by a depressed portion in each semicylindrical element. These cavities may be filled with smoke-modifying material such as activated carbon bonded by polyethylene or silica gel particles. A smooth outer surface is provided on the composite inner member for attaching a conventional plug wrap without the need for an internal glue line. Separate cylindrical elements are reformed into semicylindrical elements and crimped to provide depressed pockets, following which the semicylindrical elements are mated and overwrapped to produce the final product. Filter efficiency ranges from 66.2 to 75.0 percent retention. (Auth. Abs. Mod.)

80-0490. Berger, R. M. **Method and Apparatus for Making Tobacco Smoke Filter.** United States Patent No. 4,075,936, February 28, 1978, 12 pp.

Tobacco smoke filters and methods for manufacturing them are described. The filters have both internal and external cavities, with the external cavities providing a smooth outer surface for attaching conventional plug wrap without the need for an internal glue line. The external cavities comprise from 5 to 50 percent of the total outer surface of the inner filter member, and preferably 20-30 percent. This enables the inner filter member to provide uniform support to the overwrap. The outer cavities may be in the form of elongated, discrete grooves, a knurled-like pattern, or a continuous helical groove. The resultant filter has high filtration (70.8-71.8 percent retention) and correspondingly low pressure drop characteristics (2.7-2.8 inches). The outer grooves are formed by various mechanical ironing or grooving techniques, by crimping, or by utilizing jets of steam. (Auth. Abs. Mod.)

80-0491. Berger, R. M. **Tobacco Smoke Filter.** United States Patent No. 4,026,306, May 31, 1977, 7 pp.

A tobacco smoke filter is described which has an elongated annular intermediate member overwrapped with a conventional plug wrap and discrete inner plugs secured against axial displacement within the intermediate member. The inner member may be composed of either a filtration material or a nonpermeable material. The intermediate member is composed of a filtration material. Portions of the intermediate member may be crimped to embed them into the inner member. The inner member may be centrally positioned within the intermediate member to form cavities at each end of the filter or secured at one end of the intermediate member to provide only a single inner cavity. The smooth outer surface of the intermediate member enables the attachment of a conventional plug wrap without the need for an internal glue line. Means are described for cutting an inner rod to form a multiplicity of inner plug members, and then axially spacing the inner members from each other by injecting jets of air between adjacent inner members. (Auth. Abs. Mod.)

80-0492. Berger, R. M. **Tobacco Smoke Filter.** United States Patent No. 4,022,221, May 10, 1977, 11 pp.

Tobacco smoke filters are described. They have both internal and external cavities with the external cavities providing a relatively smooth outer surface for attaching a conventional plug wrap without the need for an internal glue line. The external cavities comprise from 5 to no more than 50 percent of the total outer surface of the inner filter member, preferably between 20 and 30 percent. This enables the inner filter member to provide substantially uniform support to the overwrap. The outer cavities may be in the form of elongated, discrete grooves, a knurled-like pattern, or a continuous, helical groove. The resultant filter has high filtration and correspondingly low pressure drop characteristics, and may be rapidly manufactured. (Auth. Abs. Mod.)

80-0493. Brackmann, W. A.; Di Ianni, D. **Filter for Tobacco Smoke.** United States Patent No. 4,059,121, November 22, 1977, 7 pp.

A tobacco smoke filter is described which is composed of a single filter element consisting wholly of numerous randomly oriented, noncrimped, smooth-surfaced solid fibers of thermoplastic polymeric nonabsorbent material. Each of the fibers has a uniform diameter less than 5 microns. The filter element has a generally circular cross-section taken across the intended flow path of the tobacco smoke and a diameter substantially equal to the diameter of a tobacco smoke flow path. The weight of polymeric material exceeds 6 mg, and the filter element has a pressure drop from 1 to 10 inches of water at a flow rate of 17.5 ml/sec. The quantity of tar and nicotine removed from cigarette smoke by an Acrylite filter is about 30 percent greater than that removed by a cellulose acetate filter at the same total pressure drop across the cigarette. (Auth. Abs. Mod.)

80-0494. Cohn, C. C. **Cigarettes.** United States Patent No. 4,044,778, August 30, 1977, 7 pp.

This patent describes a cigarette having a wrapper coated with an aqueous alkali silicate solution which will cause the cigarette to extinguish itself when coming in contact with a flammable or other surface. The silicate solution contains from about 17 to 27 percent by weight of silicon dioxide. The coating area of the silicate solution on the wrapper is from 40 to 84 percent of the entire outside wrapper area. Various additives such as acids, carbohydrates, colloids, humectants, fire retardant compounds, and alumina gel compounds may be added to the silica solution to improve the quality of the coating.

80–0495. Crellin, R. A.; Jenkins, C. R.; Phelpstead, J.W.P. **Tobacco-Smoke Filters.** United States Patent No. 4,062,368, December 13, 1977, 4 pp.

A material was developed for filtering tobacco smoke by reducing vapor phase constituents without adversely affecting the taste of the smoke. The filtering material comprises carbon particles within the range of 300 to 1700 μ which are coated over their surfaces, individually and without being bonded together, with a barrier layer. The coating layer has a thickness within the range of 5 x 10^{-4} to 0.5 μ and is discontinuous in that it has a porosity within the range of 7,000 to 200,000 cm^3/min/10 cm^2 per 10 cm water gauge and a permeability for organic vapors contained in tobacco smoke such that it permits the passage of molecules within the size range 5 x 10^{-4} to 2 μ. The coating layer is composed of an organic nonnitrogen-containing polymeric material which is nonvolatile, substantially nonwater soluble and nontoxic. The efficiency of these filters, measured by the reduction of total volatile aldehydes in cigarette smoke, ranged from 45 to 69 percent, depending on the coating level. (Auth. Abs. Mod.)

80–0496. Fair, G. D.; Hasmall, D.L.A.; Ryan, W. P.; Sellman, E. J. **Method of Preparing Tobacco Smoke Filter.** United States Patent No. 3,966,912, June 29, 1976, 6 pp.

A tobacco smoke filter element for use in cigarettes, cigarette holders, cigar holders, or pipes is described. The filter elements are prepared from a bonded nonwoven fabric composed wholly or partly of wool fibers and should contain as high a proportion of wool fibers as is economically feasible. In the case of a filter for a cigarette, the filter is made by converting a web of bonded nonwoven fabric to a filter rod of substantially circular cross-section. Filter elements prepared from wool showed a higher filtration efficiency for tar and nicotine than cellulose acetate filters (38.3 vs. 33.6 percent for tar and 38.6 vs. 31.5 percent for nicotine, respectively). (Auth. Abs. Mod.)

80–0497. Fiore, J. V.; Kelly, T. K.; Nellen, W. J. **The Method of Making a Reconstituted Tobacco Foamed Sheet.** United States Patent No. 3,872,871, March 25, 1975, 9 pp.

Cigar tobacco sheet made from novel cigar tobacco foams is described. The foam is characterized by fine bubbles (i.e., at least 80 percent of the bubbles are less than 100 μ) and low density (0.85-1.0 g/cc). The cigar foam is formed in a high shear system which utilizes tobacco particles generally smaller than 100-120 mesh. The foam is cast and dried to form reconstituted tobacco sheet which is utilized as cigar wrapper, binder, and/or filler. This cigar tobacco sheet is characterized by a density of 0.28 to 0.45 g/cc, a regular grainy texture, a lighter color, and an increased opacity. On combustion, cigars constructed with foamed wrapper and binder deliver 20-25 percent less tar than a natural leaf wrapper/conventional reconstituted binder cigar, at an equivalent weight. Further, the organoleptic properties of the reconstituted cigar tobacco sheet are acceptable at 100 percent substitution. (Auth. Abs. Mod.)

80–0498. Fiore, J. V.; Slanski, J. M. **Novel Reconstituted Tobacco Sheets and Process.** United States Patent No. 4,176,668, December 4, 1979, 9 pp.

This patent describes a process for monitoring hydrophobic coatings as weight per unit area by spectrophotometric measurement of coating metal content. Coating metal levels are at least three and preferably four to six times the natural level found in tobacco. In one study, a zinc-containing coating solution was prepared using zinc chloride and ethyl cellulose in isopropyl alcohol. The calculated ratio of zinc to ethyl cellulose was 1:66 parts by weight. The zinc-coated sheets were then used as a wrapper for perfecto and cigarillo shaped cigars. In cigarillos, the percentage of zinc transferred from whole cigar to smoke was 1.7, 2.4, and 1.3 for control, 1X zinc, and 10X zinc, respectively. In perfectos, corresponding percentages were 2.1, 2.6, and 1.3.

80–0499. Frazier, T. C.; Ashworth, J. T.; Chumney, R. D., Jr.; Meyre, R. W. **Smoking Article and Method.** United States Patent No. 4,094,323, June 13, 1978, 6 pp.

A method for expanding and fiberizing tobacco stem and stalk materials which have increased filling capacity is described. The stem material is conditioned by (1) adjusting the moisture content to at least 10-50 percent by weight while maintaining the temperature at 115-170°C and the pressure at 10-100 psig for a 0.1- 5-min period and (2) mechanically fiberizing the material while it is under a 10-100 psig pressure between fiberizing surfaces 0.05-0.3 inches apart. Cigarettes prepared with fiberized, expanded stems, conventional rolled cut stems, and no stem byproducts (controls) were analyzed for various physical and chemical properties. The nicotine concentrations (mg/puff) were 0.11, 0.12, and 0.19, respectively. The corresponding values for tar were 1.8, 2.2, and 2.9.

80–0500. Frederickson, J. D.; Hickman, E. S.; Harper, E. D. **Tobacco Expansion Process.** United States Patent No. 3,882,874, May 13, 1975, 3 pp.

Tobacco is treated with an organic compound, such as dicarboxylic acid or its derivative, which is capable of thermal decomposition to form a gas. The tobacco is then heated to decompose the compound and produce the gas within the tobacco and thereby expand it. At the time of gas release the tobacco should have a sufficiently high moisture content (in excess of 10 percent by weight) to make the structure of the tobacco sufficiently pliable for expansion. (Auth. Abs. Mod.)

80–0501. Goavec, E. **Method for the Production of Filter Structure for Cigarette Filters.** United States Patent No. 4,125,061, November 14, 1978, 16 pp.

A method for assembling a highly efficient cigarette filter by making a new type of filter structure using a sheet of fiber shaped as a cylindrical casing is described. The sheet of fiber material is transversally corrugated and acts as a tobacco smoke product absorbent. On certain portions of this sheet a wafer relief configuration is impressed in order to obtain two different materials. The sheet is then cut into ribbons of the same width, gathered in the form of a pile, and geometrically shaped in a homogeneous cylindrical casing. (Auth. Abs. Mod.)

80-0502. Godfrey, F. D., Jr. **Hydrophobic Coating for Tobacco Sheet Material.** United States Patent No. 4,140,135, Feburary 20, 1979, 7 pp.

A method for applying a hydrophobic film onto at least one surface of a tobacco product is described. The coating solution consists of cellulose propionate with an acetyl content of less than 6 percent by weight, a hydroxyl content of at least 3 percent by weight, and an acyl group (with more than 3 carbon atoms) content of less than 10 percent by weight. The cosolvents are alcohol and water. The process for coating a reconstituted tobacco product is comprised of shaping a suspension of particulate tobacco and a suitable binder into a desired shape, heating the shaped product to remove the suspension liquid, and applying the coating solution to the uncooled, dried, reconstituted tobacco product. The coating provides an appearance, taste, aroma, and feel similar to that of a natural tobacco leaf before and during burning.

80-0503. Grossman, H. **Body of Tobacco With a Deposit of Particles Added Thereto.** United States Patent No. 4,084,596, April 18, 1978, 4 pp.

One of the serious problems in the filtration of tobacco smoke is the hydrophobic character of the carcinogenic tars, which are oleophilic. A wet filter will not adsorb the particulates. In this patent, a filter for tobacco smoke which acts to attract the negatively charged particles dispersed in tobacco smoke is described. A suitable porous substrate has deposited thereon particles of a positively charged, water-insoluble, hydrophobic, oleophilic substance which acts to attract and neutralize the negatively charged particles dispersed in the tobacco smoke so that the filter will hold on to the neutralized particles and prevent such particles from passing with the gases through the filter to the smoker's mouth. Based upon tests and observations of these filters, the deposit on the porous substrate of the positively charged, water-insoluble, hydrophobic, oleophilic substance acts as a coalescing agent upon the colloidally dispersed negative particles in the smoke to enlarge them and to render them more easily filterable. Materials which have been found to be particularly suitable as coalescing agents are colloidal alumina and colloidal silica having a positive charge. A surface-active cationic or nonionic wetting agent may be added to further enhance the ability of the positively charged material to take on moisture which comes in contact with it to produce a synergistic effect. An additional hydrophilic absorber for the alkaloids, e.g., magnesium trisilicate, can be added to the mixture containing the coalescing agent. (Auth. Abs. Mod.)

80-0504. Grossman, H. **Filter and Cigarette Including a Filter.** United States Patent No. 4,126,141, November 21, 1978, 5 pp.

This patent describes a filter for tobacco smoke consisting of a porous cellulosic material to which a water-insoluble, hydrophobic, moisture-laden substance is added. When activated by tobacco smoke, the substance releases moisture. One such substance is a colloidal methylated silica-water system with particles of methylated silica deposited around a mist of dispersed water. The presence of moisture provides a cool, comparatively smooth smoke. The filter may be in the form of a cartridge for a cigarette or cigar holder, or as a filter tip associated or made integral with a body of shredded tobacco.

80-0505. Grossman, H. **Cigarette and Filter for Tobacco Smoke.** United States Patent No. 4,037,607, July 26, 1977, 4 pp.

A filter for tobacco smoke is comprised of a porous substrate which has a deposit of particles of a positively charged, water-insoluble, hydrophobic, oleophilic coalescing agent (colloidal alumina or silica) which acts to attract and neutralize the negatively charged particles which are dispersed in tobacco smoke. In order to substantially increase the effectiveness of the filter, the tobacco with which the filter is associated is treated with hydrophobic, colloidal, negatively charged methylated silica so that during pyrolysis the resulting smoke will contain more negatively charged particulates. These colloidally dispersed negative particles become clumps of increased mass and size, improving filter efficiency.

80-0506. Grossman, H. **Filter and Cigarette Including a Filter.** United States Patent No. 4,076,031, February 28, 1978, 5 pp.

The filter may be in the form of a cartridge for a cigarette or cigar holder, or as a filter tip for divided or shredded tobacco. The filter contains a colloidal methylated silica-water system with particles of methylated silica deposited around a mist of dispersed water. The silica is carried by a nontobacco porous substrate such as cellulose acetate tow, cotton fibers, or mixtures of these. When used in a cigarette filter, the cigarette tobacco also has a deposit of the silica-water system on it. The tobacco pyrolysis temperature activates the system to release moisture and to furnish a cool smoke. Any condensation of the tars and nicotines caused by the release of the moisture and the consequent cooling is enhanced by the inclusion of the silica-water system in the tobacco. Further, by selecting a substance which negatively polarizes the particles in the smoke, the tars and nicotines carried in the smoke stream are substantially eliminated by inclusion of a fiber or material which has a positive zeta potential at the pH of tobacco smoke. (Auth. Abs. Mod.)

80–0507. Grossman, H. **Cigarette and Filter.** United States Patent No. 4,059,119, November 22, 1977, 5 pp.

A tobacco smoke filter employs synthetic, inorganic, water-insoluble, anhydrous fibers having a positive zeta potential at the pH of tobacco smoke. To increase the filter's effectiveness, the tobacco associated with the filter has particles of a water-insoluble, hydrophobic, moisture-laden, negative charge-imparting substance (e.g., alumina, alumina-silica, or zirconia fibers) mixed with and deposited on it. (Auth. Abs. Mod.)

80–0508. Hernandez, A. C.; Prohoroff, J. P. **Filtering Apparatus for Cigarette Smokers.** United States Patent No. 4,049,005, September 20, 1977, 7 pp.

A cigarette holder with a filter is described which utilizes both condensation and momentum to remove unwanted components of the smoke with minimal changes in suction pressure and taste. An ambient air inlet opening is included at a point downstream from the point at which the smoke is accelerated to increase the momentum of its solid and liquid materials. The smoke is accelerated and then introduced into an expansion chamber where ambient air is drawn in not only to cool and condense tar and nicotine products, but also to cause a flow turbulence that will insure that the condensed products collide with one another and with the surface of the filter element. The condensed materials adhere to the latter and are effectively removed from the smoke. The apparatus is suitable both as a filter unit for persons who want to continue smoking and as an element of a smoker's withdrawal kit.

80–0509. Hind, J. D.; Hopkins, W. C. **Smoking Article.** United States Patent No. 4,129,134, December 12, 1978, 6 pp.

A new wrapper for cigarettes and little cigars and a method for producing it is described. The wrapper is a film made of a natural polysaccharide, preferably in combination with an alkaline earth metal component. In appearance and other physical characteristics the wrapper is distinctly different from conventional wrappers in a number of ways: (1) it acts to compress the burning or smoldering tobacco, reducing the availability of new fuel to the tobacco in the interval between puffs; (2) it possesses the ability to shrink at the char line of the smoking product during the pyrolysis of the tobacco and greatly diminishes the production of sidestream smoke; (3) it makes possible a smoking article which produces less smoke when it is not being puffed, for example, when it is left in an ashtray; and (4) cigarettes and cigars with this wrapper were found to have improved aroma and flavor.

80–0510. Hoelzel, C. B.; Rainer, N. B.; Wilson, P. A. **Smoking Article and Process for Filtering Tobacco Smoke Employing a Cross-Linked Organic Oil Filter Material.** United States Patent No. 3,977,417, August 31, 1976, 6 pp.

This patent describes a filtering material and the process of filtering tobacco smoke by passing it through a particulate, solidified cross-linked organic oil. An inorganic or organic powder may be used with the oil in order to improve bulk handling characteristics and the filtering quality of the material. When cigarettes were smoked with filters prepared from oils alone, the filter efficiency (measured in percent total particulate matter removal) ranged from 48 to 51 percent; in combination with various powders, filter efficiency increased in all instances (up to as high as 74 percent), depending on the particular powder.

80–0511. Holst, A.; Kostrzewa, M.; Lask, H. **Cigarette Filter.** United States Patent No. 4,015,611, April 5, 1977, 4 pp.

A cellulosic cigarette filter is composed at least partially of (1) cellulose modified with an organic isocyanate or higher molecular weight fatty acid groups, or (2) a naturally water soluble cellulose ether which becomes largely or completely water insoluble by cross-linking or other means, yet remains capable of adsorbing water. As compared with the smoke tar and nicotine contents of unfiltered cigarettes, nicotine can be reduced from 50 to 72 percent and the tar content can be reduced from 45 to 77 percent.

80–0512. Horsewell, H. G.; Crellin, R. A. **Cigarettes.** United States Patent No. 4,088,142, May 9, 1978, 3 pp.

This patent describes a wrapped cigarette and a method for wrapper production. A very highly porous wrapping paper, with air porosity within the range from 2,450 to 20,000 $cm^3min^{-1}10cm^{-2}10cmWG^{-1}$ is produced by perforation from a base paper having a tensile breaking strength of not less than 85 g/mm width of paper. The mean number of perforations is within the range of 10 to 150 per cm^2 of the paper and the mean diameter is between 30 and 200 μ. A filter cigarette wrapped in this paper may result in a nicotine filtration efficiency within the range of 15 to 30 percent. In one experiment a blend of flue-cured tobacco was wrapped with this porous paper. When smoked under standard conditions of 1 puff per minute of 35 cm^3 volume and 2-sec duration, this cigarette delivered 13.7 mg total particulate matter (TPM), 1.0 mg nicotine, and 4.0 mg carbon monoxide (CO). The ratio of CO to nicotine was 4.0. A comparable, normally wrapped cigarette with nonperforated paper delivered 43.0 mg TPM, 2.36 mg nicotine, and 23.3 mg CO, the ratio of CO to nicotine being 9.9.

80–0513. Horsewell, H. G.; Harris, I. R. **Tobacco-Smoke Filters.** United States Patent No. 4,033,361, July 5, 1977, 5 pp.

This filter contains a macroporous amine-type anion-exchange resin as an adsorbent for volatile smoke constituents, particularly aliphatic aldehydes. The resin contains a cross-linked polyvinyl aromatic compound such as polystyrene cross-linked with divinyl benzene. The resin is used in particulate form between fibrous, filamentary, or paper, filter sections or dispersed in tow. The resin may be half-neutralized with acetic acid or mixed with solid acid. The resin moisture content is between 5 and 40 percent. In an experiment with cigarette filters containing 150 mg of free-base resin vs. 150 mg of half-neutralized resin, the aldehyde filtration efficiency was calculated to be 60 vs. 79 percent.

80–0514. Ihrig, A. M.; Williams, D. L. **Article for the Selective Removal of Hydrogen Cyanide From Tobacco Smoke.** United States Patent No. 4,091,822, May 30, 1978, 7 pp.

An improved filter for the selective removal of hydrogen cyanide (HCN) from tobacco smoke is described. The selective adsorbant is a complex formed from a polydentate amine and a metal ion selected from the group consisting of zinc, iron (II), copper, and nickel. The complex is made more effective by combining it with a polyalkylene oxide which acts as a synergist. In two experiments with cigarette filters containing the zinc complex and polyethylene glycol, the percent removal of HCN was 83 and 97 percent, as compared with controls. A nickel-containing cigarette filter resulted in 68 percent removal of HCN.

80–0515. Joyce, P. J. **Non Fibrous Cigarette Filter.** United States Patent No. 4,034,768, July 12, 1977, 3 pp.

A cigarette filter comprised of a porous sponge of nonwoven particles of a 2-pyrrolidone polymer is described. This filter screens out the carbon monoxide and carbon dioxide gases generated by incomplete combustion of the cigarette paper and tobacco. These gases function as catalyst activators extending the prepolymer to a larger polymer which eventually hardens. The molecular weight ranges from 8,500 to 500,000 or greater.

80–0516. Kaswan, J. **Tobacco Product.** United States Patent No. 4,027,679, June 7, 1977, 6 pp.

A smoke vector for a tobacco product, (e.g., cigarettes) for modifying or altering the mainstream smoke at or near the combustion zone or burning zone is described. The smoke vector comprises a thin-walled elongated, impervious tube-like member or vector which is hollow and empty. The vector extends longitudinally through the cigarette and terminates with an impervious mouth end. During an indrawn breath by the smoker, the smoke comprising the products of the combustion of tobacco in systemic heat is below the formation temperatures of temperature dependent hazardous chemicals. (Auth. Abs. Mod.)

80–0517. Lee, C. B.; Lim, B. C. **Filter for Cigarettes, Cigars and the Like.** United States Patent No. 4,120,310, October 17, 1978, 6 pp.

A cup-like and an H-shaped filter for cigars and cigarettes are described. The cup-like filter deflects the passage of smoke through a smoke pooling chamber, and has perforations and a side corridor space for removal of smoke constituents such as tar and nicotine. The H-shaped filter is a duplicate of the cup-like filter with the bottom portions affixed so as to be double-faced. The H-shaped filter deflects the passage of smoke through a front smoke chamber, and has perforations, a side chamber, perforations again, and finally a rear smoke chamber. Being double-faced, the H-shaped filter can be incorporated into cigarettes in either direction. The filters can be used in cigars and cigarettes singly or in combination with the conventional fiber filter, or as disposable filter cartridges for cigarette holders and tobacco pipes. (Auth. Abs. Mod.)

80–0518. Luke, J. A. **Porous Wraps for Smoking Articles.** United States Patent No. 4,104,431, August 1, 1978, 5 pp.

A method and apparatus for treating porous wrapping material for a smoking article such as a cigarette filter is described. The porous material is provided with an impervious region or regions positioned to coincide with a region or regions at which adhesive is to be applied by coating with a film-forming substance which has dried or set before application of the adhesive. The film-forming substance seals off pores in the porous wrapping material and prevents penetration of the adhesive. Suitable film-forming substances may be selected from heat-drying or air-drying types. Substances suitable for heat-drying are synthetic polymers such as polyvinyl alcohol or ethylene vinyl acetate; cellulose derivatives such as ethyl-, methyl-, and carboxy methyl cellulose; acrylates; and natural gums such as gelatines, alginates, dextrins, and starch. Suitable substances which can be air-dried are extruded thermoplastics such as polyethylene and polypropylene. (Auth. Abs. Mod.)

80–0519. McGeady, J. C.; Lewis, C. I. **Stiffening of Tobacco With Dialdehyde.** United States Patent No. 4,072,155, February 7, 1978, 5 pp.

Tobacco is treated with a dialdehyde in the presence of a suitable catalyst to increase its stiffness and filling capacity. The catalyst is selected from a group consisting of monobasic ammonium phosphate, sulfuric acid, phosphoric acid, hydrochloric acid, ammonium carbonate, ammonium bicarbonate, and ammonia. The reaction of the aldehyde with the tobacco may take place at room temperature (25°C), but can be accelerated by heating at 120-130°C for a period of 6 to 12 min.

80–0520. Miyake, T. **Shaped Matters of Tobaccos and Process for Producing the Same.** United States Patent No. 4,018,233, April 19, 1977, 5 pp.

A shaped matter of tobacco excellent in aroma and flavor is obtained by coating and binding starting tobacco materials with pullulan or a pullulan derivative molecular weight 10,000 to 5,000,000. The starting tobacco materials are leaf wastes, shred wastes, midribs, roots or stems of tobaccos, fragments, powders, leaf tobaccos, shredded tobaccos, or calluses or tissues of tobaccos. The ratio of starting tobacco materials to the coating and binding material (etherified or esterified pullulan) is 100:0.1 to 100:500 by weight. The coating and binding material is used in combination with at least one member selected from groups consisting of other binders, moisture-controlling agents, reinforcing agents, and flavors. (Auth. Abs. Mod.)

80–0521. Norman, V. **Cigarette Mouthpiece for Controlling Flow.** United States Patent No. 4,023,576, May 17, 1977, 6 pp.

A cigarette mouthpiece is described which has a one-piece construction in which two semicylindrical shells are hinged together along one edge and are releaseably secured together at the free edges. The mouthpiece has an internal cylindrical smoke chamber which is separated from a tobacco column at one end by a baffle means, while the opposite end of the smoke chamber is defined by a pair of abutting walls which are slotted to define an orifice. The orifice diameter is less than the diameter of the smoke passage in order to control the amount of smoke flowing out of the smoke passage for a given draw. The exterior surface of the mouthpiece has longitudinal grooves which cooperate with an overlying perforated tipping paper to define flow paths for ventilation air. For a given draw, an amount of undiluted unfiltered smoke is drawn from the tobacco column into the smoke chamber and through the outlet orifice. At the same time, a quantity of ventilation air is drawn in through the tipping paper and the longitudinal grooves to envelop the undiluted smoke within a smoker's mouth. (Auth. Abs.)

80–0522. Norman, V.; Moogalian, C. M. **Cigarette Filter.** United States Patent No. 4,109,666, August 29, 1978, 8 pp.

A cigarette filter is described which allows a portion of substantially unfiltered but dispersed smoke to enter a smoker's mouth at high velocity while diluting the smoke with drawn in air in the mouth. The draw resistance through the tipping paper perforations and the filter complements the resistance of the smoke through the tube and diffuser. The filter is comprised of an axially aligned tube extending from the cigarette tobacco section, a layer of filter material positioned circumferentially around this tube, and a diffuser at one end of the tube for dispersal of the smoke before it enters the smoker's mouth. (Auth. Abs. Mod.)

80–0523. Ogasa, K.; Ono, J.; Kudo, T.; Doi, K.; Noguchi, K.; Maeda, K. **Granular Composition for Tobacco Filter.** United States Patent No. 4,038,992, August 2, 1977, 8 pp.

A granular composition suitable for use in tobacco filters and capable of selectively removing harmful substances from tobacco smoke is described. The granules have a size of 10 to 50 mesh and a bulk density of 0.5 to 0.7 g/cm^3 and are composed of protein powders of bovine milk whey and/or egg white, and one or more excipients selected from the group consisting of powdered cellulose, wheat flour, starch, rice flour, sugar, glucose, lactose, talc, alumina, zeolite, or silica gel; or composite granules which are a blend of said granules and charcoal granules. The capacity of a conventional triple filter containing protein granules to remove benzo(*a*)pyrene is fivefold higher than it is for the equivalent acetylcellulose monolayer filter. The rate of removal of volatile phenol was significantly elevated when the protein content in granules was higher than 5 percent. However, the rate of removal of tar was lowered when the protein content exceeded 70 percent. The protein content in the protein granule should, therefore, be higher than 5 percent but lower than 70 percent in order to achieve the satisfactory removal of all of these harmful substances from tobacco smoke. The protein granules possess the unique property that they exhibit a dual affinity to both the polar (phenol) and nonpolar (3,4-benzpyrene) components in tobacco smoke, while acetylcellulose and charcoal preferentially associate with only one of these alternatives.

80–0524. Owens, W. F., Jr. **Air Flow Limiting Filter.** United States Patent No. 4,119,105, October 10, 1978, 4 pp.

Various types of filters have been designed for removing or reducing undesirable constituents in the smoke from smoking articles. Such filters mostly take the form of a mass of fibers, paper, or other porous and semiporous materials through which the smoke must pass. Other types of smoke filtering devices have been proposed which utilize valves or various designs which tend to open upon a predetermined amount of suction being applied by the smoker when drawing on the smoking article. Typical devices of this nature are disclosed in U.S. Pat. Nos. 2,709,441; 3,533,414; 3,616,802; and 3,685,522. However, filter valves of this type have the disadvantage of increasing the amount of smoke drawn through the smoking article as the pressure or suction is increased by the draw in any individual puff, thereby opening the valve and increasing the amount of smoke and attendant constituents passing through the valve to the smoker. Cigarette filter devices have also been proposed that contain valves which are designed to be closed by the hot smoke from the approaching coal as the cigarette is smoked and thereby reduce the amount of smoke that can be drawn through the cigarette. Examples of this type of filter valve are disclosed in U.S. Pat. No. 3,800,805. However, such valves have the disadvantage that they are responsive to the heat from the smoke and only operate to close when the temperature is high enough or the burning coal is close enough to activate the valve. In this patent, a smoke control device is provided which contains a valve means responsive to pressure drop in any puff. When the puff draw rate exceeds a predetermined level the valve closes, limiting and controlling the amount of smoke that can be inhaled in each puff. The structure is designed so that only a normal puff can be taken for the valve to remain open, and when a greater rate is attempted by the smoker the valve automatically closes. After each puff, the valve returns to its open position and remains open unless an excessive pressure drop occurs in the next puff whereby it closes again. The delivery of tar and nicotine in each puff is limited to the delivery that would normally be obtained from the smoking article such as a low tar/nicotine cigarette and cannot be increased by the smoker by increasing the draw rate in any single puff. (Auth. Abs. Mod.)

80–0525. Owens, W. F., Jr. **Flavored Cigarette.** United States Patent No. 4,082,098, April 4, 1978, 5 pp.

A ventilated cigarette whose function is to introduce volatile flavoring agents such as menthol into the mainstream smoke of the cigarette, to improve the delivery rate, and to prolong the shelf life of the flavoring agent is described. The cigarette has a porous or perforated air permeable wrap encircling the filter plug which is impregnated with the volatile flavoring agent. The flavorant is contained in a perforated or porous air permeable tipping envelope. As the cigarette is smoked, air is drawn through the ventilation area of the tipping, through the flavor-impregnated filter plug wrap, and into the filter where the volatile flavoring agent mixes with the smoke. Since the flavor-impregnated plug wrap is tightly enclosed between the tipping envelope and the filter plug, loss of the volatile flavoring agent is minimized, enabling longer shelf life without reduction in flavor potency. High delivery rates are achieved using minimum amounts of flavoring agent. (Auth. Abs. Mod.)

80–0526. Parker, F. J.; Sen, D. **Process for Producing Cigarette Filters.** United States Patent No. 4,054,550, October 18, 1977, 6 pp.

An improved process for producing cigarette filters is described. A foamable mixture is comprised of a thermoplastic polypropylene resin, a volatile blowing agent, a nucleating agent, and a synergist. The mixture is extruded through a die maintained at 155-165°C, while the resin temperature is maintained at 160-180°C within 5 mm of the die to form a strand of open cell foam. The strand is first sized to the desired shape, then cut into individual filter elements. The filter has a tar particulate matter efficiency (as measured on a 10 mm filter) greater than 12 percent. The nucleating agent is a particulate compound of a Group IIA, IIIA, or IVA metal and is used in proportions ranging from 0.1 to 40 percent based on resin weight. The synergist is an organic compound containing both a hydrophobic and a hydrophilic group, where the hydrophilic group is ionic and the hydrophobic group is a hydrocarbon or a hydroxyl- or halogen-substituted hydrocarbon. The synergist is present from 0.01 to 5.0 percent based on the weight of the nucleating agent. An effective combination uses calcium carbonate as the nucleating agent and potassium benzoate as the synergist.

80–0527. Pasche, R. C. **Cigarette Filter Tip.** United States Patent No. 4,015,610, April 5, 1977, 4 pp.

A cigarette filter tip is described which consists of three filter elements surrounded by a transparent outer coating. The elements at each end of the filter tip are composed of fibrous material while the central element is granular and contains inorganic iron sulfate. The latter changes color upon reaction with hydrogen sulfide in the cigarette smoke and forms iron sulfide indicating the presence of hydrogen sulfide in the smoke and its removal by the filter. A paper band which unites the filter with the cigarette extends around the fibrous elements but not the granular filter element leaving the color change visible to the smoker, regardless of how the cigarette is turned.

80–0528. Perkins, P. R.; Bale, C. R. **Smoking Product.** United States Patent No. 4,142,535, March 6, 1979, 4 pp.

Increased filling power of tobacco has been achieved only by physical or mechanical methods until now. However, it is possible to increase filling power over that of conventional flue-cured blends by use of a smoking product composed of tobacco and nontobacco additives. The additives include a pectin having a degree of methylation between 30 and 75 percent and an inherent viscosity between 2 and 6.5 dl/g as a filmforming or binding agent. The proportions of tobacco and additives by weight are 5-60 percent and 40-95 percent, respectively.

80–0529. Rainer, N. B.; Wilson, P. A. **Microporous Styrene Polymers and Method of Making Same.** United States Patent No. 4,002,177, January 11, 1977, 4 pp.

A method for preparing microporous styrene polymer filters is described. Polystyrene latex or a styrene copolymer latex containing monomers such as butadiene, acrylic acid, 2-(aminoethyl) methacrylate, styrene sulfonic acid, acrylonitrile, N-vinyl-pyridine, and N-vinylpyrrolidone, is mixed with a water-soluble ether. The resulting mixture is cast, molded, or impregnated into a fibrous material or other porous structure such as an open-celled foam, which is then heated at 80-120°C for 1 min to 1 hour. An open-celled structure with ether-filled pores is then formed. The ether is readily removed by conventional extraction techniques. The resulting microporous styrene polymer is a randomly interconnected open network of irregularly shaped pores whose walls are defined by cohered spherical particles. The average pore size is less than 20 μ and the pore volume is at least 1 cm^3/g. The polymer has the form of ciliated granules and has a mesh size of 40 to 80. This filter is capable of removing 65 percent of the total particulate material in tobacco smoke which passes through at a draw resistance of approximately 5 inches of water. The filter is further characterized by the substantial absence of any bonding agents.

80-0530. Rosen, W. E. **Tobacco Expansion Process.** United States Patent No. 4,013,082, March 22, 1977, 3 pp.

A method of expanding tobacco is described where solutions of hydrogen peroxide and catalase are applied to the tobacco surface. Exposure to negative pressure immediately after application facilitates tobacco cell wall expansion by evolving oxygen gas. In experiments with lamina and stems the increase in expansion over untreated tobacco ranged from 72 to 170 percent.

80-0531. Rosen, W. E. **Method of Treating Tobacco With Catalase and Hydrogen Peroxide.** United States Patent No. 3,889,689, June 17, 1975, 10 pp.

A method of treating tobacco is described which involves the use of physical force to cause a solution containing catalase and hydrogen peroxide to permeate the interstices of the tobacco where they are reacted with each other. The reaction improves the quality of the tobacco and in certain cases expands the volume. The physical force necessary for penetration of the material may take the form of rollers, flat pressure means, or continuous expression means. (Auth. Abs. Mod.)

80-0532. Sanford, R. A. **Reinforcement of Tobacco Structure.** United States Patent No. 4,034,766, July 12, 1977, 3 pp.

Prior to or following expansion, tobacco is treated with nontoxic natural and synthetic resins in order to reinforce the fibrous structure and to prevent collapse of the expanded tobacco. The strengthening agent (e.g., methyl cellulose) is added in an amount ranging from 0.25 to 5 percent, based on the weight of tobacco. The solvent for the impregnating solution can be water; inert, low boiling organic solvents; or a mixture of the two. Among the organic solvents are aromatic hydrocarbons, ethers, aliphatic alcohols, aliphatic hydrocarbons, and halohydrocarbons.

80-0533. Sawada, H.; Kotani, M. **Method for Preparing Cellulose Acetate Fiber Rods.** United States Patent No. 4,007,746, February 15, 1977, 4 pp.

A method is described for preparing a cellulose acetate fiber rod for use as a cigarette filter by adding a plasticizer consisting of 1,4-butanediol diacetate or a mixture of 1,4-butanediol diacetate and one or more plasticizers to a cellulose acetate fiber tow. As a result, the tow can be cured at a remarkably higher speed than with conventional methods and a cellulose acetate rod is obtained which does not harm cigarette flavor and smoking taste. The plasticizer is added in an amount of from 1 to 20 percent by weight, especially 3 to 10 percent, based on the weight of the cellulose acetate fibers.

80-0534. Schmidt, O. K.; Taylor, R. P. **Reconstituted Tobacco Composition and Process for Manufacturing Same.** United States Patent No. 4,144,894, March 20, 1979, 5 pp.

Tamarind gum adhesive which is used as the tobacco particle binding agent herein is a polysaccharide derived from the seed kernels of the tamarind tree, *Tamarindus indica*(L), which is cultivated throughout India, Bangladesh, Ceylon, and Burma. Tamarind gum is cold water insoluble and its degree of hydration with accompanying viscosity increase is temperature related. In the course of heating, the gum becomes gelatinized or hydrated. Therefore, the viscosity of the gum can be kept sufficiently fluid, making it possible to incorporate relatively large proportions of tobacco in the slurries while maintaining a suitable level of viscosity. Most slurries containing 85 percent tobacco require adjustment to 9-11 percent total solids by weight to provide acceptable film formation. By regulating the proportion of gelatinized tamarind gum to cold water dispersed tamarind gum, the total solids content of the castable slurries can be up to about 20 percent by weight of the slurry. In the finished tobacco sheet, the gum system can comprise between 0.5 percent and 33 percent of the weight of the product, and preferably, from 1 percent to 20 percent of the weight of the tobacco sheet. The viscosity of the castable tobacco slurry can be from 500 to 500,000 centipoises and preferably is within the range of 6,000 to 30,000 centipoises.

80-0535. Sexstone, J. H. **Method and Apparatus for the Manufacture of Filter Rods Containing Particulate Material by a Split Rod Technique.** United States Patent No. 3,957,563, May 18, 1976, 10 pp.

A method and apparatus for forming filter rods containing particulate material are described. The method is characterized by providing a filter rod of filtering material with a longitudinal axis and (1) cutting in the direction of the axis, (2) exposing the interior, (3) providing pockets at axially spaced intervals in the interior, (4) depositing particulate material, and (5) closing and sealing the rod. (Auth. Abs. Mod.)

80-0536. Smith, A. B. **Filtering Tobacco Smoke.** United States Patent No. 3,998,232, December 21, 1976, 5 pp.

A tobacco smoke filter cartridge is comprised of an electrically insulating wrapper, a bed of conductive or semiconductive particles packed into the filter chamber, and two electrodes in contact with the particle bed. The cartridge may be mounted in a cigarette or cigar holder, or in a pipe stem which contains a built-in electric supply circuit. (Auth. Abs. Mod.)

80–0537. Tiggelbeck, D. D.; Manes, M. **Mentholated Cigarette Filter.** United States Patent No. 3,972,335, August 3, 1976, 5 pp.

A cigarette filter containing menthol or another smoke-flavoring agent with an improved shelf life and delivery rate is obtained by first impregnating a granular, activated carbon with a nontoxic pore modifying agent (e.g., sucrose) which is adsorbed and retained by the effective pore structure to the extent that at least 50 percent of the pore volume and 60 percent of the surface area of the activated carbon is blocked. Subsequently, the activated carbon is impregnated with 0.2 to 20 percent menthol (w/w). The granular, activated carbon is then formed into a filter.

80–0538. Toyoshima, I.; Ishii, K.; Sawada, H.; Kawamoto, J.; Yokota, H. **Tobacco Filter.** United States Patent No. 4,085,760, April 25, 1978, 6 pp.

This patent describes a tobacco filter made of cellulose acetate fiber which is obtained by acetylating cellulose fiber from any one of a variety of sources such as pulp, cotton, jute, viscose rayon, and copper-ammonia rayon, while maintaining its fibrous state. The filter was superior to currently available cellulose acetate and pulp fiber sheet filters in removing nicotine and total particulate matter from cigarette smoke.

80–0539. Uehara, M.; Maeda, K.; Fujii, S.; Nishio, H. **Mouthpiece for Cigarette.** United States Patent No. 3,978,869, September 7, 1976, 4 pp.

Most cigarette mouthpieces are "straight type." An axially extending straight wall or partition inside a thin exterior tube divides the tube interior into at least two parallel smoke passages. Another mouthpiece is the "baffle type." This mouthpiece has axially spaced walls inside the tube which extend perpendicular to the axis. The walls have transversely offset holes so that the walls serve as baffles and the smoke flows through a zig-zag path. A mouthpiece of the "straight type" has only a minor effect of separating and depositing smoke components on the surfaces of the mouthpiece primarily providing an improved feel in the mouth of the smoker. A mouthpiece of the "baffle type," however, separates condensible smoke components, but because of the high pressure drop a stronger suction must be applied by the smoker. The cigarette mouthpiece described in this patent is comprised of an exterior hollow cylinder which has at least one deflecting wall bonded to its inner surface. The deflecting wall is helical so that along its length it occupies most of the exterior cylinder radii. The juncture of the deflecting wall with the inner surface of the exterior cylinder forms a helix around the cylinder's central longitudinal axis.

80–0540. Uroshevich, M. **Two-Stage Tobacco Smoke Filter.** United States Patent No. 4,016,887, April 12, 1977, 4 pp.

A cigarette filter comprised of a tube having a conical disc at its upstream end adjacent to the tobacco is described. A capillary orifice through the apex of the conical disc permits a high velocity discharge of tobacco smoke into a downstream chamber for sublimation of alkaloids and other harmful matter to solid particles by rapid expansion. A parabolic disc is positioned in line with the high velocity jet to trap the solid particles, to redirect and deflect the smoke, and to promote turbulence. The disc has a periphery closely adjacent to the inner wall of the tube to promote a high speed flow around its edge. The resulting turbulence causes additional deposition of solids on the disc's downstream face. A tube integral with the disc extends to a circular opening in the downstream end of the tube for discharge of filtered smoke. Certain surfaces in the filter, such as the upstream side of the disc, may be coated with a catalyst (e.g., platinum) to enhance a beneficial chemical reaction of constituents in the tobacco smoke. (Auth. Abs. Mod.)

80–0541. Van Hall, F. **Method and Apparatus for Making Tobacco Smoke Filter.** United States Patent No. 4,088,065, May 9, 1978, 9 pp.

A cigarette filter is described which consists of two concentric cylinders of fibrous filter materials such as cellulose acetate. The inner cylinder has a lower draw resistance than the outer one. This filter delivers a smoke stream balanced by ventilation air which results in low smoke solids and a high taste value. Nicotine reductions of 44 to 53 percent were found in the smoke of cigarettes with this filter as compared to those without a filter. A method and apparatus for making a cigarette filter rod are also described.

80–0542. Van Hall, F. **Tobacco Smoke Filter.** United States Patent No. 4,034,765, July 12, 1977, 8 pp.

This patent describes a cigarette filter composed of two concentric cylinders of fibrous filter materials which have different filtering characteristics and air dilution means. Through a low resistance filter core, the filter delivers a concentrated smoke stream which is balanced with ventilation air to yield a smoke stream containing low smoke solids and a high taste value in each puff. The filter materials used to make the concentric layers can be of any suitable filter material such as cellulose acetate, provided the inner cylinder has a lower draw resistance than the outer cylinder. Nicotine reductions of 44 to 53 percent were found in the smoke of cigarettes with the filter as compared to those without the filter.

80–0543. Zizka, J.; Janousek, K.; Papanek, M. **Cigarette Filter.** United States Patent No. 4,124,033, November 7, 1978, 4 pp.

This patent describes a cigarette filter prepared from cellulose material to which finely comminuted bark particles from coniferous trees have been added. The bark contains suberine, a natural resin, which is effective in polycyclic hydrocarbon filtration. The particles, which have a diameter between 1 and 40 microns, may comprise from 5 to 40 percent by weight of the fibrous material. The use of 12.5 percent by weight of pine bark in paper pulp resulted in high absorption capacity and refined flavor and taste. Cigarettes with a 40 percent pine bark filter (by weight) effectively retained most components except carbon dioxide.

See also, **80–0474, 80–0556, 80–0558, 80–0631**

TOBACCO CONSTITUENT ALTERATION

80–0544. Artho, A.; Beringer, M.; Buchmann, P.; Koch, R. **Smokeable Product With Meerschaum Particles as Absorbents.** United States Patent No. 3,987,801, October 26, 1976, 6 pp.

A smoking product is described which includes meerschaum (magnesium silicate hydrate) particles of 100μ diameter as adsorbent for harmful smoke components. At least a part of the tobacco is regenerated tobacco formed of pulp (containing natural tobacco or tobacco waste) and/or tobacco substitute formed of pulp (containing mainly nontobacco plants or other substances). Meerschaum is selected as adsorbent because it has a large adsorption area with a strong adsorption affinity for substances having an electric molecular structure, such as harmful smoke components. For substances which, like most harmless aromatic substances of the smoke, are not polar, the adsorption affinity of the meerschaum is considerably lower. Meerschaum is also particularly suitable because in contrast to, for example, active carbon, it does not decompose in the incandescent zone giving off harmful gases. When the meerschaum particles arrive in the incandescent zone, the main proportion of adsorbed harmful substances either remain adsorbed or are decomposed into products which are either harmless or substantially less harmful than the starting products.

80–0545. Bryant, H. G., Jr.; Norman, V.; Williams, T. B. **Tobacco Composition.** United States Patent No. 4,177,822, December 11, 1979, 5 pp.

A novel smoking composition is described which is comprised of tobacco and a catalytic agent for decreasing the yield of polycyclic aromatic compounds. The catalytic agent consists of finely divided unsupported palladium or palladium salts which are decomposable, preferably by heat, directly or indirectly into metallic palladium. One form of palladium which has been found to be particularly effective in combination with tobacco is palladium black. The proportion of palladium associated with the tobacco in the smoking composition ranges from 0.001 to 1 percent by weight of the tobacco. One method of applying palladium to tobacco is to dry blend the palladium, ground tobacco, a fibrous material, and a binder. Dry blending effectively distributes the palladium over the surface and into the surface pores of the tobacco. The palladium can also be suspended in casing solution and applied directly on cut blended tobacco and manufactured into cigarettes or other products in this form.

80–0546. Casey, W. J. **Nicotine Removal Process.** United States Patent No. 4,068,671, January 17, 1978, 6 pp.

A simple and economical method of removing nicotine from tobacco to produce a tobacco of reduced nicotine content without degrading the tobacco, or without necessitating costly and time-consuming extractive procedures is described. The nicotine is removed by rapidly drying an alkaline aqueous dispersion of tobacco. To improve the efficiency of nicotine removal, the dried tobacco is remoistened with water and rapidly redried. The remoistening and redrying steps are repeated until the desired, or optimum, level of nicotine content is reached. Nicotine reductions of at least 70 percent and higher are attainable with this method.

80–0547. DeBrunn, A. W.; Feist, G. R. **Tobacco Processing Method.** United States Patent No. 3,995,646, December 7, 1976, 5 pp.

A method for processing tobacco to remove harmful agents and to restore the tobacco to a condition suitable for use in a cigarette is described. The tobacco is placed in boiling water for 15 min and is subsequently dried to a point at which the tobacco is friable. The dried tobacco is then rehydrated to restore it to a condition suitable for use in cigarettes by subjecting it to an atmosphere containing water vapor at a reduced temperature for approximately 7 hours. The processed tobacco contained substantially lower amounts of tar (1.6 vs. 2.9 mg), nicotine (0.06 vs. 0.6 mg), nitrogen dioxide (0.04 vs. 0.12 mg NO_2/cm smoked), and carbon monoxide (0.7 vs. 0.22 percent CO (v/v)/cm smoked) than unprocessed tobacco. (Auth. Abs. Mod.)

80–0548. De Brunn, A. W.; Feist, G. R. **Tobacco Processing Method.** United States Patent No. 3,874,392, April 1, 1975, 5 pp.

A method for processing tobacco to remove harmful agents and to restore the tobacco to a condition suitable for use in cigarettes is described. The tobacco is placed in boiling water for 15 min and is subsequently dried to a point at which it is friable. To restore it to a condition suitable for use in cigarettes, the tobacco is then rehydrated by placing it in a sealed, air-filled receptacle containing water vapor which is subjected to such a low temperature that the moisture in the air surrounding the tobacco is condensed. The processed tobacco contained substantially lower amounts of tar, nicotine, nitrogen dioxide, and carbon monoxide than unprocessed tobacco. (Auth. Abs. Mod.)

80–0549. Fiore, J. V. **Mechanical Lipid Removal From Tobacco Leaves.** United States Patent No. 4,018,234, April 19, 1977, 6 pp.

A process for the reduction of lipid content in tobacco compositions with retention of favorable organoleptic properties by the selective removal of the neutral lipid fraction is described. Tobacco leaves, preferably freshly harvested, are subjected to physical treatment adapted to remove lipid material from glandular trichome and cuticle while retaining the structural integrity of the leaf. Tobacco compositions based upon treated leaf structures exhibit significantly reduced lipid levels, reported to interrelate with production of aromatic hydrocarbons upon pyrolysis. (Auth. Abs. Mod.)

80–0550. Geiss, V. L.; Gregory, C. F.; Newton, R. P.; Gravely, L. E. **Process for Reduction of Nicotine Content of Tobacco by Microbial Treatment.** United States Patent No. 4,140,136, February 20, 1979, 11 pp.

A tobacco product with reduced nicotine content is described. Tobacco is subjected, under controlled conditions, to the action of a microorganism (*Cellulomonas sp.* or one of two *Pseudomonas putida* strains) effective in biochemical degradation of nicotine. When tobacco treated by this process is incorporated into a smoking product, it produces a mild smoke with a reduced nicotine content. However, there is no loss of desirable flavor, taste, or smoking properties. This process is particularly useful for treating Burley tobacco which has a relatively high nicotine content and which produces a rather harsh smoke. (Auth. Abs. Mod.)

80–0551. Geiss, V. L.; Gregory, C. F.; Newton, R. P.; Gravely, L. E. **Process for Reduction of Nicotine Content of Tobacco by Microbial Treatment.** United States Patent No. 4,038,993, August 2, 1977, 12 pp.

This patent describes a process for reducing the nicotine content of tobacco using cultures of microorganisms able to degrade nicotine through a biochemical mechanism in which 3-succinoylpyridine is formed. After adding the microorganism to the tobacco, the moisture level is maintained at a 50 percent level by weight of the tobacco and water mixture. Subsequent to the addition of the microorganism to the tobacco, the temperature is maintained between 20 and 45°C while the initial pH of the mixture is maintained between 5 and 8. Tobacco treated by this process has a reduced nicotine content and a mild, pleasant tasting smoke.

80–0552. Gellatly, G.; Uhl, R. G. **Method for Removal of Potassium Nitrate From Tobacco Extracts.** United States Patent No. 4,131,118, December 26, 1978, 5 pp.

A process for selectively removing and recovering potassium nitrate in a relatively pure state from tobacco, and especially from Burley tobacco stems is described. The process entails (1) obtaining an aqueous tobacco extract and a fibrous tobacco residue, (2) concentrating the extract, (3) cooling the extract to crystallize the potassium nitrate, and (4) separating and recovering the potassium nitrate crystals. The denitrated aqueous extract is then recombined with the fibrous tobacco residue for use in reconstituted tobacco. Without further purification, the potassium nitrate is suitable for use as a fertilizer, eliminating costly disposal problems. (Auth. Abs. Mod.)

80–0553. Gravely, L. E.; Geiss, V. L.; Newton, R. P. **Process for Maximizing the Growth and Nicotine Degrading Activity of Microorganisms.** United States Patent No. 4,011,141, March 8, 1977, 9 pp.

This patent describes a process for maximizing the growth and nicotine degrading activity of microorganisms used for treating tobacco to reduce its nicotine content without adversely affecting the desirable smoking qualities of the tobacco. Suitable microorganisms are selected from *Cellulomonas sp.* and *Pseudomonas putida* which degrade nicotine through a biochemical mechanism in which 3-succinoylpyridine is formed. The process involves inoculating a nicotine-containing broth with the microorganisms and subjecting the broth to aeration and agitation. During the culture growth period, the broth is maintained at a pH between 6 and 7.8, while the temperature is maintained between 10 and 45°C. The broth should have an initial nicotine concentration of 0.5 mg/ml up to an amount which is toxic to the microorganisms.

80–0554. Horowitz, C.; Dichter, M.; Abrams, N.; Mangaraj, D. **Removal of Harmful Components From Tobacco Smoke.** United States Patent No. 4,006,749, February 8, 1977, 7 pp.

A process is described for treating tobacco with polymers to remove the harmful components of the tobacco smoke before it reaches the smoker. The process consists of bonding onto the cellulosic tobacco constituents a graft polymer or copolymer having free functional groups reactive with the undesirable component of the tobacco smoke. The cellulosic constituents can be activated by an ionic or free radical process to yield a reactive site on the cellulosic chain which can subsequently be reacted with monomers containing active double bonds, e.g., olefins, vinyl monomers, or acrylic monomers as well as polymerizable ring compounds each possessing the desired active substituents. The graft polymer or copolymer preferably contains acidic groups which can react with the basic, or alkaline, components of tobacco smoke. The smoke from cigars treated with various graft polymer solutions contained between 12 and 49 percent less nicotine, 37 to 56 percent less pyridine, and 20 percent less tar than smoke from untreated cigars.

80–0555. Kite, G. F.; Gellatly, G.; Uhl, R. G. **Method for Removal of Potassium Nitrate From Tobacco Extracts.** United States Patent No. 4,131,117, December 26, 1978, 5 pp.

A process is described for selectively removing and recovering potassium nitrate in a relatively pure state from tobacco, especially from Burley tobacco stems. The process consists of: (1) adding water to tobacco plant parts to obtain an aqueous extract and a fibrous tobacco residue; (2) concentrating the extract; (3) cooling the extract; and (4) separating and recovering the potassium nitrate crystals which are formed. The denitrated aqueous extract is recombined with the fibrous tobacco residue to make reconstituted tobacco. The purified potassium nitrate is suitable without further purification as a fertilizer, thus eliminating costly disposal problems. (Auth. Abs. Mod.)

80–0556. Martin, R. H.; Owens, W. F., Jr. **Gamma Alumina Filled Paper Wrapper for Smoking Articles.** United States Patent No. 4,108,151, August 22, 1978, 3 pp.

Cigarette paper containing a filler of gamma alumina for selectively reducing the organic vapor phase constituents in tobacco smoke is described. The paper is comprised of cellulosic fibers and a gamma alumina filler with the amount of alumina filler ranging from 50 to 80 percent by weight of the paper. In addition to reducing organic vapor phase constituents, the paper has unique advantages over conventional cigarette paper wrappers in that the visible sidestream smoke emanating from the smoking article is reduced. (Auth. Abs. Mod.)

80–0557. Mergens, W. J.; Newmark, H. L. **Treatment of Tobacco With Ascorbic Acid.** United States Patent No. 3,958,580, May 25, 1976, 6 pp.

A method for reducing the nitrogen dioxide (NO_2) content in tobacco smoke is described. The smoke is passed through uncombusted tobacco containing a salt of ascorbic or erythorbic acid, or mixtures of these salts with their respective acids where the molar ratio of acid to salt does not exceed 3:1. As the average cigarette contains approximately 1 g of tobacco, the preferred amount of salts used per cigarette is 10 to 40 mg. In one series of tests, smoke from cigarettes prepared from tobacco treated with approximately 12 mg of an equimolar mixture of the sodium salt of ascorbic acid and ascorbic acid per cigarette contained about one-third the NO_2 content of controls, as measured during a fast puff test. Smoke from cigarettes containing approximatley 25 mg of the above equimolar mixture had essentially no NO_2. The reduction in the nitrogen dioxide content of the smoke also results in a reduction in the nitrosamine content of the smoke.

80–0558. Mitchell, T. G.; Pritchard, J. A. **Smoking Materials.** United States Patent No. 3,974,838, August 17, 1976, 4 pp.

A process for improving the smoking properties of a tobacco smoking material in which the tobacco material is subjected to treatment with at least one amylolytic enzyme capable of converting the starch contained in the tobacco into sugar. The amylolytic enzyme or a source of it is added to the cured or uncured tobacco material which may be in the form of aqueous slurry, tobacco fibers or extract, or moistened or shredded cut leaf, stem, or stalk. (Auth. Abs. Mod.)

80–0559. Newton, R. P.; Geiss, V. L.; Jewell, J. N.; Gravely, L. E. **Process for Reduction of Nicotine Content of Tobacco by Microbial Treatment.** United States Patent No. 4,037,609, July 26, 1977, 9 pp.

A process for the reduction of the nicotine content of tobacco by microbial treatment is described. Tobacco is subjected, under controlled conditions, to the action of microorganisms (*Pseudonomas putida* and *Cellulomonas sp*) capable of degrading nicotine through a biochemical reaction in which 3-succinoyl-pyridine and other byproducts are formed. Prior to subjecting the tobacco to the action of the microorganism, the tobacco is steamed to increase its moisture content. Tobaccos with lowered nicotine content but without loss of mass result from this process following short treatment periods. Tobacco treated in accordance with this process, when incorporated into a tobacco smoking product, produces a mild smoke with reduced nicotine content and no loss of desirable flavor, taste, and smoking properties. This process is particularly useful for treating Burley tobacco which has a relatively high nicotine content and produces a rather harsh smoke. (Auth. Abs. Mod.)

80–0560. Newton, R. P.; Jewell, J. N.; Geiss, V. L.; Gravely, L. E. **Tobacco With Reduced Nicotine Content Due to Microbial Treatment.** United States Patent No. 4,151,848, May 1, 1979, 9 pp.

This patent describes a process for reducing the nicotine content of tobacco. The tobacco is first subjected to steam in order to bring the moisture level of the tobacco to at least 15 percent by weight. Subsequently, the tobacco is inoculated with a microorganism (e.g., *Pseudomonas putida* or *Cellulomonas sp.*) effective in degrading nicotine through a biochemical mechanism in which 3-succinoylpyridine is formed. After adding the microorganism to the tobacco, the moisture level must be maintained at a level of at least 50 percent by weight, based on the total weight of the tobacco and water. Subsequent to the addition of the microorganism to the tobacco, the temperature must be controlled so that it is maintained between 20 and 45°C. while the initial pH of the mixture is maintained between approximately 5 and 8. The microorganism is kept in contact with the tobacco for a period of time sufficient for the microorganism to act on the nicotine in the tobacco. The nicotine content is reduced by degradation to 3-succinoylpyridine. Substantial reductions in nicotine content may be obtained with relatively short treatment periods. No substantial tobacco mass loss occurs and there is no loss of desirable flavor, taste, or smoking properties. Treated tobacco has a mild, pleasant-tasting smoke. The taste may be due to the presence of two nicotine degradation products, 3-succinoylpyridine and 6-hydroxy-3-succinoylpyridine. The process of this patent is particularly useful for treating Burley tobacco. Burley has a relatively high nicotine content and produces a rather harsh smoke. Conventionally, Burley tobacco is treated with casing compositions to reduce harshness. Treatment by this process not only reduces the nicotine content, but reduces harshness to the extent that Burley may be employed in smoking products without casing. (Auth. Abs. Mod.)

80-0561. Newton, R. P.; Jewell, J. N.; Geiss, V. L.; Gravely, L. E. **Tobacco With Reduced Nicotine Content Due to Microbial Treatment.** United States Patent No. 4,151,848, May 1, 1979, 9 pp.

A process for the reduction of the nicotine content of tobacco by microbial treatment (*Pseudomonas putida* and *Cellulomonas*) is described. Tobacco is subjected, under controlled conditions, to the action of a microorganism which degrades nicotine through a biochemical reaction in which 3-succinoylpyridine, as well as 6-hydroxy-3-succinoylpyridine and other byproducts are formed. Prior to this treatment, the tobacco is steamed to increase its moisture content. Tobaccos with lowered nicotine content, but no mass loss, result from this process following short treatment periods. Also, tobacco treated in accordance with this process, when incorporated into a tobacco smoking product, produces a mild smoke with a reduced nicotine content. However, there is no loss of desirable flavor, taste, and smoking properties. (Auth. Abs. Mod.)

80-0562. Norman, V.; Bryant, H. G., Jr. **Tobacco Composition.** United States Patent No. 3,893,464, July 8, 1975, 4 pp.

The concentration of polycyclic aromatic hydrocarbons in tobacco smoke is reduced by incorporating zinc oxide and a Group IIa or IIb nitrate (magnesium, calcium, or zinc) into tobacco. Unlike the Group Ia metal nitrates, the Group IIa and IIb metal nitrates, do not accelerate cigarette burn rate. In addition, they yield more nitric oxide per mole. The amount of zinc oxide utilized ranges from 0.1 to 15 percent by weight of the tobacco in the mixture. The proportion of nitrate associated with zinc oxide and tobacco ranges from 0.25 to 0.75 percent, calculated as nitrate nitrogen.

80-0563. Norman, V.; Bryant, H. G., Jr. **Tobacco Composition.** United States Patent No. 4,055,191, October 25, 1977, 9 pp.

The concentration of the polycyclic aromatic hydrocarbon (PCAH) fraction found in the smoke of a natural leaf smoking tobacco can be substantially reduced without adverse organoleptic effect on tobacco smoke by incorporating both palladium, in metallic or salt form, and an inorganic salt of nitric acid. These tobacco compositions are unique in their ability to significantly reduce the biological activity of the neutral smoke fraction and, specifically, the PCAH-containing subfraction of tobacco smoke produced upon pyrolysis. Palladium may be incorporated into the tobacco composition in finely divided metallic form, for example palladium black, and/or in the form of a salt which is decomposable in situ, preferably by heat, into metallic palladium. Water-soluble palladium salts are preferred because they are readily incorporated into and distributed throughout the tobacco composition. Illustrative examples of suitable palladium salts include simple salts such as palladium nitrate, palladium halides such as palladium chloride, diammine complexes such as palladous dichlorodiammine and palladate salts, especially ammonium salts such as ammonium tetrachloropalladate and ammonium hexachloropalladate. One form of palladium which has been found to be particularly effective in combination with tobacco to provide this smoking composition is ammonium hexachloropalladate. The catalytic amount of palladium associated with the tobacco in the smoking composition is in the range of between 0.001 to about 1 percent by weight of the tobacco used to prepare the smoking composition. Although the reduced yield of polycyclic compounds arising from pyrolytic reactions of the composition have been achieved at these levels, it has been found that the best results are obtained when the palladium is in the preferred range of 0.01 to 0.1 percent based upon the weight of the tobacco.

80-0564. Rainer, N. B.; Hoelzel, C. B. **Smoking Article.** United States Patent No. 4,022,223, May 10, 1977, 5 pp.

Certain heavy metal salt-amine complexes deposited on suitable bases are highly effective in absorbing or removing hydrogen cyanide from the smoke of tobacco or a tobacco substitute. After exposure to cyanides, the metal salt-amine complexes undergo readily visible color change. The transition metals which may be employed in the present invention are selected from the third period elements and of these, copper, nickel, iron, cobalt, and manganese are preferred. Divalent nickel salts and either monovalent or divalent copper salts are particularly active. The salts should be soluble in water and in aqueous ammonia. Metallic salts formed from organic acids of 1 to 12 carbon atoms may also be used to form the metal salt-amine complexes, the formate and acetate ions being most effective. Where the complex is to be utilized in a cigarette filter to remove or sequester the cyanide component in the mainstream tobacco smoke, cellulosic cigarette filter paper or cellulose acetate filaments of 1.5 to 5 denier may be used as substrates. Preferred substrates are porous and have a surface area greater than 0.4 m^2/g. The deposited metal salt-amine complex may be present in the supporting material in an amount from 0.1 to 40 percent, preferably 1 to 10 percent. The complexes generally have a green, blue, or purple color and, as cyanide is absorbed, become colorless. As much as 90-100 percent of the hydrogen cyanide in tobacco smoke can be removed with the metal salt-amine complex described in this patent.

80–0565. Roselius, W.; Vitzthum, O.; Hubert, P. **Process for the Extraction of Nicotine From Tobacco.** United States Patent No. 4,153,063, May 8, 1979, 10 pp.

A process for extracting nicotine is disclosed where tobacco is exposed to an extracting solvent in either a liquid or gaseous state at temperatures below 100°C and at high pressures. The aroma-generating substances can be removed by extraction of dry tobacco. The tobacco can then be moistened, and the nicotine removed. The aroma-generating substances can be recombined with nicotine-free tobacco. In a typical application of this process, the moisture content of tobacco is increased to 25 percent, the pressure is increased to 300 atmospheres, and the temperature is briefly increased to approximately 70°C. The tobacco is then exposed to CO_2 in the supercritical state and the nicotine dissolves. The nicotine can be removed by converting the gas solvent to a liquid in the subcritical state where nicotine precipitates. A particular advantage of this process is that aroma-producing constituents of the tobacco do not dissolve in the extraction solvent. After drying the tobacco to the desired moisture content, the aroma constituents are added to the tobacco. For this extraction process, CO_2 is the preferred solvent. Alternative solvents include N_2O, SF_6, Ar, $CClF_3$, and other lower halogenated hydrocarbons. (Auth. Abs. Mod.)

80–0566. Rudner, B. **Smoking Compositions.** United States Patent No. 4,125,118, November 14, 1978, 4 pp.

The amounts of tar, nicotine, carbon monoxide, hydrogen cyanide, formaldehyde, and other toxic materials generated during tobacco or tobacco substitute pyrolysis are reduced by the incorporation of a transition metal compound. A cigarette tobacco containing cobalt reduced tar, carbon monoxide (CO), and hydrogen cyanide (HCN) levels in smoke by 46, 26, and 21 percent, respectively, as compared to controls. With a manganese constituent, corresponding reductions were 28, 43, and 33 percent. Nickel reduced CO by 25 percent and HCN by 9 percent. The amount of transition metal added to a smoking composition ranges from 0.0005 to 5 percent by weight.

See also, **80–0668**

TOBACCO ADDITIVES

80–0567. Banks, J. F. **Deposition of Vaporized Flavorant on Tobacco.** United States Patent No. Re. 29,298, July 12, 1977, 6 pp.

Tobacco is treated by introducing a tobacco flavorant vapor such as menthol vapor into a conduit and pneumatically conveying the vapor through the conduit as described in U.S. patent No. 3,991,771. Tobacco is injected into the conduit at a point downstream from the vapor injection. The flavorant vapor is uniformly deposited on the tobacco as it is carried down the conduit. A predetermined level of flavorant is maintained in the conduit through which the tobacco and flavorant vapor are conveyed. For example, in order to achieve a mentholation level from 0.2 to 0.5 percent by weight of tobacco during a single pass of tobacco through the system, a contact time of tobacco and menthol vapor of 3 to 15 sec, preferably 5-12 sec, is necessary. The air in the conduit is maintained at 75-150°F, which is sufficient to prevent condensation of the vaporized flavorant in the conduit prior to adsorption on the tobacco. (Auth. Abs. Mod.)

80–0568. Cartwright, W. F.; Means, R. E.; Kallianos, A. G. **Cinnamic Derivatives as Tobacco Additives.** United States Patent No. 3,943,943, March 16, 1976, 5 pp.

Cinnamic derivatives, especially the methyl or ethyl ethers of cinnamyl alcohol, para-methoxycinnamyl alcohol, and para-ethoxycinnamyl alcohol, are added to tobacco to improve the flavor and aroma of the tobacco and tobacco smoke. The derivatives also proved useful in charcoal filter cigarettes when added in an amount sufficient to reduce the typical charcoal effect of the cigarettes. (Auth. Abs. Mod.)

80–0569. Cartwright, W. F.; Means, R. E.; Kallianos, A. G. **Cinnamic Derivatives as Tobacco Additives.** United States Patent No. 4,052,993, October 11, 1977, 5 pp.

The flavor and aroma of tobacco and tobacco smoke are improved by adding cinnamic derivatives, especially the methyl or ethyl ethers of cinnamyl alcohol, *p*-methoxycinnamyl alcohol, and *p*-ethoxycinnamyl alcohol to tobacco.

Cinnamic derivatives also reduce the "charcoal effect" of charcoal filter cigarettes. (Auth. Abs. Mod.)

80–0570. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,014,350, March 29, 1977, 7 pp.

Various compounds (amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, carophyllene derivatives) are useful in flavoring tobacco products. A process is described for improving, enhancing, or modifying the organoleptic properties of a tobacco product, by the addition of 1 to 1000 ppm (based on the weight of the tobacco product) of at least one compound from two groups, caryophyllenepoxides and caryophyllene alcohols.

80–0571. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,029,108, June 14, 1977, 6 pp.

The flavor and aroma of tobacco smoke are improved by adding to a natural tobacco or a tobacco substitute, a flavoring composition containing at least one compound from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, or carophyllene derivatives. A process described in this patent is comprised of the addition of 1 to 1000 ppm (based on tobacco weight) of 2-methyl-5-(α-methyl-α-hydroxyethyl)-cyclohex-2-en-1-one to the tobacco product.

80–0572. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,031,902, June 28, 1977, 6 pp.

Tobacco smoke aroma and flavor are improved by adding a flavoring composition containing at least one compound from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, and heterocyclic ketones to a natural tobacco or a tobacco substitute. A process for improving, enhancing, or modifying the organoleptic properties of tobacco smoke is described in this patent as the addition of 1 to 1,000 ppm of octa-3,5-dien-2-one (based on weight) to the tobacco product. (Auth. Abs. Mod.)

80–0573. Demole, E. P. **Flavored Tobacco Product.** United States Patent No. 3,920,027, November 18, 1975, 6 pp.

Tobacco smoke aroma and flavor are improved by adding to natural tobacco or a tobacco substitute of natural or synthetic origin, a flavoring composition containing at least one compound selected from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, and carophyllene derivatives. In this patent, the addition of 6-methyl-hepta-3,5-dien-2-one is described as improving the organoleptic properties of a tobacco product. (Auth. Abs. Mod.)

80–0574. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,029,107, June 14, 1977, 6 pp.

The flavor and aroma of tobacco smoke are improved by adding to tobacco or to a tobacco substitute a flavoring composition containing at least one compound selected from the following groups: amides, cycloaliphatic derivatives, heterocyclic ketones, caryophyllene derivatives, and other ketones. For example, the ketone nonane-2,5,8-trione may be added to a tobacco product at a concentration ranging from 1 to 1,000 ppm based on the weight of the tobacco.

80–0575. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,092,989, June 6, 1978, 6 pp.

The addition of a flavoring composition containing at least one compound selected from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, caryophyllene derivatives, and other ketones to tobacco or to a tobacco substitute improves the aroma and flavor of the smoke. This patent concerns epoxy-β-ionone a ketone which is added to a tobacco product in the proportion of 1 to 1,000 ppm based on the tobacco weight.

80–0576. Demole, E. P. **Aromatic Compositions.** United States Patent No. 4,029,106, June 14, 1977, 6 pp.

Tobacco smoke flavor and aroma are improved by the addition of a flavoring composition containing at least one compound selected from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, caryophyllene derivatives, and other ketones to a tobacco product. One ketone described in this patent is 3-isopropyl-cyclopent-2-en-1-one which may be added to a tobacco product in concentrations ranging from 1 to 1,000 ppm based on the weight of the tobacco.

80–0577. Demole, E. P. **Aromatic Compositions.** United States Patent No. 3,957,061, May 18, 1976, 6 pp.

The aroma and flavor of tobacco smoke are improved by the addition of a flavoring composition which contains at least one compound selected from the following groups: amides, cycloaliphatic derivatives, phenol derivatives, heterocyclic ketones, and caryophyllene derivatives to natural tobacco or a tobacco substitute. This patent described amides of the structure $RCONHR^1$ (where R and R^1 represent either an alkyl radical comprising 1 to 6 carbon atoms or a hydrogen atom) which are added to a tobacco product at a concentration between 1 and 1,000 ppm based on the weight of the tobacco.

80–0578. Demole, E. P.; Enggist, P. F. **Flavoring Ingredient.** United States Patent No. 4,099,531, July 11, 1978, 3 pp.

A flavoring compound (2-phenyl-3-(fur-2-yl)-prop-2-en-1-al) for tobacco and tobacco substitutes as well as for foodstuffs and beverages is described. When added to tobacco used for the manufacture of test cigarettes, the smoke of the test cigarettes was found to possess a particularly pleasant sweeter taste when compared to the smoke of control cigarettes flavored with 95 percent ethanol.

80–0579. Dickerson, J. P.; Roberts, D. L. **Tobacco Product.** United States Patent No. 3,996,940, December 14, 1976, 3 pp.

The addition of substituted pyrrole compounds to tobacco (in amounts of from 0.0005 to about 1.0 percent by weight of the tobacco) enhances its flavor and/or aroma. While this invention is principally useful in the manufacture of cigarette tobacco, it is also suitable for use in connection with the manufacture of pipe tobacco, cigars, or other tobacco products. Furthermore, the substituted pyrrole compounds can be added to certain tobacco substitutes of natural or synthetic origin. In addition, the compounds need not be mixed directly with the tobacco, but can be applied to the paper of the cigarette or to the wrapper of a cigar. Also, they can be incorporated into the filter tip, the packaging material, or the seam paste employed for gluing the cigarette paper.

80–0580. Dietrich, P. **Flavored Tobacco Composition.** United States Patent No. 3,915,175, October 28, 1975, 3 pp.

The compound 8,13:13,20-diepoxy-15,16-dinorlabdane is described as a useful agent for improving the flavor and aroma of tobacco products. The smoke from cigarettes manufactured from tobacco sprayed with the compound was found to have a pleasant woody and amber-like flavor.

80–0581. Dietrich, P.; Sundt, E. **Tobacco Compositions Employing Flavoring Agents Comprising Unsaturated Butyrolactone Derivatives and Precursors Thereof.** United States Patent No. 3,884,247, May 20, 1975, 4 pp.

The flavor of tobacco was enhanced by adding substituted unsaturated butyrolactone derivatives (also called 2-buten-4,1-olides) and their acid precursors to tobacco mixtures. One hundred kg of a pipe tobacco blend sprayed with 2 kg of a 1 percent solution of 2-hydroxy-4-methyl-2-buten-4,1-olide in a 1:1 mixture of ethanol and propylene glycol, dried, and tested had a more pleasant smoke than the untreated tobacco and a distinct maple-like flavor.

80–0582. Evers, W. J.; Sieczkowski, J. **Novel Flavoring Compositions and Processes.** United States Patent No. 3,942,537, March 9, 1976, 5 pp.

Processes for altering the flavors of consumable products, including foodstuffs and tobaccos, by adding a trace amount of at least one oxocyclic pyrimidine are discussed. Novel oxocyclic pyrimidines and preparative processes are described. Use of these oxocyclic pyrimidines in a flavoring composition can be combined with conventional flavoring materials or adjuvants, including vehicles, stabilizers, thickeners, surface active agents, conditioners, and flavor intensifiers. (Auth. Abs. Mod.)

80–0583. Flynn, C.; Fredericks, G. R.; Hochstetler, A. R. **Tobacco Compositions Flavored With 2-Hydroxymethylenecyclohexanones.** United States Patent No. 4,183,363, January 15, 1980, 5 pp.

It has been found that the organoleptic properties of tobacco products can be improved by addition of a 2-hydroxymethylenecyclohexanone. The addition of a 2-hydroxymethylenecyclohexanone to a blended tobacco improves both the aroma of the fresh tobacco and the aroma and taste of the blend during smoking. Untreated cigarettes have an undesirable harshness upon smoking, an effect which is diminished by adding a 2-hydroxymethylenecyclohexanone. The treated cigarettes have a smoother, mellower, more rounded taste. The hydroxymethylene compounds were prepared from the corresponding ketone by a base catalyzed reaction with a formate such as ethyl formate. The formyl group appears to exist exclusively as the hydroxy-methylene tautomer, a hydrogen bond existing between the hydroxyl hydrogen and the carbonyl oxygen.

80–0584. Grubbs, H. J. **Poly(Isopropenyl Esters) as Carboxylic Acid-Release Agents on Tobacco.** United States Patent No. 4,171,702, October 23, 1979, 6 pp.

In this patent polymers of isopropenyl esters are presented as carboxylic acid flavorant-release oligomeric and polymeric derivatives which are incorporated into tobacco to enhance the flavor and aroma of the smoke. In one method, the carboxylic acid flavorant-release agent is dissolved in a solvent, such as ethanol, and then sprayed or injected into the tobacco matrix. In an alternate method, the flavorant-release agent can be admixed in solid form with the components of a reconstituted sheet of tobacco prior to the forming of the sheet. There is substantially no loss of carboxylic acid flavorant through sublimation or volatilization during the manufacturing and storage operations. In addition, diffusion of the flavorant within the tobacco is successfully eliminated, and tobacco quality and uniformity are maintained.

80–0585. Grubbs, H. J.; Van Auken, T. V.; Johnson, W. R., Jr. **Flavorant-Release Resin Compositions.** United States Patent No. 4,119,106, October 10, 1978, 9 pp.

The low molecular weight oligomeric and polymeric alcohol flavorant-release carbonate ester resin compositions described in this patent consist of α-substituted-vinyl carbonate ester compounds. Regular filter cigarettes (17.9 mg tar) and low delivery cigarettes (2 mg tar) were injected with a poly(2-

phenylethyl isopropenyl carbonate) solution to give a final concentration of 50 ppm. The regular cigarettes had a yeasty, bread-like, metallic, and harsh taste with green and honey-like notes. The low delivery cigarette had a bread-like, yeasty, and metallic taste.

80–0586. Grubbs, H. J.; Van Auken, T. V.; Johnson, W. R., Jr. **α-Substituted Vinyl Menthyl Carbonates.** United States Patent No. 4,127,601, November 28, 1978, 7 pp.

The α-substituted vinyl menthyl carbonate ester compounds are synthesized by reacting menthyl haloformate with an enol alcoholate, and can be converted into menthol-release agents by vinyl homopolymerization. In one smoking composition, a solution of poly(α-methylvinyl *l*-menthyl carbonate) in cyclohexane was sprayed onto tobacco. The amount of polymer applied was 1.74 percent of the weight of the tobacco. Cigarette rods (60 mm) were made by machine with treated tobacco weights of 780 mg per cigarette. Active-carbon filters were attached and 52 mm of the rod was consumed by machine smoking the cigarettes. Menthol delivery to the mainstream smoke was 0.36 mg with the filter retaining 0.62 mg. The efficiency of the filter in terms of removing gas phase components from the smoke was the same as that obtained when identical filters were used on control cigarette rods which contained no added polymer.

80–0587. Hall, J. B.; Hruza, D. E.; Vock, M. H.; Vinals, J. F.; Shuster, E. J. **Flavored Tobacco Article.** United States Patent No. 4,069,828, January 24, 1978, 18 pp.

C_1-C_6 alkyl-2-methyl-3,4-pentadienoates, together with a flavor adjuvant including ethyl butyrate, ethyl valerate, and maltol, are used to enhance the flavor and aroma of tobacco. Cigarettes containing ethyl-2-methyl-3,4-pentadienoate in the tobacco or filter were judged to have a sweeter and fruitier aroma and a more aromatic cigarette smoke flavor.

80–0588. Hall, J. B.; Hruza, D. E.; Vock, M. H.; Vinals, J. F.; Shuster, E. J. **Alkyl-2-Methyl-3,4-Pentadienoates.** United States Patent No. 4,041,069, August 9, 1977, 18 pp.

Tobacco flavoring compositions capable of imparting a sweet, fruity, and woody character to tobacco and tobacco articles may be provided by the utilization of isomer mixtures containing one or more C_1-C_6 alkyl-2-methyl-3,4-pentadienoates. The C_1-C_6 alkyl-2-methyl-3,4-pentadienoates are formed by reaction of propargyl alcohol and a tri-C_1-C_6 alkyl orthopropionate in the presence of a lower alkanoic acid at pressures from 2 to 10 atmospheres, and at temperatures from 140 to 180°C. The mole ratios of triethyl orthopropionate:propargyl alcohol may vary from 1:2 up to 2:1 with a slight molar excess of propargyl alcohol preferred. In aroma, the cigarettes containing the ethyl-2-methyl-3,4-pentadienoate in the tobacco or in the filter have been found to be sweeter and fruitier. In smoke flavor, the cigarettes containing the ethyl-2-methyl-3,4-pentadienoate are more aromatic, sweeter, fruitier, and slightly less harsh in the mouth and throat. In addition, the presence of this ester in the cigarette tobacco results in a woody nuance on smoking.

80–0589. Hall, J. B.; Sanders, J. M. **Novel Tobacco Product Comprising One or More Isomers of an Octahydrotetramethyl Acetonaphthone.** United States Patent No. 3,907,321, September 23, 1975, 20 pp.

Isomers of octahydrotetramethyl acetonaphthone are added to tobacco to enhance its aroma and flavor. The amount of additive is preferably between 200 and 2,000 ppm based on the dry weight of the tobacco product, but will depend upon the amount of flavor and aroma desired and the particular compound or mixture that is used. The additive may be incorporated at any step in the treatment of the tobacco but is preferably added after aging, curing, and shredding and before the tobacco is formed into cigarettes. The smoke from cigarettes prepared from tobacco sprayed with these compounds is described as having a "sweet, spicy, sandalwood-like and cedarwood-like" aroma and flavor.

80–0590. Hall, J. B.; Sprecker, M. A.; Schmitt, F. L.; Vock, M. H. **Process for Producing Substituted-1-Acetyl-3,3-Dimethylcyclohexane.** United States Patent No. 4,102,928, July 25, 1978, 22 pp.

A method for preparation of substituted 1-acetyl-3,3-dimethylcyclohexanes for use in tobacco and tobacco flavorants is described. Green, sweet, fruity, floral, and Virginia tobacco-like nuances are augmented in the tobacco prior to smoking and in the smoke on smoking. In the preparation reactions the preferred mole ratio of 1-acetyl-3,3-dimethylcyclohexane to alkyl halide or methallyl halide reactant ranges from 1:1 to 1:1.2. The preferred mole ratio of base:allyl halide or base:methallyl halide reactant is 1:1 to 1.2:1. The base used is critical. Sodium hydroxide, potassium hydroxide, and potassium-t-butoxide are preferred.

80–0591. Hall, J. B.; Sprecker, M. A.; Vock, M. H.; Shuster, E. J.; Vinals, J. F.; Novak, R. M. **Use of 1-(2-Propenyl)-3-(4-Methyl-3-Pentenyl)-Δ^3-Cyclohexene-1-Carboxaldehyde and 1-(2-Propenyl)-4-(4-Methyl-3-Pentenyl)-Δ^3-Cyclohexene-1-Carboxaldehyde.** United States Patent No. 4,068,012, January 10, 1978, 14 pp.

A process for preparing the compounds 1-(2-propenyl)-3-(4-methyl-3-pentenyl)-Δ^3-cyclohexene-1-carboxaldehydes and 1-(2-propenyl)-4-(4-methyl-3-pentenyl)-Δ^3-cyclohexene-1-carboxaldehydes where the carboxaldehyde moiety is bonded at either the alpha-carbon or beta-carbon atom of the cyclohexenyl moiety is described. These compounds impart cocoa liquor-like, sweet-rich, and creamy oily notes in tobacco and tobacco flavor compositions. Experimental cigarettes containing 500 to 1,000 ppm of 1-allyl-4-(4-methyl-

3-pentenyl)-3-cyclohexene-1-carboxaldehyde and 1-allyl-3(3-methyl-3-pentenyl) -3- cyclohexene -1- carboxaldehyde had more smoke body and a fuller smoke body sensation in the mouth on smoking than control cigarettes.

80–0592. Hall, J. B.; Tseng, C. Y.; Vock, M. H.; Vinals, J. F.; Shuster, E. J. **Novel Process for Altering the Organoleptic Properties of Tobacco Using 2-Methyl-4-Pentenoic Acid and the Ethyl Esters Thereof.** United States Patent No. 3,987,803, October 26, 1976. 11 pp.

The addition of 2-methyl-4-pentenoic acid and/or ethyl-2-methyl-4-pentenoate to tobacco compositions alters the flavor and aroma by providing Turkish-like characteristics as well as aromatic, sweet, sour, bitter, fruity, green, and strawberry notes. Cigarettes containing 100 to 200 ppm of ethyl-2-methyl-4-pentenoate had a more aromatic aroma, and their flavor was slightly less harsh in the mouth and throat, and left a slight, sweet chemical mouth-coating effect similar to Turkish tobacco, as compared to controls.

80–0593. Kallianos, A. G.; Simpson, M. I. **7-Alkoxy-1,2-Benzopyrones as Tobacco Flavorants.** United States Patent No. 4,163,453, August 7, 1979, 4 pp.

The flavor and aroma of tobacco smoke from a natural or synthetic tobacco composition are improved and characterized as sweet, vanilla-like, and similar to new or freshly mown hay by addition of a flavoring composition containing at least one compound of the benzo-1,2-pyrone family to the tobacco. These compounds are used primarily as flavorants for cigarette tobacco. The flavorant is added in an amount to give a final concentration of 0.001 to 0.01 weight percent, based on the weight of the tobacco or the tobacco product. The flavoring agents may be incorporated at any step in the processing of tobacco. They may be applied to the individual tobacco blend components, such as the natural tobaccos, reconstituted tobacco sheet, or tobacco substitutes of natural or synthetic origin. Preferably the compound is added after aging, curing, and shredding, and before the tobacco is formed into cigarettes. There are many methods for blending the flavoring agents with the tobacco. They can be dissolved in ethanol, acetone or any other suitable solvents, or mixed with a carrier such as casing solution to form a suspension or dispersion. The resulting combination can then be sprayed, dipped, or otherwise applied to the tobacco. Alternatively, the agent or agent mixture can be blended with other additives and then mixed into the tobacco. The flavoring agent can also be incorporated into blends normally used in reconstituted tobacco sheet or tobacco substitutes of natural or synthetic origin. Despite their somewhat volatile character, the flavoring agents may also be incorporated into the filter tip, the seam paste employed for gluing the cigarette paper, or the packaging material.

80–0594. Kallianos, A. G.; Southwick, E. W.; Simpson, M. I. **Cyclogeraniol and Derivatives Thereof as Tobacco Flavorants.** United States Patent No. 4,091,823, May 30, 1978, 8 pp.

Addition of a compound selected from the group of alpha-cyclogeraniol, beta-cyclogeraniol, and certain ether, ester, thiol, and thioether derivatives can improve, enhance, or modify the organoleptic properties of tobacco products. Some of these tobacco additives have found particular utility when incorporated in those smoking tobaccos which normally exhibit a harsh taste on smoking. The incorporation of the additives in these tobaccos results in a much smoother smoke, mouth-wetting effects, and usually a pleasant aftertaste. The monocarboxylic acid esters of alpha- and beta-cyclogeraniol, particularly the acetate, have the most pronounced effect in overcoming the harsh taste that results from certain smoking compositions. Beta-cyclogeraniol and its methyl, n-butyl, and isopropyl ether derivatives resulted in a smoother, blander cigarette smoke as compared to controls. Beta-cyclogeraniol n-hexyl ether, however, produced a bitter and peppery smoke. Alpha-cyclogeraniol and beta-cyclogeranyl thiomethyl and butyl ethers resulted in a smoke that was smooth and bland.

80–0595. Kulka, K.; Mild, F.; Fischetti, F., Jr. **Tobacco Compositions Containing 3-Ethoxy-4-Hydroxybenzaldehyde 2,2-Dimethyl Propanediol Acetal.** United States Patent No. 4,128,101, December 5, 1978, 5 pp.

3-Ethoxy-4-hydroxybenzaldehyde 2,2-dimethyl-propanediol acetal has a unique flavor and aroma which markedly enhance the flavor and aroma of tobacco products and foods. Unlike ethyl vanillin from which it is derived, the acetal has a less pronounced vanilla or ethyl vanillin odor and taste and a more intense chocolate by-note. Addition of the acetal of this invention to tobacco in varying amounts ranging from 1 ppm up to 600 ppm has the following effects: (1) prior to smoking, a pleasant, sweet, light aroma at the lower levels, and a distinctly sweet, rich, chocolate-like aroma at the higher levels, and (2) upon smoking, the mainstream becomes smoother and more mellow at the lower levels; whereas at the higher levels, a sweet rich chocolate-like aroma and taste become evident in the mainstream. (Auth. Abs. Mod.)

80–0596. Light, K. K.; Sanders, J. M.; Vock, M. H.; Shuster, E. J.; Vinals, J. F.; Schreiber, W. L.; Hall, J. B.; Hruza, D. E., Sr.; Kamath, V.; Mookherjee, B. D.; Tseng, C. Y.; Sprecker, M. A. **Substituted Norbornane Derivatives Used in the Process of Flavoring Tobacco and the Product Thereof.** Unites States Patent No. 4,120,308, October 17, 1978, 80 pp.

Norbornane derivatives added to smoking tobacco materials or substitutes result in sweet, woody, piney, cooling, and fruity notes. Satisfactory results can be obtained if the proportion by weight of the sum total of norbornane derivatives is between 50 and 15,000 ppm (0.015 to 1.5 percent). For example, an aged, cured, and shredded, domestic Burley tobacco was sprayed with a 20 percent ethyl alcohol solution of 2-(2-butenoyl)-3,3-dimethylnorbornane to provide final additive concentration of 80 ppm in the tobacco composition. Alcohol was removed by evaporation and the tobacco was manufactured into cigarettes. Compared with controls, the resulting smoke aroma was sweeter, more aromatic, more tobacco-like, and had fruity notes.

80–0597. Light, K. K.; Shuster, E. J.; Vinals, J. F.; Vock, M. H. **Tricyclic Alcohols and Lithium Salts Thereof.** United States Patent No. 3,989,760, November 2, 1976, 14 pp.

A racemic mixture of a novel organic tricyclic alcohol (2,3,3A,4,5,7A-hexahydro-6,7A,8,8-tetramethyl-1,5-methano-1H-inden-1-ol) used for altering the aroma and/or flavor of consumable products such as food, tobacco, and perfume is described. In a tobacco formulation experiment 10 and 20 ppm of 2,3,3A,4,5,7A-hexahydro-6,7A,8,8-tetramethyl-1,5-methano-1H-inden-1-ol were added to 50 percent of the cigarettes. The control and experimental cigarettes were then evaluated by paired comparison. The experimental cigarettes were found to be more aromatic, greener, richer, more cigarette tobacco-like, and slightly less harsh in the mouth than the control cigarettes.

80–0598. Light, K. K.; Shuster, E. J.; Vinals, J. F.; Vock, M. H. **Tricyclic Alcohols.** United States Patent No. 4,041,084, August 9, 1977, 13 pp.

Tricyclic alcohols are used in compositions for augmenting or enhancing the flavor and/or aroma of foods, tobacco, and perfumes. Experimental cigarettes containing 20 ppm of 8,8-dimethyloctahydro-1,5-methano-1H-inden-1-ol were woody, slightly chemical, and mouth-coating in smoke flavor. Use of this compound enhanced the tobacco-like taste of the blended cigarette.

80–0599. Light, K. K.; Spencer, B. M.; Vinals, J. F.; Kiwala, J.; Vock, M. H.; Shuster, E. J. **Tobacco Flavoring With Cyclohexadiene Derivatives.** United States Patent No. 4,135,520, January 23, 1979, 19 pp.

Tobacco and tobacco flavor compositions utilizing cyclohexadiene derivatives have notes that are "dry, black tobacco-like," spicy, celery-like, black pepper-like, sweet, and Virginia tobacco-like. The cyclohexadiene derivatives are produced by either of two reactions. One is the fractional distillation of the product of the reaction of an alkyl-trimethylcyclohexa-1,3-diene with oxygen in the presence of light, an ultraviolet sensitizer, and a basic catalyst. The second process reacts 4-alkyl-4-hydroxy-3,5,5-trimethyl-2-cyclohexen-1-one with dilute aqueous mineral acid and distills the resulting reaction product. Two possible reaction products are 2,4,4,6-tetramethylcyclohexa-2,5-diene-1-one and 4-butyl-3,4,5-trimethyl-2,5-cyclohexadiene-1-one, respectively.

80–0600. Light, K. K.; Spencer, B. M.; Vinals, J. F.; Kiwala, J.; Vock, M. H.; Shuster, E. J. **Smoking Tobacco and Smoking Tobacco Flavoring Compositions Containing Hydroxy Cyclohexenone Derivatives.** United States Patent No. 4,126,140, November 21, 1978, 19 pp.

Tobaccos and tobacco flavoring compositions having sweet, tobacco-like, floral, and green aromas prior to smoking and bright tobacco-like notes on smoking, may be provided by the utilization of one or more hydroxy cyclohexenone derivatives. The hydroxy cyclohexenone derivatives may be produced by (1) oxidizing 1,3,5,5-tetramethylcyclohexa-1,3-diene at 15 to 30°C while agitating and irradiating the diene with ultraviolet light in the presence of an alkali metal hydroxide and Rose Bengal, (2) reacting 2,7,9,9-tetramethyl-1,4-dioxaspiro-(4,5)dec-6-en-8-one with an alkyl lithium to form a lithium salt, and (3) hydrolyzing the salt while simultaneously deketalizing the resulting compound.

80–0601. Light, K. K.; Spencer, B. M.; Vinals, J. F.; Kiwala, J.; Shuster, E. J. **Process for Preparing 2-Butyl-3,5,5-Trimethyl-2-Cyclohexen-1,4-Dione.** United States Patent No. 4,126,641, November 21, 1978, 8 pp.

Processes and compositions are described for the use of 2-butyl-3,5,5-trimethyl-2-cyclohexen-1,4-dione in tobacco, perfume, and perfumed article aroma augmenting, enhancing, and imparting compositions. Addition of this compound to tobacco results in pungent, sweet, hay-, tea-, tobacco-like, floral, and fruity notes, both prior to and on smoking. The concentration of the additive in the smoking tobacco material ranges from 50 to 1500 ppm.

80–0602. Miller, C. W.; Dickerson, J. P.; Rix, C. E. **Tobacco Product.** United States Patent No. 3,996,941, December 14, 1976, 3 pp.

Substituted lactone compounds are added to tobacco (in amounts of from about 0.0005 to about 1.0 percent by weight of tobacco) to improve its flavor and/or aroma. Cigarettes manufactured from tobacco sprayed with 2-(5-hydroxymethyl-2-formylpyrrol-1-yl)-3-phenylpropionic acid lactone had a spicy, peppery flavor or aroma when smoked. While this invention is principally useful in the manufacture of cigarette tobacco, it is also suitable for use in connection with the manufacture of pipe tobacco, cigars, or other tobacco products. Furthermore, the lactones can be added to certain tobacco substitutes of natural or synthetic origin.

80–0603. Mookherjee, B. D.; Trenkle, R. W.; Vock, M. H.; Shuster, E. J. **Tobacco Article Comprising a Flavoring Composition Selected From Butanone Derivatives and the Process for Making the Same.** United States Patent No. 3,977,419, August 31, 1976, 9 pp.

A process is described for adding to tobacco or to homogenized or reconstituted tobacco an amount of 4-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-2-butanone and/or 4-(6,6-dimethyl-2-methylene-3-cyclohexen-1-yl)-2-butanone sufficient to alter the flavor or aroma. These compounds may be added to the tobacco material, to the filter, or to the leaf or paper wrapper. The tobacco material may be shredded, cured, cased, and blended tobacco material, reconstituted tobacco material tobacco substitutes (e.g., lettuce leaves), or mixtures of these. Satisfactory results are obtained if the proportion by weight of the sum total of 4-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-2-butanone and/or 4-(6,6-dimethyl-2-methylene-3-cyclohexen-1-yl)-2-butanone to smoking tobacco material is between 10 ppm and 50 ppm (0.001-0.005 percent) of the active ingredients in the smoking tobacco material. Satisfactory results are also obtained if the proportion by weight of the sum total of 4-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-2-butanone and/or 4-(6,6-dimethyl-2-methylene-3-cyclohexen-1-yl)-2-butanone used to flavoring material is between 1 percent and 10 percent. The flavoring additives may be dissolved in a suitable solvent such as ethanol, pentane, diethyl ether, and/or other volatile organic solvents and the resulting solution may either be spread on the cured, cased, and blended tobacco material, or the tobacco material may be dipped into the solution.

80–0604. Nagakura, A.; Moroe, M.; Koyama, Y.; Kurihara, H. **3,7,11,11-Tetramethyl-Spiro-(5,5)Undeca-8-Ene-1-One.** United States Patent No. 4,052,457, October 4, 1977, 4 pp.

A novel spiro compound, 3,7,11,11-tetramethyl-spiro(5,5)undeca-8-ene-1-one for altering the flavor or aroma of tobacco products is described. The spiro compound is prepared by reacting pulegone and piperylene in a solvent in the presence of a Lewis acid catalyst. It was found that the irritative smell and taste of commercially available cigarettes were markedly reduced by the addition of the spiro compound. (Auth. Abs. Mod.)

80–0605. Newton, R. P., Jr.; Johnson, R. R. **Tobacco Treatment.** United States Patent No. 3,957,060, May 18, 1976, 4 pp.

The treatment of acidic tobacco with certain nonvolatile, organic compounds, in limited amounts, causes an increased level of perception of both strength and satisfaction from the tobacco smoke. By using these compounds, high efficiency filters for tobacco products can be employed with lower deliveries of total particulate matter and without loss of smoking satisfaction. The use of urea and related compounds, particularly biuret, placed on the tobacco in amounts from 1 percent to 5 percent has been effective. Urea has been suggested for reduction of nitrosamines in smoke (South African patent No. 641,432). These compounds remain inactive when placed upon the tobacco and do not materially affect the pH or the chemistry of the tobacco. However, the pH of the total particulate matter delivered upon smoking is increased. The increased pH appears to enhance the perception of greater balance, strength, and satisfaction in treated vs. control cigarettes.

80–0606. Pittet, A. O.; Klaiber, E. M.; Vock, M. H.; Shuster, E. J.; Vinals, J. F. **Uses in Tobacco and as a Tobacco Flavor Additive of Enol Esters.** United States Patent No. 4,086,927, May 2, 1978, 42 pp.

Formulation processes and flavor compositions are described which use one or more alkyl side chain methyl unsubstituted 2,2,6-trimethyl-1-cyclohexen-1-vinyl alkanoates (enol esters) and their *cis* and *trans* isomers. The enol esters may be added to the smoking material, the filter, the leaf, or the paper wrapper. The smoking material may be shredded, cured, cased, and blended tobacco; reconstituted tobacco; tobacco substitutes; or mixtures of these.

80–0607. Pittet, A. O.; Klaiber, E. M.; Vock, M. H.; Shuster, E. J.; Vinals, J. F. **Novel Tobacco Flavoring and Aroma Compositions and Tobaccos Containing Alpha-Substituted Acetaldehyde.** United States Patent No. 4,014,351, March 29, 1977, 10 pp.

Processes and compositions are described using 2,2,6-trimethyl-1-cyclohexen-1-ylacetaldehyde in tobacco to alter, modify, or enhance the aroma and flavor. This acetaldehyde is produced by oxidation of beta-ionone with peralkanoic acid in the presence of buffer and in the absence of dimethyl formamide, to form 2,2,6-trimethyl-1-cyclohexen-1-ylvinylacetate. This product then undergoes hydrolysis to form beta-cyclohomocitral. Prior to smoking, the tobacco of the experimental cigarettes has sweet, floral, fruity, earthy, and green notes. On smoking, the experimental cigarettes are sweeter, richer, more tobacco-like, and less harsh with woody damascenone-like notes. All cigarettes were evaluated for smoke flavor with a 20 mm cellulose acetate filter. (Auth. Abs. Mod.)

80–0608. Pittet, A. O.; Pascale, J. V.; Patton, S.; Brodnitz, M. H. **Compositions and Methods Utilizing Thio Derivatives and Processes for Producing Such Derivatives.** United States Patent No. 3,895,640, July 22, 1975, 9 pp.

The organoleptic properties of tobacco or tobacco substitutes can be altered by utilizing 4-(methylthio)butane derivatives having the formula $CH_3S(CH_2)_3CYX$ where (1) X is hydrogen and Y is a carbonyl oxygen, (2) X is hydroxy and Y is two hydrogen atoms or a carbonyl oxygen, or (3) X is lower alkoxy and Y is carbonyl oxygen or hydrogen and lower alkoxy. Use of ethyl-4-(methylthio)butyrate in a fruity tobacco flavorant enhances the pineapple character and enables the acetaldehyde level to be raised tenfold as compared to the permissible levels in ordinary fruit flavors for tobacco.

80-0609. Renold, W.; Skorianetz, W.; Schulte-Elte, K.-H.; Ohloff, G. **Spirane Derivatives, Their Use and Process for Preparing Same.** United States Patent No. 4,114,630, September 19, 1978, 12 pp.

This patent describes novel spirane derivatives, a process for preparing them, and their use in enhancing the flavor of tobacco products. The smoke from cigarettes manufactured from tobacco sprayed with an isomer of 2,6,10,10-tetramethyl-1-oxa-spiro(4.5)decan-6-ol was found to have a woody and earthy taste.

80-0610. Rundberg, E.G.S., Jr.; Johnson, W. R., Jr.; Grubbs, H. J. **Smoking Compositions Incorporating a Menthol-Release Agent.** United States Patent No. 4,002,179, January 11, 1977, 9 pp.

Novel oligomers and polymers produced from monomer carbonate esters of menthol are described in this patent. These compounds are used as menthol-release agents in natural and reconstituted smoking products to improve their aroma and flavor.

80-0611. Schumacher, J. N.; Best, F. W.; Green, C. R. **Tobacco Product.** United States Patent No. 4,056,108, November 1, 1977, 4 pp.

Addition of imidazole and benzimidazole compounds to tobacco or foodstuffs enhances the aroma and/or flavor. The amount of additive used is between 0.001 and 0.1 percent by weight. The additives can be incorporated at any step in treatment of the tobacco, but it is preferable to add them after aging, curing, and shredding, and before the tobacco is formed into cigarettes or other products. Most imidazole additives (4,5-dimethylimidazole; 4-methylimidazole; 2,4-dimethylimidazole; 2-isopropylimidazole; 2-isopropyl-4-methylimidazole; 2,4,5-trimethylimidazole) have an aroma or flavor which can be characterized as sweet, nutty, and flue-cured like when compared with a control. Benzimidazole adds body. One of its derivatives has a toasted note (4-acetyl-5-methylimidazole) and one (2-methylbenzimidazole) is somewhat hot and peppery. The benzimidazole derivatives 2-acetyl-4-methylimidazole and 2-isovalerylimidazole are very similar to their parent compound.

80-0612. Skorianetz, W. **Polycyclic Lactones as Odor- and Taste-Modifying Agents.** United States Patent No. 3,981,892, September 21, 1976, 4 pp.

Polycyclic lactones are useful as perfuming and odor-modifying agents in the manufacture of perfumes, and as flavoring and taste-modifying agents in the aromatization of foodstuffs and imitation flavors for foodstuffs, beverages, animal feeds, pharmaceutical preparations, and tobacco products. These compounds are used in proportions of 10 to 100 ppm (w/w) and create a spicy and fruity note in tobacco products. (Auth. Abs. Mod.)

80-0613. Teng, L. C. **Smoking Composition.** United States Patent No. 4,036,237, July 19, 1977, 7 pp.

A smoking composition comprised of certain aromatic beta-hydroxy compounds as sources of smoke flavor or aroma is described. Compounds which may be employed as a source of cherry-like or fruity flavor or aroma include beta-hydroxy esters formed by a Reformatsky-type condensation reaction which uses an alkyl alpha-bromo-isobutyrate or a homologous compound with an aromatic aldehyde such as benzaldehyde, anisaldehyde, or piperonal. Other compounds which may be employed are the acids and/or metal salts which are formed by hydrolysis of such beta-hydroxy esters. The amount of beta-hydroxy compound employed in the smoking product is from 0.005 to 0.05 percent by weight, based on the total weight of smoking material present. Novel compositions of matter provided by the present invention include ethyl 2,2-dimethyl-3-hydroxy-3-(3',4'-methylenedioxyphenyl)-propionate, n-amyl 2,2-dimethyl-3-hydroxy-3-phenylpropionate, methyl 2,2-dimethyl-3-hydroxy-3-phenylpropionate, 2,2-dimethyl-3-hydroxy-3-(3',4'-methylenedioxyphenyl) propionic acid, ethyl 2,2-dimethyl-3-hydroxy-3-(*p*-methylphenyl) propionate, and ethyl 2,2-dimethyl-3-hydroxy-5-phenyl-*trans*-4-pentenoate. These compounds release flavors or aromas and can be maintained and preserved during subsequent processing and storage of the tobacco. The compounds may be added at any manufacturing stage. One or more may be added to the wrapper or to the tobacco at an early stage of processing. However, it is preferable to add them to the shredded tobacco or other filler prior to formation into a smoking article. (Auth. Abs. Mod.)

80-0614. Van Auken, T. V.; Grubbs, H. J.; Johnson, W. R., Jr. **Smoking Tobacco Compositions.** United States Patent No. 4,092,988, June 6, 1978, 11 pp.

This patent describes a class of novel carbonate ester (e.g., poly(1-anisyl-1-propyl allyl carbonate) and poly(allyl 3-methylbut-1-en-3-yl carbonate) resin compositions which are adapted for incorporation into tobacco compositions, and which under normal smoking conditions release a highly desirable type of flavorant such as isoprene, anethole, or menthene. A smoking composition is comprised of a 0.0005 to 10 weight percent mixture of natural or reconstituted tobacco, the polymeric carbonate ester composition having a molecular weight in the range between 500 and 2,000,000. For one smoking composition, poly(1-anisyl-1-propyl allyl carbonate) was dissolved in acetone, and portions of the solution were injected into test cigarettes. Two types of cigarettes were used: (a) a regular filtered cigarette delivering 17.9 mg of Federal Trade Commission (FTC) "tar" per cigarette, and (b) a low-delivery filtered cigarette giving 2 mg of FTC "tar" per cigarette. Each type of cigarette was injected with sufficient solution to place 5, 50, and 100 ppm of polymer in the filler.

These cigarettes were then smoked and compared to control cigarettes. The regular (17.9 mg) tar cigarette containing 5 ppm polymer added some sweetness, was slightly harsher, and had more body than the low-delivery (2 mg) cigarette, which added a little sweetness. At 100 ppm polymer levels, the regular cigarette was smoother, added sweetness and a slight anethole aftertaste, whereas the low-delivery cigarette added sweetness, reduced "gas phase" character, and reduced dryness.

80–0615. Vinals, J. F.; Kiwala, J.; Evers, W. J.; Heinsohn, H. H., Jr. **Novel Tobacco Product Comprising One or More Sulfides and Process.** United States Patent No. 4,083,373, April 11, 1978, 8 pp.

Preparation of two sulfide compounds, 3-(methallylthio)-2, 6-dimethyl-4-heptanone, and (3,6-dimethyl-2-pyrazinyl) (2-methyl-3-furyl) sulfide, and their use in tobacco flavor formulations are described. In one experiment, an aged, flue-cured, and shredded tobacco was sprayed with a 1 percent ethyl alcohol solution of (2-methyl-3-furyl) (3,6-dimethyl-2-pyrazinyl) sulfide in an amount to provide a tobacco containing 100 ppm of the additive on a dry weight basis. The alcohol was removed by evaporation, and the tobacco was manufactured into cigarettes. The resultant cigarettes had an aroma described as nutty-bread/hazelnut. This aroma/flavor was detectable in both main and side smoke streams. The preferred sulfide additive concentration range is from 100 to 500 ppm based on tobacco dry weight. However, the concentration may range from 50 to 5,000 ppm.

80–0616. Vinals, J. F.; Kiwala, J.; Hruza, D. E., Sr.; Hall, J. B.; Vock, M. H. **2-Alkyl-4-Phenyl-Dihydropyrans and Processes for Augmenting the Organoleptic Properties of Tobacco Using One or More of Said Pyrans.** United States Patent No. 4,071,034, January 31, 1978, 15 pp.

Pyran derivatives are added to tobacco or applied to a smoking article or its component parts in amounts between 75 and 300 ppm by weight in order to provide a tobacco product having a desired flavor and aroma. The additive may be incorporated at any step in the treatment of the tobacco but is preferably added after aging, curing, and shredding, and before the tobacco is formed into cigarettes. The use of 200 ppm of 2-isopropyl-4-phenyl-dihydropyran in cigarettes results in a sweet, spicy, coumarin-like aroma prior to smoking. On smoking, this compound promotes salivation, decreases harshness, and enhances the natural tobacco-like character. The smoke flavor has sweet, creamy, vanilla, and coumarin-like character.

80–0617. Vinals, J. F.; Kiwala, J.; Hruza, D. E., Sr.; Hall, J. B.; Vock, M. H. **2-Alkyl-4-Phenyl-Dihydropyrans and Processes for Augmenting the Organoleptic Properties of Foodstuffs and Tobacco Using One or More of Said Pyrans.** United States Patent No. 4,070,491, January 24, 1978, 14 pp.

A compound from the group 2-alkyl-4-phenyl-dihydropyrans may be added to food, chewing gum, or toothpaste to augment or enhance neroli-like and grape-like flavors. When added to cigarette tobacco at the rate of 100 to 200 ppm, 2-isopropyl-4-phenyl-dihydropyran results in a sweet, spicy, coumarin-like aroma prior to smoking. On smoking, this compound enhances the natural tobacco-like character, promotes salivation, decreases harshness, and has a sweet, creamy, vanilla, and coumarin-like character.

80–0618. Vinals, J. F.; Kiwala, J.; Hruza, D. E., Sr.; Hall, J. B.; Vock, M. H. **2-Alkyl-4-Phenyl-Dihydropyrans and Processes for Augmenting the Organoleptic Properties of Foodstuffs and Tobacco Using One or More of Said Pyrans.** United States Patent No. 4,071,535, January 31, 1978, 14 pp.

The addition of 2-alkyl-4-phenyl-dihydropyrans to tobacco augments the natural aroma and flavor. Addition to food, chewing gum, or toothpaste augments and enhances neroli- and grape-like flavors. Cigarette tobacco containing 100 to 200 ppm of 2-alkyl-4-phenyl-dihydropyran results in a smoke stream flavor that promotes salivation, decreases harshness, and has a sweet, creamy, vanilla, and coumarin-like character.

80–0619. Vinals, J. F.; Kiwala, J.; Hruza, D. E., Sr.; Hall, J. B.; Vock, M. H. **2-Alkyl-4-Phenyl-Dihydropyrans and Processes for Augmenting the Organoleptic Properties of Foodstuffs and Tobacco Using One or More of Said Pyrans.** United States Patent No. 4,115,406, September 19, 1978, 14 pp.

The compound group 2-alkyl-4-phenyl-dihydropyrans is added to food, chewing gum, and toothpaste to augment or enhance neroli-like and grape-like flavors. Used in conjunction with tobacco products at 100 and 200 ppm, these compounds promote salivation, decrease harshness, and have sweet, creamy, vanilla, and coumarin-like effects. The natural tobacco-like character is also enhanced.

80–0620. Vinals, J. F.; Kiwala, J.; Withycombe, D. A.; Mookherjee, B. D.; Mussinan, C. J. **Novel Tobacco Product Comprising One or More Thiazolines and Process.** United States Patent No. 4,067,344, January 10, 1978, 16 pp.

Tobacco flavoring compositions and tobacco products having sweet, aromatic, nutty, bready, chocolate-like, and/or slightly fatty flavor and aroma nuances prior to and on smoking may be provided by adding to tobacco flavors and/or to tobaccos themselves one or more thiazoline compounds. For instance, an aged, flue-cured, and shredded tobacco can be sprayed with a 1 percent ethyl alcohol solution of a 40:40:20 (weight:weight:weight) mixture of 2-(2′-methyl-n-propyl)-4,5-dimethyl-Δ^3-thiazoline: 2-(2′-n-butyl)-4,5-dimethyl-Δ^3-thiazoline: 2-(2′-methylthioethyl)-4,5-dimethyl-Δ^3-thiazoline in an amount to provide a tobacco containing 100 ppm by weight of the mixture of thiazoline additives on a dry basis. The alcohol is then removed by evaporation, and the tobacco is manufactured into cigarettes by the usual techniques. Water or volatile organic solvents, such as alcohol, ether, acetone, and volatile hydrocarbons may be used as the additive carrying medium during application to the tobacco. The compound may be applied to the paper of the cigarette or to the wrapper of a cigar. Also, it may be incorporated into the filter tip, the packaging material, or the seam paste.

80–0621. Vinals, J. F.; Vock, M. H.; Mookherjee, B. D.; Kamath, V. **Tobacco Article Comprising the Flavoring Composition Tricyclene-9-Butenone and the Process for Making the Same.** United States Patent No. 3,977,418, August 31, 1976, 9 pp.

The flavor and aroma of a tobacco mixture are improved by the addition of tricyclene-9-butenone (1,7-dimethyl-7(1-pent-2-en-4-onyl nortricyclene) to homogenized or reconstituted tobacco. Prior to smoking, cigarettes containing tricyclene-9-butenone had a sweet, floral, spicy-sandalwood, green aroma. On smoking, these cigarettes tricyclene-9-butenone are found relative to those cigarettes not were not only richer, sweeter, and spicier than control cigarettes but they also contained floral and woody notes.

80–0622. Warfield, A. H.; Galloway, W. D.; Kallianos, A. G. **Tobacco With Flavor Enhancer.** United States Patent No. 3,920,026, November 18, 1975, 4 pp.

This patent describes a tobacco flavor enhancer from the reaction of valine with a sugar, other hydroxycarbonyl compound, or dicarbonyl compound. The reagents are heated in the presence of a solvent, optionally including a catalyst in the form of a flavanoid or hydroxyacid. The reaction mixture can be used directly in tobacco compositions, or can be stripped, and either or both the volatile and nonvolatile portion can be used in tobacco compositions. (Auth. Abs.)

80–0623. Watson, H. R.; Rowsell, D. G.; Browning, J.H.D. **Tobacco and Tobacco-Containing Manufactures Containing an Ingredient Having Physiological Cooling Activity.** United States Patent No. 4,059,118, November 22, 1977, 5 pp.

This patent describes a group of 3-substituted-*p*-menthanes which have a pronounced physiological cooling activity, little or no odor, relatively low volatility, and no toxicity. The physiological cooling activity can be imparted to tobacco and tobacco products, e.g., pipe tobacco, cigarettes, cigars, chewing tobacco, or snuff, by the incorporation of certain cold receptor stimulating derivatives of *p*-menthane (i.e., *p*-menthane-3-carboxylic acid, its salts, and certain esters; and *p*-menthane-3-carboxamide). The "cool" sensation occurs when the ingredient comes into contact with the nasal and oral mucosa, by either tobacco smoke inhalation, or by direct contact of the tobacco on the nasal or oral mucosa.

See also, **80–0525, 80–0544, 80–0628**

80–0624. Watson, H. R.; Rowsell, D. G.; Spring, D. J. **Tobacco and Tobacco-Containing Manufactures Containing an Ingredient Having Physiological Cooling Activity.** United States Patent No. 4,060,091, November 29, 1977, 6 pp.

Physiological cooling activity is imparted to tobacco or to tobacco products such as cigarette or pipe tobacco, cigars, chewing tobacco, or snuff by the addition of *p*-menthane-3-carboxamides. These compounds indirectly (by inhalation) or directly stimulate the cold receptors in the oral and nasal mucosa. The *p*-menthane-3-carboxamides have little or no odor, low volatility, and are substantially nontoxic.

80–0625. Willis, B. J.; Fischetti, F., Jr.; Eilerman, R. G. **Flavoring Compositions Containing Alkyl-2,3-Dihydro-3(1′-Hydroxyalkylidene)-2-Oxo-5-Alkyl-Furan-4- Carboxylates.** United States Patent No. 4,109,662, August 29, 1978, 4 pp.

The flavor compositions described in this patent include alkyl-2,3- dihydro -3(1′-hydroxyalkylidene) -2- oxo -5- alkyl-furan-4- carboxylates. The amount of flavorant added to the smoking compositions may vary widely from 0.0001 to 30 percent by weight. When added to natural or synthetic tobacco, the resulting product is increased in sweetness, the taste of the smoke is softened, and the flow of saliva is increased. (Auth. Abs. Mod.)

80–0626. Withycombe, D. A.; Mookherjee, B. D.; Vock, M. H.; Vinals, J. F. **Flavoring With 2,4,6-Triisobutyl-1,3,5-Trioxane.** United States Patent No. 4,093,752, June 6, 1978, 23 pp.

The flavoring agent 2,4,6-triisobutyl-1,3,5-trioxane is used in food, chewing gum, toothpaste, medicinal products, and tobacco. Cigarettes treated with 500 or 1,000 ppm of 2,4,6-triisobutyl-1,3,5-trioxane and control (untreated) cigarettes were evaluated for smoke flavor with a 20 mm cellulose acetate filter. Prior to smoking, the tobacco of the experimental cigarettes had chocolate-like, cocoa-like, and sweet creamy notes compared to controls. On smoking, the experimental cigarettes had very pleasant chocolate-like aroma notes and more tobacco-like nuances as compared with controls.

80–0627. Wolt, J.; Giacino, C.; Quinn, A. D.; Shuster, E. J.; Vinals, J. F. **Tobacco Articles and Compositions Containing 1,2-Cyclohexanedione and Methods for Producing Same.** United States Patent No. 3,903,900, September 9, 1975, 5 pp.

Novel solid and liquid tobacco flavoring compositions having sweet burnt notes reminiscent of licorice and cyclotene and which can be described as sweet caramel and maple-like; novel tobacco aroma imparting compositions having maple, black walnut, and foenugreek character; and tobacco or tobacco substitute compositions having maple and coumarin notes may be provided by the utilization of 1,2-cyclohexanedione alone or in combination with such di-ketones as maltol, cyclotene, and 2,5-dimethyl-4-hydroxy-3(2H)-furanone. Methods for preparing these flavoring compositions are described.

TOBACCO SUBSTITUTES

80-0628. Anderson, R. C.; Gibson, J. A. **Smoking Mixtures.** United States Patent No. 3,924,644, December 9, 1975, 5 pp.

This patent describes a smoking mixture which has the general flavor of flue-cured Virginia tobacco and is formulated from a mixture of nicotine, a modified (thermally degraded) carbohydrate, and flue-cured Virginia tobacco. Thermally degraded carbohydrate may be prepared as described in United Kingdom Patent No. 1,113,979 by subjecting carbohydrate to a catalyzed degradation process at a temperature of 100° to 250°C, until the weight of the degraded material is less than 90 percent of the dry weight of the original carbohydrate. The mixture includes as flavorants isovaleric or β-methyl valeric acids and/or mixtures of both in amounts from 0.02 percent to 0.2 percent by weight of the mixture. When testing a number of different smoking mixtures with varying amounts of flavorant, it was found that the flavored cigarettes were always preferred over those without flavorant.

80-0629. Anderson, R. C.; Hall, R. A. **Smoking Mixture.** United States Patent No. 3,965,911, June 29, 1976, 6 pp.

This patent describes a tobacco substitute mixture which contains protein in order to give the smoke a flavor resembling that of tobacco. The mixture consists of an organic combustible material as smoke-producing fuel and a protein in proportions ranging from 60:1 to 1:1. The smoke-producing fuel may be a carbohydrate or an aldol condensation product; the protein should be pure and have any residual fat removed. Proteins particularly useful in this mixture include animal proteins, such as albumin, casein, gelatine, peptone, hemoglobin or wool protein, and vegetable proteins such as protein from maize (zein), wheat (glutin and gliadin), soybean, or groundnut. Protein acid and protein enzyme hydrolysates, particularly casein acid hydrolysate and casein enzyme hydrolysate, are also suitable. The smoke of cigarettes prepared from the tobacco substitute had a tobacco-like flavor and aftertaste.

80-0630. Anderson, R. C.; Herd, A. J. **Smoking Mixtures.** United States Patent No. 3,943,942, March 16, 1976, 5 pp.

This patent describes a thermally degraded carbohydrate tobacco substitute containing sufficient alkaline inorganic matter such that mixing it with water to give a 15 percent solution/suspension results in a solution with a pH in the range of 8.5 to 13. The preferred alkaline matter for the tobacco substitute is sodium carbonate which produces a pH of 9 to 10. Ammonia-producing substances may also be used. These substitutes are in a form suitable for blending with tobacco to increase the nicotine delivery in the tobacco smoke. Blends of tobacco with these substitutes gave more satisfaction to the smoker than comparable blends in which the tobacco substitute produced a solution with lower pH.

80-0631. Austin, P. R. **Chitin as an Extender and Filter for Tobacco.** United States Patent No. 3,987,802, October 26, 1976, 4 pp.

Chitin (a mucopolysaccharide, believed to be poly-N-acetyl-D-glucosamine), either as such or in toasted form, has been found to be an effective extender and filter for tobacco. It can be used in substantial amounts with tobacco blends without adversely affecting such physical properties as packing ability, burning rate, or retention of ash. Organoleptic properties such as aroma, taste, and smoothness are little affected. Although smoking mixtures may contain from 5 to 75 percent chitin, those having 10 to 35 percent were found to give the best balance of physical and organoleptic properties. To the degree that chitin is used in the mixture, nicotine and noxious tars are reduced. The chitin modified tobaccos are themselves good filters for the smoke; the unburned mixture acts as a filter, or a cigarette containing the mixture may be used in a pipe or cigarette holder designed to hold such a filter. The chitin mixtures have special aptitude for absorbing phenolic, acidic and other organic compounds of types found in tobacco smoke and hence make effective filter compositions alone, or as filter tips or other filter elements. (Auth. Abs. Mod.)

80-0632. Beringer, M.; Buchmann, P. **Process for the Manufacture of Regenerated Tobacco.** United States Patent No. 3,929,141, December 30, 1975, 5 pp.

A homogeneous regenerated tobacco of reduced nicotine content is composed of 5,000 g of filler consisting of one or more of gramineous plants or shells of nuts, cocoa beans, or coffee beans; 3,000 to 5,000 g tobacco; 600 g magnesium formate; 50 g tartaric acid; 300 g potassium nitrate; 1,000 g paraffinurea; 300 g diammonium hydrogen phosphate; 10 g vanillylideneurea; 1,500 g sodium carboxymethylcellulose; 1,400 g glycerine; 150 g diethylene glycol; 1,050 g fruit concentrate; 600 g raw molasses; and 105 g malt extract. The components of the filler may be varied in the following ways: (1) 4,250 g wheat chaff, 500 g coconut shells, 250 g cocoa bean shells; (2) 2,500 g rice chaff and 2,500 g coffee bean shells; and (3) 5,000 g of rice chaff. Some of the smoking mixtures described include asbestos. (Auth. Abs. Mod.)

80-0633. Beringer, M.; Sporri, H. **Continuous Process for the Production of a Paste With Additives Which Can Be Formed Into a Smokable Material.** United States Patent No. 4,058,129, November 15, 1977, 6 pp.

A continuous simplified process for the production of artificial tobacco from pulverized plant parts (tobacco or grain), liquid, and additives is described. The liquid is forced into a grinding chamber simultaneously but separate from the plant powder to form a paste which is continuously removed to be shaped and dried. This process eliminates the need for making a paste from the plant parts prior to grinding. (Auth. Abs. Mod.)

80-0634. Borthwick, J.; Morman, J. F. **Smoking Mixture.** United States Patent No. 4,033,359, July 5, 1977, 5 pp.

This patent describes a smoking mixture which contains a minimum amount of tobacco, a maximum amount of inorganic filler (one or more inorganic salts) and a binder (carboxylated carbohydrates). The tobacco content of the mixture may vary from 15 to 80 percent by weight of the mixture (preferably 20 to 30 percent) and the filler content from 15 to 65 percent (preferably 40 to 60 percent). The composition of the filler is selected by balancing its ion content in order to give a commercially acceptable burning rate to the mixture. The inorganic filler may comprise salts like calcium carbonate, calcium borate, sodium borate, calcium orthophosphate, magnesium carbonate, calcium sulphate, calcium oxalate, iron sulphate, titanium dioxide, magnesium aluminate or sodium aluminium silicate or mixtures thereof. Tests carried out on these smoking mixtures showed a reduction in the amount of benzopyrene and other harmful chemicals in the smoke due to the reduced tobacco content of the mixture.

80-0635. Borthwick, J.; Morman, J. F. **Smoking Mixture.** United States Patent No. 4,008,723, February 22, 1977, 10 pp.

A smoking mixture based on tobacco substitutes is described which contains a minimum amount of organic combustible material and a maximum amount of harmless inorganic filler to reduce the amount of harmful products produced by combustion of the mixture. The organic combustible material, such as carbohydrates, modified carbohydrates, and aldol condensation products, preferably comprises 20 to 30 percent by weight of the smoking mixture and the inorganic filler 40 to 60 percent by weight. The composition of the filler is chosen to give the mixture a commercially acceptable burning rate.

80-0636. Boyd, D.; Porter, G. **Synthetic Smoking Product.** United States Patent No. 4,044,777, August 30, 1977, 4 pp.

A fuel for use in a smoking product consists of a fibrous, flexible, self-coherent carbonaceous material. This material is at least 80 percent carbon by weight, the remaining composition being hydrogen or oxygen. The fuel fibers aggregate into clusters. Volatile constituents to give smoking satisfaction are added to the fuel and are distilled or sublimed into a smoke stream upon burning. In one experiment, a smoking material produced from heavy weave cotton fabric was treated with a glow retardant and packed into a cigarette form made of slow-burning cigarette paper and a cellulose acetate filter. The total volatile organic phase of cigarette smoke was 4.3 percent of that found in a similar analysis of a standard cigarette made from flue-cured tobacco.

80-0637. Briskin, T. S. **Smokable Material and Method for Preparing Same.** United States Patent No. 4,019,521, April 26, 1977, 4 pp.

This patent describes a smoking material which is prepared by thermally degrading a cellulosic material at a temperature between 150° and 750°C (preferably between 400° and 700°C) in an inert, nonoxidizing atmosphere such as nitrogen gas. The material is heated until it experiences a weight loss of more than 50, but less than 90 percent (preferably at least 60, but no more than 80 percent). The thermally treated product can be compounded alone or in combination with cured tobacco and with other additives to provide a smoking product which can be used in the fabrication of cigars and cigarettes.

80-0638. Bustamante, C. R. **Tobacco Substitute Made From Coffee Cherries and a Process for Making Such.** United States Patent No. 4,165,752, August 28, 1979, 5 pp.

A method for making a tobacco substitute from the whole coffee cherry is described. The process consists of successively dehydrating the entire coffee cherry, grinding the entire cherry into particles, and roasting or processing the particles to form a coffee product. This product is then hydrated and air dried. At this time, the product is in the form of granules or shreddable sheets which may be either placed into cigarettes or pipes and smoked, or mixed with tobacco.

80-0639. Cook, L. C. **Smoking Compositions.** United States Patent No. 3,978,866, September 7, 1976, 5 pp.

A new smoking material is described which consists of peanut shells with or without tobacco. The use of peanut shells as a tobacco extender serves to reduce the amount of tobacco as well as the level of nicotine in the smoking product without adversely affecting the taste and aroma of the mixture. The amount of the peanut shell extender, which is blended with the tobacco, can vary widely from about 1 to 50 percent or more by weight of the tobacco. For cigarettes, it is generally preferable, however, that the peanut shell material be used in an amount ranging from about 1 to 25 percent by weight of the tobacco. When peanut shells are used alone as a tobacco substitute, the burning rate, flavor, and other properties of the smoking product can be altered by incorporating suitable additives such as flavorants, tobacco extracts, nicotine, humectants, and ash improving additives.

80-0640. Hedge, R. W. **Smoking Materials.** United States Patent No. 4,109,664, August 29, 1978, 6 pp.

A tobacco substitute containing a binder and an inert water-insoluble inorganic diluent is described. The binder consists of either a substituted thermo-gelling cellulose or methyl cellulose, both of which contain at least 1.5 methoxyl groups per anhydroglucose unit. The diluent or filler is calcium carbonate, calcium sulfate, calcium phosphate, aluminum trihydrate, or an aluminate. The smoke composition may also contain caramel or tobacco extract. Further, it may include a humectant such as glycerol.

80-0641. Maeda, K.; Noguchi, K.; Kawabata, M.; Sato, S.; Sato, Y. **Tobacco Product Containing a Thermo-Gelable β-1,3-Glucan-Type Polysaccharide.** United States Patent No. 4,109,663, August 29, 1978, 13 pp.

A smoking tobacco product, which is produced by incorporating in smoking material a thermo-gelable β-1,3-glucan-type polysaccharide, either as the sole smoking material or as its partial replacement, has improved smoking characteristics (flavor, taste, irritability) and improved physical properties (thickness, wet-proof qualities, tensile strength, elongation, filling capacity). The polysaccharide contains no nicotine and produces less tars than tobacco shreds. The polysaccharide may be added to smoking material before, during, or after processing. In sheet tobacco manufacturing, the polysaccharide may be added directly to the tobacco or indirectly via a sheet-like gelatinous product jointed to the sheet tobacco. In the case of cigarettes, the polysaccharide may also be coated on wrapping paper or incorporated in the paper adhesive. Approximately 0.1 to 10 percent by weight of the polysaccharide is used for improvement of the smoking characteristics, approximately 0.5 to 15 percent by weight is used for improvement of the physical properties, and approximately 1 to 30 percent by weight is used for improvement of the binding action and other properties of reconstituted or synthetic tobacco. (Auth. Abs. Mod.)

80-0642. Martin, R. H.; Owens, W. F., Jr.; McCarty, S. W. **Tobacco Substitute.** United States Patent No. 3,993,082, November 23, 1976, 4 pp.

A paper tobacco substitute which produces a neutral taste when blended with tobacco is comprised of cellulose fibers and a filler. The filler contains at least 55 percent finely pulverized carbon particles based on the weight of the cellulose. In addition to carbon, the filler may include an alkaline earth metal carbonate or an oxide such as calcium carbonate or magnesium oxide to further enhance the properties of the paper as a tobacco substitute. The carbon-filled paper may be cut or shredded to simulate natural tobacco and can be used as a blend with tobacco in smoking articles. (Auth. Abs.)

80-0643. Morman, J. F.; Parker, J. B. **Smoking Material.** United States Patent No. 4,014,349, March 29, 1977, 5 pp.

A process for the manufacture of a tobacco substitute is described, in which carbohydrates (preferably cellulose) are subjected to catalytic thermal degradation at a temperature above 250°C (preferably 250°-300°C) until the weight of the degraded carbohydrate residue is 50 to 90 percent (preferably 65 to 90 percent) of the weight of the original carbohydrate. The residue, which may be bleached, is combined with a binder and processed into a material simulating tobacco. Catalysts used may be acidic or basic in nature; strong acids and salts of strong acids are preferred. British Patent No. 1,113,979 describes a similar process for the preparation of carbohydrates at temperatures of 100° to 250°C. Smoking materials manufactured by either process are superior in smoke quality and taste to the corresponding smoking products made from untreated carbohydrates, and they give a smoother, less irritating smoke.

80-0644. Prouse, R. E.; West, A. A.; King, D. A.; Poulson, R. **Smoking Materials.** United States Patent No. 4,005,718, February 1, 1977, 8 pp.

This patent describes a smoking material consisting of a base of water-insoluble calcium-alginate fibers and additional material which provides both a flavor-modifying and combustion agent. The additional material may consist of (1) tobacco alone; (2) cellulosic fibers (wood pulp), a coloring agent, nicotine supplied as the sulfate, citrate, malonate or malate, a flavoring agent, and an inorganic salt to sustain the burning of the smoking material; and (3) tobacco in combination with the above ingredients. Where tobacco is added, its proportion should not be more than 55 percent by weight. If the mixture includes no tobacco, the proportions should preferably be over 80 percent calcium-alginate fibers, less than 10 percent cellulosic fibers, and not more than 10 percent other ingredients.

80-0645. Prouse, R. E.; West, A. A.; King, D. A.; Poulson, R. **Smoking Materials.** United States Patent No. 3,951,155, April 20, 1976, 7 pp.

A smokeable material is described which consists of a base material such as calcium-alginate fibers, and additional material which renders the substitute suitable for replacement in smoking tobacco. The additional material may consist of tobacco alone, but can include cellulosic fibers (wood pulp), a coloring agent, a nicotine donor, flavor-modifiers, and a burning sustainer. Calcium-alginate fibers will papermake in the traditional manner used for nonsynthetic fibers, and the additional materials described above can be incorporated during such papermaking. Any incorporated tobacco is 55 percent or less, by weight, added by blending or otherwise. Where there is no tobacco, there may be at least 80 percent of calcium-alginate fibers, and 10 percent each of the cellulosic fibers, and total other constituents by weight. Tobacco and tobacco substitute cigarettes were smoked by a smoking machine, and the particulate matter from the smoke was collected on a fiberglass filter pad. Total particulate matter from tobacco (control) vs. tobacco substitute cigarettes was 29.9 vs. 5.8 mg/cigarette. On combustion the tobacco substitute produces so little smoke for a given weight of material as compared with tobacco that it is free from pungent acrid or objectionable burning odors.

80–0646. Rainer, N. B.; Majewski, T. E. **Process for the Manufacture of Synthetic Smoking Materials.** United States Patent No. 4,079,742, March 21, 1978, 8 pp.

An improved process for the manufacture of synthetic smoking materials is described. The resultant product has a tobacco-like brown color, is not brittle, and has improved smoking characteristics. The cellulosic materials which may be utilized include materials of natural vegetable origin which are primarily but not necessarily composed of cellulose. Such materials include wood, preferably in shredded form, such as excelsior, as well as cotton, jute, ramie, sisal, hemp, and tree bark. (Auth. Abs. Mod.)

80–0647. Roth, D. S. **Tobacco Substitute Having Improved Ash Characteristics.** United States Patent No. 4,119,104, October 10, 1978, 8 pp.

This patent describes tobacco substitutes which eliminate excessive blooming (expansion) of cigarette ash. The substitutes consist of an organic binder; an inorganic filler; a boric oxide, boron oxyacid, or boron oxyacid salt as an ash stabilizer; and tobacco fines (10 to 40 percent by weight) as a bloom control agent. The substitute may also include a burn rate accelerator such as sodium nitrate, sodium citrate, or potassium tartrate in proportions up to 10 percent by weight. These tobacco substitutes may be mixed with natural tobacco in amounts up to 50 percent by weight. (Auth. Abs. Mod.)

80–0648. Strubel, D. G.; Johnson, R. R. **Tobacco Substitute Containing Boric Oxide, Boron Oxyacids, and Ammonium, Alkali Metal, or Alkaline Earth Metal Salts of Boron Oxyacids.** United States Patent No. 4,019,520, April 26, 1977, 5 pp.

Tobacco substitutes composed of an organic binder containing boric oxide or boron oxyacids and their ammonium, alkali metal, and alkaline earth metal salts are described. The tobacco substitute may be comprised of 2 to 95 percent by weight boric oxide, boron oxyacid, or boron oxyacid salt and from 5 to 50 percent by weight of the organic binder where the boron compound melts or fuses to form a stable ash during combustion. The tobacco substitute may also optionally include up to 93 percent by weight of a filler such as calcium carbonate, magnesium carbonate, charcoal, alumina, or alumina trihydrate, and up to 15 percent by weight monoammonium or diammonium phosphate. (Auth. Abs. Mod.)

80–0649. White, J. L. **Compositions for Smoking.** United States Patent No. 3,964,494, June 22, 1976, 4 pp.

Puffed milo (*Sorghum vulgare*) is used as a smoking material. When used with tobacco, puffed milo is blended with tobacco and the blend is then processed to form tobacco products. For example, puffed milo is shredded to a size approximating that of cut filler tobacco with which it is blended. The blend is then processed in conventional cigarette making machines to form cigarettes of either filter or nonfilter type. Additive materials such as flavorants, humectants, ash improvers, combustion modifiers, and fillers can be incorporated with the tobacco/puffed milo blends. The use of puffed milo with tobacco in a smoking product reduces the amount of tobacco employed with a concomitant decrease in tar and nicotine. This is achieved without adverse effect on the taste or aroma of the final product. The amount of the puffed milo which is blended with tobacco can vary widely up to 50 percent or more by volume of the tobacco.

80–0650. White, J. L. **Compositions for Smoking.** United States Patent No. 3,964,496, June 22, 1976, 4 pp.

Puffed rice is employed as a tobacco substitute by itself or with other nontobacco materials to form smoking products such as cigarettes, cigars, and pipe smoking products. The burning rate, flavor, and other properties of nontobacco smoking products can be altered by incorporating with the puffed rice suitable additives such as flavorants, tobacco extracts, nicotine, humectants and ash improving additives. The burning rate of puffed rice is somewhat faster than most natural tobaccos and accordingly, to decrease the burning rate of nontobacco smoking products, suitable filler materials or combustion modifiers such as magnesium carbonate, calcium carbonate, potassium carbonate, sodium carbonate, magnesium nitrate, and calcium nitrate can be incorporated with the puffed rice. When used with or without tobacco to form smoking materials, the puffed rice is conditioned to a moisture content of 6 to 15 percent to form smoking products. The use of puffed rice with tobacco serves to extend the tobacco or to reduce the amount of tobacco employed in a product with a concomitant decrease in the amount of tar and nicotine. This is achieved without adverse effect on the taste or aroma of the final product. The amount of the puffed rice which is blended with tobacco can vary widely up to about 50 percent or more by volume of the tobacco.

80–0651. White, J. L. **Smoking Compositions.** United States Patent No. 3,964,495, June 22, 1976, 5 pp.

Corn in an expanded condition and of relatively low density is used as smoking material. The patent describes a smoking mixture of tobacco and popped corn, as well as a smoking product consisting of popped corn alone or in a combination with other nontobacco materials. The use of popped corn with tobacco serves to extend the tobacco or reduce the amount of tobacco employed in a product with a concomitant decrease in the amount of tar and nicotine. This is achieved without adverse effect on the taste or aroma of the final product. The amount of the popped corn which is blended with tobacco can vary widely up to about 50 percent or more by volume of the tobacco.

80–0652. White, J. L. **Smoking Materials.** United States Patent No. 3,964,498, June 22, 1976, 4 pp.

This patent describes a smoking mixture of tobacco and puffed millet as well as a smoking product consisting of puffed millet alone or in combination with other nontobacco materials. The use of puffed millet with tobacco serves to extend the tobacco or reduce the amount of tobacco employed in a product with a concomitant decrease in the amount of nicotine therein. This is achieved without adverse effect on the taste or aroma of the final product. The amount of the puffed millet which is blended with tobacco can vary widely up to about 50 percent or more by volume of the tobacco.

80–0653. White, J. L. **Smoking Compositions.** United States Patent No. 3,964,497, June 22, 1976, 4 pp.

The use of puffed wheat with tobacco serves to extend the tobacco or reduce the amount of tobacco employed in a product with a concomitant decrease in the amount of nicotine. This is achieved without adverse effect on the taste or aroma of the final product. The amount of the puffed wheat which is blended with tobacco can vary widely up to about 50 percent or more by volume of the tobacco. Puffed wheat can also be employed as a tobacco substitute by itself or with other nontobacco materials to form smoking products such as cigarettes, cigars and pipe smoking materials.

80–0654. White, J. L. **Smoking Materials.** United States Patent No. 3,977,412, August 31, 1976, 4 pp.

Triticale is a wheat and rye hybrid. Puffed to a low density (approximately 0.20 gm/cm^3) the triticale may be cut, sliced, or shredded to a size and shape similar to filler tobacco and blended. The amount blended can vary up to more than 50 percent by volume.

80–0655. White, J. L. **Smoking Materials.** United States Patent No. 3,977,413, August 31, 1976, 4 pp.

Puffed barley serves as a tobacco extender with no significant effect on the organoleptic properties of the tobacco products in which it is present. After shredding to cut filler tobacco size, the puffed barley is added in amounts of 50 percent or more by tobacco volume.

80–0656. White, J. L. **Smoking Materials.** United States Patent No. 3,977,414, August 31, 1976, 4 pp.

The use of puffed oats as a tobacco substitute is described. The oats are shredded to the size of cut filler tobacco and blended with tobacco in proportions up to 50 percent or more by volume. The nicotine-free puffed oats are easily processed and do not detract from the aroma or the taste of the tobacco product.

80–0657. White, J. L. **Smoking Compositions.** United States Patent No. 3,977,415, August 31, 1976, 4 pp.

Puffed rye is used alone or with tobacco to provide smoking materials. The amount blended with tobacco varies widely up to 50 percent or more by tobacco volume. The use of puffed rye with tobacco permits a significant reduction in the amount of tobacco used and in the reduction of nicotine.

80–0658. Wood, W.M.L. **Smoking Mixtures.** United States Patent No. 4,117,850, October 3, 1978, 7 pp.

This patent describes a smoking material fortified with nicotine. The mixture consists of smoke-producing fuel (tobacco or tobacco substitutes); nicotine, calcium and/or magnesium carbonate as incombustible filler; and at least one water-soluble salt to inhibit dissolution of the carbonate in water. In addition, the mixture contains a phosphate buffer which consists of an acid salt of orthophosphoric or polyphosphoric acid in an amount sufficient to improve the nicotine stability and to maintain the pH of a 15 percent aqueous solution of the smoking material between 4 and 6. Nicotine incorporated in the smoking materials may be in the form of the free base or an organic acid salt, or in the form of a tobacco extract. Nicotine may also be added in the form of high nicotine-containing tobacco, such as *Nicotiana rustica*, or as a callus tobacco made by cell culture in a synthetic medium containing sugar. The proportion of nicotine (calculated as free base) in the smoking materials ranges from 0.1 to 5 percent by weight, depending upon the degree of fortification required.

80–0659. Yokota, H.; Namikoshi, H.; Watanabe, M.; Kato, K.; Ohnishi, A. **Smoking Composition.** United States Patent No. 4,068,670, January 17, 1978, 7 pp.

A smoking composition is described which includes an ester of one or more aliphatic acids and a carbohydrate such as cellulose or starch. This mixture may be used to replace all or a part of the natural tobacco as the principal filler for smoking compositions, and/or as a diluent for modifying aroma and taste, and/or as a water-resistant binder or reinforcement for smoking compositions. It was found that the smoking composition was very mild and nonirritating when smoked and that no unpleasant aftertaste remained.

SMOKING CESSATION AIDS

80–0660. Adamo, C. **Time-Delayed Single Cigarette Dispenser.** United States Patent No. 4,010,869, March 8, 1977, 4 pp.

A cigarette dispenser is described which rations cigarettes by only making them available one at a time at certain predetermined time intervals in order to help smokers reduce their cigarette consumption. The dispenser includes a magazine which rotates within a housing and numerous cigarette holding chambers. The housing has an opening which is aligned with one of the chambers during cigarette dispensation.

80–0661. Boroski, H. M.; Boroski, S. T. **Rationing Cigarette Dispenser.** United States Patent No. 3,999,412, December 28, 1976, 4 pp.

This patent describes a cigarette case with a cover secured with a latch which is opened only after a fixed interval of time by a clockwork mechanism. When the cover is opened, one cigarette is lifted out of the case and access to the cigarettes remaining in the case is prevented.

80–0662. Camps Mestre, J. M.; Tremoleda Dilmer, M. **Mouthpieces to Facilitate Giving up Smoking.** United States Patent No. 3,957,064, May 18, 1976, 4 pp.

This patent describes a mouthpiece which comprises an external air inlet, adjustable between zero and a maximum, which permits mixing the smoke drawn in by the smoker with a variable proportion of fresh air, reducing the toxicity of the mixture, and permitting the metabolism of the smoker to adjust to progressively decreasing nicotine levels. The mouthpiece contains a diluting chamber provided with one opening for the smoke coming from the cigarette and at least one other adjustable opening to the outside. In this way the proportion of fresh air coming from the outside, which in each case is mixed with the smoke drawn in by the smoker, can be regulated with full precision and in a perfectly safe manner. To conceal the air inlets from sight, a metal sleeve is used to envelop the diluting chamber; a pair of peripheral ribs constitute the only contact between chamber and sleeve.

80–0663. Corman, W. **Cigarette Case With Timer.** United States Patent No. 4,084,415, April 18, 1978, 3 pp.

A cigarette case with a timer to help the user in reducing his cigarette consumption is described. The timer, attached to the side of the case, has a dial which can be set by the user to any desired time interval and prevents opening of the case except after a predetermined interval of time. The cigarette case is inexpensive to make and easy to use.

80–0664. Di Costanzo, F. **Compositions for Combating Nicotinism in Man.** United States Patent No. 3,989,823, November 2, 1976, 6 pp.

Compositions are described for treating nicotinism, for modifying the taste and smell sensations of smokers, for smoking detoxification, and for treating withdrawal symptoms. These compositions contain (1) valerian extract as a sedative and an agent for changing the smoker's taste and smell, (2) vitamin B_1 for correcting hypoglycemia, (3) vitamin B_6 for treating neuritis and trembling due to smoking deprivation, and (4) either a sleeping agent for evening or night administration or ascorbic acid for daytime administration. The effect of the compositions was evaluated in toxicological and clinical studies. The effectiveness of the compositions in nicotine detoxification appears remarkable: (1) overall results are positive in 66 percent of the cases, (2) the desire to smoke is influenced in 70 percent of the cases, (3) the tranquilizing effect on the frustration syndrome is beyond doubt, (4) a favorable influence was observed in 75 percent of the cases, and (5) the clinical and biological tolerance of the compositions is perfect.

80–0665. Fox, A. F. **Safety Smoking Device.** United States Patent No. 3,955,585, May 11, 1976, 4 pp.

This safety smoking device is comprised of casing with a bottom wall, side and end flanges, and corner legs for seating the device on a table or other supporting surface. Projecting through the end wall is a cigarette holder which has a fitting at its outer end adapted for connection to a flexible hose with a smoking tip at its free end. Removable and disposable ashtrays are held under the cigarette holder. Also provided in the casing is a cigarette lighter and a compartment to store a package of cigarettes. The cigarette holder is held in the casing in an arrangement such that it cannot be removed by the smoker but is adjustable longitudinally to accommodate cigarettes of different lengths. The lid may be open or closed as desired, but the device has the advantage that the lid may be locked and the key removed if it is desired that the smoker not have access to the cigarette being smoked. Vents are provided to allow the desired circulation of air. (Auth. Abs. Mod.)

80–0666. Guerra, R. E. **Method and Apparatus for Breaking the Habit of Smoking.** United States Patent No. 4,068,672, January 17, 1978, 8 pp.

This patent describes a method and device for assisting a smoker in breaking his smoking habit by producing an electrical shock when a cigarette holder is placed between his lips. The holder contains a DC voltage source (such as a dry cell battery) of at least six and preferably nine volts which is rechargeable. The connectors to the battery extend on both sides of the cigarette holder to its mouthpiece end. Lip contact with the mouthpiece causes a closed circuit which delivers an electric shock. A potentiometer is optionally provided to adjust the flow of current from a minimum of about one milliamp (in order to be discernible) to a maximum of about five milliamps (so as to avoid intolerable sensations).

80–0667. Prugnaud, R. L. **Therapeutic Composition With Marked Polydetoxifying Properties, Especially With Regard to Tobacco.** United States Patent No. 4,057,620, November 8, 1977, 4 pp.

The composition described in this patent is comprised of an endogenous detoxifying substance, vitamins of groups B and C, an organic salt of quinine, and, optionally, a sympathoparalytic sedative. It may be formulated with conventional binders or pharmaceutically acceptable carriers. A preferred composition is composed of muramidase, nicotinamide, thiamine, ascorbic acid, pyridoxine, a quinine salt having an organic anion, and optionally the dry extract of Crataegus root. The composition is markedly effective in detoxification therapy for solanaceous and ganglioplegic toxins such as nicotine and related toxic compounds. The composition constitutes a synergistic entity. It permits the suppression of the use of tobacco in the intoxicated smoker, reestablishes the equilibrium of the smoker during his stage of tobacco deprivation, and combats the stress which results from the suppression of tobacco. In heavy smokers who were resistant to all types of therapies previously attempted, cessation of smoking is often observed in the first 15 days following the beginning of treatment. In other persons whose treatment required one month or more, it is necessary to provide a second cure. Among light smokers (one package of cigarettes per day), cessation of smoking in general is achieved in the first week of treatment. The number of failures observed has been small (only 15 percent for the entire group of patients). These failures were found to be due either to irregularity in following the treatment doses or to stopping treatment prematurely. The usual treatment duration with this composition is 1 month if the product is taken four times a day.

GENERAL

80–0668. Brantl, V.; Seeger, W. **Hand Operated Device For Producing Tobacco Articles Low in Noxious Substances.** United States Patent No. 4,120,309, October 17, 1978, 21 pp.

A portable manually operated device is described which enables a smoker to modify conventional tobacco articles (e.g., cigarettes) so that they produce only a small amount of noxious substances, particularly carbon monoxide, when smoked. The device consists of a holder for the tobacco article and a device for creating a tobacco-free space adjacent to the longitudinal axis of the tobacco article. An insert substantially impermeable to air is introduced into the tobacco-free space. When cigarettes are modified in this manner, the carbon monoxide content in the smoke is reduced by more than 50 percent if the diameter of the incorporated insert is approximately 3 mm and the cigarette has a diameter of 8 mm. It has also been found that the filter shows less brown discoloration after smoking than is the case with the filter of a nonmodified cigarette, indicating a lower proportion of discoloring condensate and therefore, presumably, also of nicotine.

80–0669. Claflin, W. E.; Watson, F. M., III. **Variable Parameter Smoking Machine.** United States Patent No. 4,019,366, April 26, 1977, 8 pp.

A smoking machine for smoking one or more cigarettes that allows adjustment of the puff flow, duration, and interval for the cigarettes being smoked is described. Previous devices, such as those shown in U.S. Patent Nos. 3,460,374 and 3,433,054 were successful in simulating human smoking; however, they generally had very restricted operational parameters. In this smoking machine, cigarette holders are placed in fluid circuit parallel with dummy air-flow resistance valves, and either the holder or dummy load is placed in communication with a continuous vacuum source by a solenoid-operated valve associated with each holder and dummy load. A flow-adjustable valve is also associated with each holder and dummy load. Each holder-dummy-valve system is connected to a pressure manifold, which in turn is connected through a circuit having a flowmeter and flowmeter bypass line arranged in parallel to the continuous vacuum source. (Auth. Abs. Mod.)

80–0670. Ensor, J. E. **Smoke Reducer for Cigarette Smokers.** United States Patent No. 4,066,088, January 3, 1978, 4 pp.

A lighter-extinguisher for cigarettes is described which permits smokers to enjoy smoking while reducing the possibility of annoying nonsmokers, wasting smoking materials, staining fingers, or scattering ashes. A cigarette-package-sized device with a cigarette mouthpiece end projecting from it is held in the smoker's hand at all times during smoking of the cigarette; between puffs the cigarette is extinguished by closing a valve to shut off air from the burning end held inside the device, and the cigarette is conveniently relit by opening the valve, which automatically activates the lighter. An ash pit receives ashes down a slide and the lighter mechanism follows the tip, assuring ready re-ignition. (Auth. Abs. Mod.)

80–0671. Fedorchenko, I. M.; Pugin, V. S.; Ablov, V.I.N.; Fridman, J. Y.; Goldberg M. S. **Mouthpiece for Smoking Tobacco.** United States Patent No. 4,011,877, March 15, 1977, 4 pp.

A mouthpiece for smoking tobacco and tobacco articles is described which comprises two filters for purifying tobacco smoke: a rough and a fine purification filter. The rough filter is intended to convert the resin vapors contained in the smoke into a liquid and to settle down the vapors on the inner surface of the body. The fine purification filter is fabricated from a porous sintered metal material which removes up to 80 percent of the toxic agents of the smoke (not less than 70 percent of the aromatic hydrocarbons, and not less than 40 percent of toxic agents such as DDT and its metabolites). (Auth. Abs. Mod.)

80–0672. George, P. F. **Smoking System.** United States Patent No. 4,037,610, July 26, 1977, 5 pp.

A tamper and disc for use in a pipe having a bowl with a cylindrical inner surface is described. The tamper has a holding portion and a tubular portion extending from the holding portion. The thin-walled tubular portion has an outer surface adapted to fit snugly into the inner surface of the bowl so that the tamper compresses only the tobacco adjacent to the inner surface of the bowl. The disc which rests in the bowl to support the tobacco has a central aperture. With this apparatus, the smoke results from the combustion of tobacco, not from the burning of accumulated tars and ash. Further, the tobacco burn rate is slower and the tobacco smoke is cooler. (Auth. Abs. Mod.)

80–0673. Goldstein, J. J. **Cigarette Filter Holder.** United States Patent No. 4,003,387, January 18, 1977, 4 pp.

A cigarette filter holder which includes a cigarette receiving socket at one end and a mouthpiece at the other end is described. The interior of the holder includes a fibrous filtering material impregnated with water. The mouthpiece end of the holder is formed with a removable plug, which can be twisted off to provide an opening to the interior of the cigarette filter. The use of multiple materials for sealing the mouthpiece end of the filter is thus eliminated. (Auth. Abs. Mod.)

80–0674. Heggestuen, J. E. **Smoking Device.** United States Patent No. 4,044,781, August 30, 1977, 4 pp.

Water pipes in the form of hookahs or narghilas are well known. They are functional and frequently ornate, but are large and heavy and generally consume a relatively large quantity of the herb being smoked at each lighting, of which the smoker actually gets the benefit of only a relatively small portion. The smoking device described in this patent retains the advantages of cooling and purification characteristic of a modest-sized water pipe. A visible element of the pipe is a hopper or canister. However, the combustion of the herb takes place in a relatively small bowl cavity filled from the hopper. The bowl cavity contains a quantity of the herb only sufficient to supply smoke for the interval of one long puff. At the end of the puff the ashes pass with the smoke into the liquid in the chamber, and the rising smoke is stored in an inhalation chamber. From there the smoke may be drawn in short successive inhalations, during which air is supplied to replace the smoke being withdrawn.

80–0675. Jacobsen, E. L. **Smoking Device.** United States Patent No. 4,083,374, April 11, 1978, 7 pp.

A pipe-like smoking device utilizing a bag which the smoker fills with smoke prior to inhaling is described. The tobacco is loaded into the pipe and lit when a valve in the smoking device is turned to a first position. In a second position, the stem of the smoking device is coupled to the bag through the smoldering tobacco, enabling the smoker to blow the smoke into the bag. When the valve is in a third position, the stem of the smoking device is connected directly to the smoked filled bag. The smoker can thus inhale the smoke and exhale it back into the bag. The smoke is thereby reused many times and maximizes the utilization of flavor in the smoke, while minimizing pollution of the environment. A fourth position may be provided where the flame of a match or lighter is used to clean a filter screen through which the smoke is forced into the bag. (Auth. Abs. Mod.)

80–0676. Kahler, R. W. **Liquid Filtered Smoking Device.** United States Patent No. 4,041,960, April 16, 1977, 6 pp.

A liquid-filtered smoking device is described. It consists of a container which has an opening, hollow at one end; a liquid filtering medium; and an open, hollow, cylindrical tube whose lower end extends through the peripheral wall of the container into the liquid filtering medium and whose upper end projects outwardly from the container. The device also consists of a smoking bowl which is secured upon the upper end of the tube and a filter which is inserted inside the bowl in order to prevent tobacco ash and unburnt or partially burnt tobacco particles from passing through the tube into the liquid filtering medium. The bowl may be either of the reversible or nonreversible type and the filter may be utilized in conjunction with both types of bowls. (Auth. Abs. Mod.)

80-0677. Kahler, R. W. **Fluid-Cooled Smoking Device.** United States Patent No. 4,029,109, June 14, 1977, 12 pp.

A fluid-cooled smoking device includes numerous concentrically disposed tubular members which, depending upon the predetermined length of some of such members, and/or the provision of apertures, together define an elongated serpentine fluid flow path. An ash receptacle is defined within the upstream portion of the fluid flow path while the downstream portion of the flow path includes a liquid chamber. The smoke is air-cooled within the upstream portion of the flow path, ash deposits are segregated from the fluid flow prior to the same being further cooled within the water chamber, and consequently, the water chamber does not readily become contaminated. (Auth. Abs.)

80-0678. Kahler, R. W. **Fluid-Cooled Smoking Device.** United States Patent No. 4,014,353, March 29, 1977, 11 pp.

A fluid-cooled smoking device includes a housing for containing a liquid coolant and a tubular member which projects into the housing to support a bowl-ash receptacle. Concentric tubular members within the ash receptacle portion of the assembly define a serpentine flow path where the smoke and fumes are initially air-cooled. Subsequently, the air-cooled smoke and fumes pass into the liquid coolant and then reach the smoker. The ash receptacle accumulates burnt tobaccos and ash deposits and prevents them from entering the liquid coolant. (Auth. Abs. Mod.)

80-0679. Murray, J. E., Jr. **Smoking Apparatus With Replaceable Filtering Cartridge.** United States Patent No. 4,036,240, July 19, 1977, 6 pp.

A smoking apparatus comprised of a pipe bowl, a hollow pipe stem, and a pipe body is described. The pipe body has a detachable base and contains a replaceable filtering cartridge. The cartridge holds a liquid for filtering, cooling, and/or flavoring the smoke flowing through it. The cartridge has two smoke ports, each having one tubular extension. The first extension goes below the top surface of the liquid when the apparatus is upright and otherwise is above that surface. The second extension is always above the top surface of the liquid. The smoke path of this apparatus consists of the bowl, the first smoke port, the first extension member, the liquid, the second extension member, the second smoke port means, and the pipe stem. (Auth. Abs. Mod.)

80-0680. Orel, J. V. **Smoke Sorbing Device.** United States Patent No. 4,043,776, August 23, 1977, 8 pp.

Various devices have been developed for filtering the smoke emanating from cigarettes or other tobacco products resting in an ashtray. Such a device was disclosed in U.S. Patent No. 3,516,232, issued on June 23, 1970. This device consisted of a stand supporting an ashtray, a housing, and a motorized fan above the ashtray. The housing defined a passageway, and the associated fan generated a flow of air through the passageway. The housing also supported a filter between the fan and the ashtray. Such devices have been elaborate, expensive, bulky, and ineffective. This patent describes a device for filtering the smoke commonly associated with cigarettes and cigars and includes an ashtray which supports a shroud for confining the smoke. The shroud supports a motorized fan which creates a flow of air to carry the smoke through a filter. A motor for driving the fan is removed from the flow of air to prevent contamination of the motor by the smoke fumes. An elongated filter and a cylindrical filter have cartridge configurations to facilitate the removal and replacement of the filters. An absorbent material impregnated with a perfume enhances the fragrance of the air flow through the device. (Auth. Abs. Mod.)

AGRICULTURE

80-0681. Gopalachari, N. C.; Reddy, M.C.M. **Effect of Plant Density on Certain Chemical and Physical Characteristics of F.C.V. Tobacco Grown in the Northern Light Soils of Andhra Pradesh.** *Tobacco Research* 4(2): 59-64, December 1978.

Three plant densities (16,666, 20,833, and 27,777 plants/hectare (ha)), 3 fertilizer levels (30 N : 80 P_2O_5 : 80 K_2O; 37.5 N : 100 P_2O_5 : 100 K_2O; and 45 N : 120 P_2O_5 : 120 K_2O kg/ha) and two varieties (Kanakaprabha and HR 65-35) were compared for certain chemical and physical characteristics in a field experiment conducted from 1971 to 1974 in the Northern Light Soils of Andhra Pradesh. None of the different plant densities, fertilizer levels, and varieties affected the chemical and physical characteristics and quality ratios of flue-cured tobacco beyond the acceptable limits. The lowest fertilizer level resulted in a significantly lower nicotine content than the other levels. Intermediate plant density (20,833 plants/ha) resulted in a significantly higher nicotine content than the other densities. Variety Kanakaprabha had higher levels of reducing sugars and nicotine than HR 65-35, but the latter had a higher filling value and pore volume. Wherever nicotine content varied significantly, the ratio of reducing sugars/nicotine changed in the reverse direction. Increasing plant densities and fertilization levels within the limits studied did not significantly alter the quality of the cured leaf. (Auth. Abs. Mod.)

80-0682. Jorgovic-Kremzer, J. **Uticaj Prskanja Cinebom na Sadrzaj i Distribuciju Cinka u Pojedinim Organima Biljke Duvana. [Effect of Zineb Spraying on Zinc Content and Distribution in Organs of the Tobacco Plant.]** *Agrohemija* (9/10): 357-365, 1975, Serbo-Croatian.

Tobacco plants of the "Prilep" type were sprayed once with 0.4, 0.6, or 0.8 percent zineb suspensions in an experiment designed to explore the possibility of foliar feeding by spraying. Zinc (Zn) content of the collected plant organs was determined by atomic absorption spectroscopy. The plant samples were prepared for analysis by drying and by mineralizing the dried samples with a mixture of nitric and perchloric acids. The sensitivity for Zn determination was 0.009 μg/ml. Zinc concentration in the organs of sprayed plants was found to be higher than in the controls. However, distribution of Zn in the plants' organs was unequal. The highest concentrations (2.14 to 3.82 ppm) were found in leaf blades of the older leaves from the middle of a plant. The Zn content decreased toward the top of the plant (stem and flower) and toward its base to less than 1 ppm in the root. The highest increase (11.0 to 20.4 ppm) in Zn concentration after spraying occurred in the leaf blades of the middle leaves. Smaller increases were found in the upper leaf blades and stems of the middle leaves. There was no significant difference between Zn concentrations in the organs sprayed with 0.6 and 0.8 percent suspensions. It is concluded that Zn from zineb has only a limited value as foliar nutrient of the plant. Shortened flowering time and varied number of leaves in zineb-sprayed plants indicated an unspecified physiological effect on plant processes in addition to its fungicidal effect.

80-0683. Ogonowski, J. W.; Rudra, L. **The Yield, Quality and Chemical Composition of Flue-Cured Virginia Tobacco at Two Locations in Zambia.** *East African Agricultural and Forestry Journal* 41(4): 298-303, April 1976.

The yield, grade index, and chemical composition of six flue-cured Virginia varieties, Kutsaga 51 (K51), Hicks, White Gold (WG), Zambia Mammoth (ZM), Kutsaga E1 (KE1), and Kutsaga E2 (KE2) were compared at Kabwe and Mochipapa in Zambia. The yield of cured tobacco at Kabwe was significantly higher than that at Mochipapa with lower grade index. Total nitrogen, nicotine, potassium, calcium, and magnesium percentages were higher, but reducing sugar and phosphorus were lower at Kabwe. The ZM variety contained the lowest percentage of nicotine and produced the highest yield at both the sites. KE1 and KE2 produced good yields and the best qualities and had the highest concentrations of sugar. Hicks at Mochipapa produced the lowest yield and quality with the lowest sugar and highest percentage of nicotine. There was a large seasonal variation in yield, grade index, and chemical composition of the varieties tested. (Auth. Abs.)

80-0684. Saleh, M. M.; Makarem, M.; El-Gamasy, A. M. **Effect of Irrigation on the Growth, Alkaloids and Rutin of *Nicotiana glauca*.** *Acta Horticulturae* 73: 199-203, May 1978.

To study the effect of varying irrigation intervals on the growth, alkaloid, and rutin content of tobacco, *Nicotiana glauca* plants were irrigated every 10, 20, 30, or 40 days. The fresh weight of the leaves was increased by lengthening intervals between irrigation, so that the weight of the leaves of plants irrigated every 40 days was more than double the weight of leaves irrigated every 10 days. The percentage of alkaloids in the leaves was considerably higher during 30- or 40-day than during 10-day irrigation intervals. The highest leaf content of rutin was found when plants were irrigated every 10 days.

80-0685. Singh, B. L.; Tripathi, S. N.; Dubey, R. S.; Pandey, R. G. **Effects of Different Crop Rotations on the Yield and Quality of Cigar Wrapper Tobacco in North Bengal.** *Tobacco Research* 4(2): 50-53, December 1978.

Studies on different crop rotations in North Bengal revealed that growing wrapper tobacco in a crop rotation, i.e., maize-paddy-wrapper tobacco or jute-paddy-wrapper tobacco, was most remunerative. These rotations gave an additional 4000-5500 kg/hectare yield of paddy. Cultivation of jute in the pre-kharif season was found to be more profitable compared to other competitive crops, like paddy, jowar, and maize. (Auth. Abs.)

80–0686. Sinha, R.; Dwivedi, S.S.L.; Pandey, A. K. **Effect of Source and Time of Nitrogen Application on Chewing Tobacco and the Succeeding Maize in North Bihar.** *Tobacco Research* 5(1): 70-76, June 1979.

Experiments to study the effect of source and time of nitrogen application on chewing tobacco (Gandak Bahar variety) and its succeeding maize were conducted at the Tobacco Research Station, Pusa (Bihar), between 1973 and 1977. Yield of total cured (27.7 q/hectare) and first grade (12.9 q/hectare) tobacco leaf and succeeding maize (37.9 g/hectare), net return per hectare and return per rupee investment from tobacco as well as from tobacco plus succeeding maize were highest when 75 percent of the total nitrogen (168 kg/hectare) in inorganic form was applied at the time of tobacco planting and 25 percent at growth initiation, i.e., 30 days after planting over the basal application of farmyard manure 1 month before tobacco planting. Nicotine percent and chewing quality of tobacco were almost the same whether nitrogen was applied in inorganic form alone or in combination with mustard cake as organic manure. The practice of using mustard cake to supply nitrogen along with ammonium sulphate in tobacco is uneconomical. The total nitrogen can be safely and economically provided through inorganic nitrogenous fertilizer without affecting the yield and quality of tobacco and the succeeding maize in rotation. (Auth. Abs.)

80–0687. Yoshida, D. **Effect of Cultural and Curing Practices of Tobacco on the Contents of Tar in the Smoke.** *Tobacco Research* 4(2): 1-6, December 1978.

The effect of tobacco variety, topping, rate of fertilization, leaf maturity, and method of curing on the tar content in cigarette smoke was studied in Japanese domestic and Burley tobacco. Two domestic air-cured varieties delivered less tar in the smoke than two flue-cured varieties. The tar content in the smoke of flue-cured tobacco decreased with no topping and with increased rate of nitrogen fertilization, but increased gradually with the advance in leaf maturity. When the same variety was subjected to flue- and to air-curing, the results showed that the curing method did not affect the tar content of the smoke. The tar contents in the smoke showed no correlation with the physical properties of leaves, such as weight of unit leaf area, bulkiness and pore content of leaf, but did show a significant correlation with the content of alcohol—benzene extractable components in the leaves. The amount of extractable components in the leaves increased with the degree of leaf maturity.

See also, **80–0703**

CHEMISTRY

80–0688. Behr, D.; Wahlberg, I.; Nishida, T.; Enzell, C. R. **(3*S*,6*R*,7*E*,9*R*)- and (3*S*,6*R*,7*E*,9*S*)-4,7-Megastigmadiene-3,9-diol. Two New Nor-Carotenoids of Greek Tobacco.** *Acta Chemica Scandinavica. Series B* 32(6): 391-394, 1978.

Two new C_{13} nor-carotenoids were isolated from Greek *Nicotiana tabacum* L. and shown by spectroscopic methods and synthesis to be the (3*S*,6*R*,7*E*,9*R*)- and (3*S*,6*R*,7*E*,9*S*)-4,7-megastigmadiene-3,9-diols. The sample of 3-oxo-*α*-ionol, previously isolated from Greek tobacco, was reinvestigated and found to contain (6*R*,7*E*,9*R*)-9-hydroxy-4,7-megastigmadien-3-one as the major component and the corresponding, partially racemic (6*R*,7*E*,9*S*)-isomer as the minor component. (Auth. Abs.)

80–0689. Behr, D.; Wahlberg, I.; Nishida, T.; Enzell, C. R. **(2*E*,6*S*)-2,6-Dimethyl-2,7-Octadiene-1,6-Diol, a New Monoterpenoid from Greek Tobacco.** *Acta Chemica Scandinavica. Series B* 32(3): 228-229, 1978.

Recent investigations have demonstrated the presence of a large number of isoprenoids in tobacco. Although the majority of these belong to the groups of nor-compounds derived from diterpenoids and carotenoids, they also include several mono-, sesqui-, di-, tri-, and polyisoprenoids. The present report describes the structure determination and synthesis of a new linear monoterpene diol isolated from Greek tobacco. (Auth. Abs.)

80–0690. Chakraborty, M. K.; Gopalam, A. **Polyphenol Content in Tobacco Cultivars.** *Tobacco Research* 5(1): 54-60, June 1979.

Total polyphenols, chlorogenic acid, and rutin have been determined in 46 tobacco cultivars, both in transplantable seedlings and in cured leaves. A wide range of polyphenols were obtained in various cultivars. The general level of polyphenols was higher in cured leaves than seedlings, indicating their biosynthesis during growth and maturation.

Tobacco cultivars having lower polyphenols, namely flue-cured varieties—Virginia Gold and Dhanadayi, improved lines like 72-67, 72-68, plus several resistant lines TMVRR-3a, TMVRR-2a, TMVRR-2, and 2658a—are suggested as important for their less carcinogenic precursory polyphenol compounds. Resistance to tobacco mosaic virus is found to be independent of the levels of polyphenols in tobacco. (Auth. Abs. Mod.)

80-0691. Collins, P. F.; Williams, J. F. **A Unified Smoking-Automated Colorimetric System for the Analysis of the Gas Phase of Cigarette Smoke and Its Application to the Determination of Hydrogen Sulfide.** *Beitraege zur Tabakforschung International* 10(1): 24-30, December 1979.

A system has been developed wherein the gas phase produced on smoking a cigarette through a Cambridge pad is analyzed on a puff-by-puff basis for a desired component using automated colorimetry. The system consists of an AutoAnalyzer I and a simple 1-port smoking machine. The continuous gas stream from the smoking machine, either gas phase or intervening air, is passed concurrently with a stream of absorbing solution through a glass coil. Following passage through the coil, the solution is separated from the gas stream and introduced into the AutoAnalyzer for continuous measurement of the concentration of the absorbed component. The system has been utilized for the determination of hydrogen sulfide (H_2S) in gas phase using 0.1 N sodium hydroxide as the absorbing solution and the "methylene blue" colorimetric reaction. Results obtained on a variety of commercial U.S. brands ranged from 18 to 50 μg of H_2S per cigarette, similar to results reported by other investigators using wet chemical methods. The accuracy of the results is supported by recovery tests wherein gaseous H_2S added to the gas phase was completely recovered. The average relative standard deviations obtained with 11 U.S. commercial or reference cigarette samples using random selection of the cigarettes was 10.8 percent for μg of H_2S per cigarette and 8.8 percent for μg of H_2S per puff for a single cigarette. The basic system is thought to be applicable to the determination of a number of other components in gas phase. (Auth. Abs. Mod.)

80-0692. Dermelj, M.; Ravnik, V.; Byrne, A. R.; Vakselj, A. **Trace Heavy Metals in Various Yugoslav Tobaccos.** *Mikrochimica Acta* 1(3/4): 261-270, 1978.

Nine varieties of Yugoslavian tobacco were analyzed for the trace metals arsenic, cadmium, copper, mercury, iron, manganese, antimony, selenium, uranium, vanadium, and zinc. Analysis methods included γ-ray spectra of isolated radionuclides with a sodium iodide detector, and spectrophotometry. The results were compared with analyses of the standard Kentucky 1R1 tobacco.

80-0693. Ferguson, R. N.; Whidby, J. F.; Sanders, E. B.; Levins, R. J.; Katz, T.; DeBardeleben, J. F.; Einolf, W. N. **Isolation and Identification of 2,3-Dimethyl-6-(4,8,12-Trimethyltridecyl)-1,4-Naphthoquinone From Tobacco and Smoke.** *Tetrahedron Letters* (30): 2645-2648, July 1978.

A previously undescribed quinone has been isolated from low alkaloid bright tobacco filler as a result of continuing efforts to identify new components from tobacco and smoke. This compound, 2,3-dimethyl-6-(4,8,12-trimethyltridecyl)-1,4-naphthoquinone (solanoquinone), appears to be the first example of a naturally occurring quinone with a saturated polyisoprenoid side chain. (Auth. Abs.)

80-0694. Fujimori, T.; Kasuga, R.; Kaneko, H.; Noguchi, M. **Neutral Volatile Components of Burley Tobacco.** *Beitraege zur Tabakforschung International* 9(5): 317-325, December 1978.

The constituents of the neutral volatiles in air-cured Burley tobacco were studied, and 19 new substances were found. Among there were (1) 6-methyl-6,9-oxidonon-4-en-2-ol; (2) 5-isopropylnon-3-en-2,8-diol; (3) *o*-methyl acetophenone; and (4) 4-ethyl-4-methylbutan-4-olide. A large number of identified compounds may be degradation products of carotenoids and thunberganoids. These compounds have a characteristic aroma and are thought to be key flavor components in the essential oils of tobacco. The presence of any labdanoid hydrocarbons or their oxygenated products was not demonstrated in the Burley tobacco extract. Burley tobacco is thought to be deficient in labdanoid compounds. (Auth. Abs. Mod.)

80-0695. Fujimori, T.; Kasuga, R.; Kaneko, H.; Sakamura, S.; Noguchi, M. **Solanascone: A Novel Sesquiterpene Ketone From *Nicotiana tabacum*. X-Ray Structure Determination of the Corresponding Oxime.** *Journal of the Chemical Society. Chemical Communications* 13: 563-564, July 1978.

Solanascone, a new tetracyclic sesquiterpene ketone, has been isolated from *Nicotiana tabacum* cv. Burley; its structure was confirmed by X-ray analysis of its corresponding oxime. (Auth. Abs.)

80-0696. Going, R. E.; Hsu, S. C.; Pollack, R. L.; Haugh, L. D. **Sugar and Fluoride Content of Various Forms of Tobacco.** *Journal of the American Dental Association* 100(1): 27-33, January 1980.

This study identifies the total sugar and the fluoride concentrations of various forms of tobacco from different areas of the United States. The total sugar content of pouch and plug forms of tobacco was, on the average, highest, followed by decreasing concentration in the pipe, cigarette, cigar, and snuff forms of tobacco. Fluoride content of the plug and pouch forms of tobacco was highest, followed by cigar, snuff, pipe, and cigarette tobacco, respectively. Generally, nonsmoking forms of tobacco are most directly related to the potential promotion of caries. The total sugar content in the control tobacco leaves was comparable to that in cigars and snuff which have the least amount of sugar, whereas the fluoride content was similar to plug and pouch which have the highest amount of fluoride. Brands of snuff on average had the lowest concentrations of sugar (average 1.9 percent) and were among the products with the lowest fluoride content (average 0.26 ppm). Pouch and plug exceeded other forms in both sugar and fluoride content. (Auth. Abs. Mod.)

80–0697. Gopalam, A.; Gopalachari, N. C. **Bio-Chemical Changes During Maturation of Flue-Cured Tobacco. II. Changes in Certain Chemical Constituents.** *Tobacco Research* 5(1): 60-65, June 1979.

Changes in certain chemical constituents in two varieties of tobacco (Kanakaprabha and CTRI Special) during different stages of maturity in the bottom, middle, and top primings have been studied. Kanakaprabha leaves had significantly higher amounts of starch, total sugars, reducing sugars, and sucrose than CTRI Special. All the forms of carbohydrates decreased during maturation in both the varieties. Both the varieties had par values of total nitrogen and nicotine. Protein nitrogen in Kanakaprabha leaves, however, was significantly lower than in those of CTRI Special. Total nitrogen decreased up to mature stage and then the level was maintained. Protein nitrogen decreased up to mature stage and increased in the over-mature leaves indicating that translocations of soluble nitrogenous constituents from leaf to stalk may have occurred during these stages. Changes in nicotine during maturation were not significant. Kanakaprabha leaves contained higher amounts of chlorogenic acid and rutin than those of CTRI Special. Rutin significantly increased in the over-mature leaves while chlorogenic acid remained more or less steady during maturation. (Auth. Abs.)

80–0698. Great Britain Laboratory of the Government Chemist. **Smoke Chemistry.** In: Great Britain Laboratory of the Government Chemist Report, 1977, pp. 105-106.

New equipment, marketed as a modular attachment to the 20-channel smoking machine system, is now used to analyze carbon monoxide content in cigarette smoke. In this system the vapor phase from cigarettes smoked for determination of tar and nicotine yields is exhausted into a collecting channel, pumped through a nondispersive infrared detector, and the carbon monoxide concentration is displayed and printed. The laboratory continues to discuss methods of tobacco smoke analysis with the tobacco industry.

80–0699. Great Britain Laboratory of the Government Chemist. **Automation of Smoke Analysis.** In: Great Britain Laboratory of the Government Chemist Report, 1977, pp. 104-105.

Soon after it had been decided to organize a program for cigarette smoke analysis in 1971 in Great Britain, it became obvious in discussions with the Department of Health and Social Security and the tobacco industry that the work would generate a vast quantity of data. A comprehensive computer-assisted data processing system has been utilized for this purpose.

80–0700. Great Britain Laboratory of the Government Chemist. **Tar and Nicotine Yields of Cigarettes.** In: Great Britain Laboratory of the Government Chemist Report, 1977, pp. 97-104.

The Great Britain Laboratory of the Government Chemist has continued to examine monthly samples of cigarette brands on sale in the United Kingdom to determine their tar and nicotine yields. The results from this testing program are published by the Department of Health and Social Security at regular intervals in the form of tables which show the brands ranked according to tar yield. Several of these tables are included here. During 1977, 134 brands were examined. The Laboratory has now developed a total systems approach for greater cost efficiency using software transfer of analysis data. The parameters measured include sample weight, water content, nicotine content (as total nicotine alkaloids), puff count, and carbon monoxide yield. The analytical instruments are linked on-line with the computer.

80–0701. Hamid, A.; McClure, W. F.; Weeks, W. W. **Rapid Spectrophotometric Analysis of the Chemical Composition of Tobacco. Part 2. Total Alkaloids.** *Beitraege zur Tabakforschung International* 9(5): 267-274, December 1978.

This paper illustrates the potential of a computerized spectrophotometer for measuring total alkaloids in tobacco. Prediction equations were developed for three optical parameters. Of the three parameters investigated $d^2(\log(1/R))/d\lambda^2$ gave the best results and $dR/Rd\lambda$ gave better results than $\log(1/R)$, where reflectance R is the ratio of the detector signal of the sample to the detector signal of the ceramic standard in the reflectance mode. The coefficient of determination r^2 for the prediction equation containing $d^2(\log(1/R))/d\lambda^2$ terms at 10 different wavelengths was 0.975. This equation predicted the total alkaloids in an independent set of samples with a standard error of 0.438 percent. Instrument noise contributed 43 percent of the variation; the remainder was attributed to anomalies of the chemical methods. (Auth. Abs.)

80–0702. Hanst, P. L.; Spence, J. W.; Clay, F. R. **Chlorofluoromethanes: Their Thermal Stability in Passing Through Cigarettes.** *American Industrial Hygiene Association Journal* 39(9): 772-776, September 1978.

Experiments were carried out to determine whether chlorofluoromethanes (CFM's) in air are significantly decomposed when passing through lighted cigarettes. Infrared spectroscopy was employed to see if trichlorofluoromethane or chlorodifluoromethane would yield carbonyl chloride, carbonyl chlorofluoride, or carbonyl fluoride. Analysis of the cigarette smoke showed that these reaction products were not formed in yields higher than 0.1 percent. As far as the instruments could show, all the chlorofluoromethane passed through the lighted cigarette without decomposition. It is concluded that chlorofluoromethane pollutants in room air do not pose any greater health hazard to smokers than to nonsmokers.

80–0703. Harris, C. R.; Chapman, R. A.; Miles, J.R.W. **Insecticide Residues in Soils on Fifteen Farms in Southwestern Ontario, 1964-1974.** *Journal of Environmental Science and Health. Part B* 12(3): 163-177, 1977.

Soils on 15 farms in southwestern Ontario were sampled in 1974, as part of a long-term study initiated 10 years earlier, to determine the extent to which residues of insecticides were occurring in agricultural soils through current insect control practices. Residues of organochlorine (OC) and organophosphorus (OP) insecticides were determined using gas-liquid chromatography. DDT residues were present on all farms, being highest in orchard > vegetable > tobacco > field crop soils. Residue levels of t-DDT were generally similar to or lower than those first detected in 1964 (3.00 vs. 3.06 ppm in tobacco soils, respectively). Cyclodiene insecticide residues were present in soil on 13 of the 15 farms, being highest in vegetable > tobacco > field crop > orchard soils. Residue levels in 1974 were 1/2 to 3/4 those found in 1964. No significant accumulation of OP insecticides was apparent in mineral soils used for production of tobacco and other field crops. However, OP insecticide residue levels increased tenfold in vegetable soils, particularly those high in organic matter, from 1964 to 1974, and in 1974 exceeded t-cyclodiene insecticide residue levels. The predominant OP insecticide detected in organic soils was ethion. (Auth. Abs. Mod.)

80–0704. Harvey, W. R. **Manual Determination of Nitrate and Sulfate in Tobacco and Tobacco Products.** *Tobacco International* 181(5): 72-75, March 9, 1979.

Simple manual procedures for the determination of nitrate and sulfate in tobacco leaf and tobacco products are described. Nitrate is determined on a dilute acidic extract of the tobacco or tobacco product by reaction with diphenylamine, ammonium chloride, and p-aminophenyl sulfone in strong acid media to form a blue color. Sulfate is determined turbidimetrically on an acid extract of the ashed sample after the addition of barium chloride. The presence of sorbitol regulates the size of the barium sulfate crystals and stabilizes them against sedimentation. Readout for both procedures is by a spectrophotometer such as the Bausch and Lomb "Spectronic 20." Use of a simple spectrophotometer makes the analysis suitable for laboratories with little instrumentation or where the sample demand does not justify automation. An acceptable comparison of sulfate and nitrate results obtained by these two procedures is made with the results obtained by recognized literature procedures. Recoveries of sulfate and nitrate added to tobacco average 97 and 98 percent, respectively. Respective standard deviations at the 2-Sigma level are +9 and +5 percent. (Auth. Abs.)

80–0705. Hiller, J. M.; Mohan, M. S.; Brand, M. J. **A Nonaqueous Conductimetric Titration for the Analysis of Alkaloids in Cigarette Tobacco.** *Journal of Chemical Education* 56(3): 207-208, March 1979.

A conductimetric titration in glacial acetic acid medium was used to determine total alkaloids in cigarette tobacco. The alkaloid content in three brands of cigarettes was found to range from 0.9 to 2.4 percent, depending on the brand.

80–0706. Jodl, R. **Identifizierung von Farbstoffen bei Papieren fur Tabakerzeugnisse unter besonderer Berucksichtigung der Huminsaure. [Identification of Dyes in Paper for Tobacco Products With Special Reference to Humic Acid.]** *Beitraege zur Tabakforschung International* 9(5): 297-307, December 1978, German.

After a brief look at the laws relating to food products, the particular problems of the presence of humic acid in paper for tobacco products are discussed. Origin and significance as well as physical and chemical properties of humic acid are described. The principle of the analytical method is that the dyes transferred from the paper to an aqueous solution are freed of humic acid by acidification and extracted from the centrifugate, using isoamyl alcohol. The concentrated aqueous eluate from the amyl alcohol is examined by paper chromatography. The Rf values for the dyes after their preparation are given and the analytical procedure explained by means of examples dealing with cigarette paper, filter wrappers, and imprints on the paper. (Auth. Abs. Mod.)

80–0707. Kerker, M.; Sculley, M. J.; Farone, W. A.; Kassman, A. J. **Optical Properties of Cigarette Smoke Aerosols.** *Applied Optics* 17(19): 3030-3031, October 1, 1978.

This paper reviews and discusses reported data on the optical properties of cigarette smoke. A value of 1.45 was obtained for the real part of the refractive index. For sidestream and mainstream smoke, cross section ratios were 7 and 0.8, respectively. Spectrophone results were inconsistent with the experimental and theoretical optical properties reported here. Estimates of the size distribution of cigarette smoke based on an angular scattering technique are dependent on the real part value of the refractive index.

80–0708. Schmeltz, I.; Wenger, A.; Hoffmann, D.; Tso, T. C. **On the Fate of Nicotine During Pyrolysis and in a Burning Cigarette.** *Journal of Agricultural and Food Chemistry* 27(3): 602-608, May-June 1979.

Products obtained from the thermal degradation of (^{14}C)nicotine in a combustion tube (under pyrolytic conditions) and in a cigarette (undergoing machine smoking) were examined by gas-liquid chromatography (GLC), by GLC-mass spectrometry, and by radiochromatography. Under pyrolytic conditions in a combustion tube, nicotine underwent extensive degradation to pyridines, quinolines, arylnitriles, and aromatic hydrocarbons. In contrast, in a burning cigarette, a substantial portion of nicotine remained intact (approximately 41 percent), 12.5 percent underwent oxidation to carbon dioxide, up to 11 percent was degraded to volatile pyridine bases, and negligible amounts were converted to neutral or acidic particulate components. A major portion of nicotine and its degradation products was also diverted to sidestream smoke. These results suggest that pyrolysis experiments may be of limited value for establishing the fate of nicotine and possibly other tobacco components in a burning cigarette. (Auth. Abs.)

80–0709. Severson, R. F.; Arrendale, R. F.; Chaplin, J. F.; Williamson, R. E. **Use of Pale-Yellow Tobacco to Reduce Smoke Polynuclear Aromatic Hydrocarbons.** *Journal of Agricultural and Food Chemistry* 27(4): 896-900, 1979.

Smoke data (cigarette density, puff number, total particulate matter, nicotine, and polynuclear aromatic hydrocarbons) and lipid data (hydrocarbons, terpenes, fatty acids, docosanol, and sterols) were correlated for five normal green tobacco cultivars (Va 115, Coker 139, NC 2326, NC 95, Coker 187-Hicks) and for two of their pale-yellow selections. Smoke nicotine levels for all pale-yellow selections were higher (2-19 percent) compared to the normal green parents. Pale-yellow tobaccos had the same or higher levels of docosanol, slightly higher levels of neophytadiene and phytol, lower levels of terpenes, and the same or lower levels of sterols than the normal tobaccos. All but one of the pale-yellow selections produced lower levels (7-25 percent) of smoke polynuclear aromatic hydrocarbons. The reduction of smoke polynuclear aromatic hydrocarbons in the pale-yellow tobaccos was attributed to their lower leaf solanesol levels. (Auth. Abs.)

80–0710. Snook, M. E. **Nitrogen Analogues of Polynuclear Aromatic Hydrocarbons in Tobacco Smoke.** In: Jones, P. W.; Freudenthal, R. I. (Editors). *Carcinogenesis. Volume 3: Polynuclear Aromatic Hydrocarbons.* New York, Raven Press, 1978, pp. 203-215.

A procedure for rapid analysis of polynuclear aromatic hydrocarbons (PAH) and nitrogen-polynuclear aromatic hydrocarbons (N-PAH) from a sample of smoke condensate is presented. The method entails water extraction, silicic acid chromatography, and gel filtration and gas chromatography. This method separates the indole and carbazole members of the N-PAH from PAH.

See also, **80–0740**

PHARMACOLOGY AND TOXICOLOGY

80–0711. Berry, C. N.; Hoult, J.R.S.; Littleton, J. M.; Moore, P. K.; Umney, N. D. **Nicotine Causes Prostaglandin Efflux From Isolated Perfused Rat Lung.** *British Journal of Pharmacology* 6691: 101, May 1979.

Male rats were exposed to an aerosol spray containing sodium hydrogen tartrate ("sham-exposed") or 6 percent (-)-nicotine hydrogen tartrate ("nicotine-treated"). The amount of prostaglandin (PG)-like material released after (-)-nicotine perfusion through the lungs varied considerably, particularly in the nicotine-treated rats. For example, (-)-nicotine (2 μg) released 18.0±4.5, 20.0±19.6, and 45.3±27.4 ng PGE_2 equivalent from lungs of control, sham-treated, and nicotine-treated rats respectively, and (-)-nicotine (15 μg) released 102.0±26.0, 114.0±43.5, and 154.0±72.3 ng (mean±s.e. mean, n = 6-8). The increase in PG efflux from the nicotine-treated lungs is not significant at the 5 percent level and further experiments are in progress. The mechanism whereby (-)-nicotine releases PG-like material from the rat lung is not known. However, the response shows that tachyphylaxis after 4 to 6 challenges is blocked by prior infusion of 12 μg hexamethonium or 7 μg indomethacin, and is not elicited by similar doses of (+)-nicotine. These findings suggest that the response may be caused by a receptor-mediated increase in PG synthesis in the lung. (-)-Nicotine may have other effects on PG metabolism. Preliminary experiments suggest that in vitro it inhibits $PGF_{2\alpha}$ breakdown by rat lung homogenates. It is possible that these effects of nicotine on PG metabolism may contribute to some of the pathological changes in lung associated with cigarette smoking.

80–0712. Boobis, A. R.; Brodie, M. J.; Kahn, G. C.; Fletcher, D. R.; Saunders, J. H.; Davies, D. S. **Monooxygenase Activity of Human Liver in Microsomal Fractions of Needle Biopsy Specimens.** *British Journal of Clinical Pharmacology* 9(1): 11-19, January 1980.

Methods are described for the determination of mixed function oxidase activity in microsomal fractions from percutaneous needle biopsies of human liver. Activities of needle biopsy samples were comparable with those of wedge biopsy samples obtained at laparotomy from different subjects. Although cytochrome P-450 content of liver from rat and man was similar, human aryl hydrocarbon hydroxylase

(AHH) activity was only 10 percent of that in the rat. In biopsies with preserved hepatic architecture, AHH activity and cytochrome P-450 content showed a significant positive correlation. Cigarette smoking significantly ($p<0.01$) increased both AHH activity and turnover number, but not cytochrome P-450 content, of biopsies with normal architecture. Mean AHH activity in nonsmokers was 52.9 ± 4.0 pmol/nmol cytochrome P-450/min and in smokers 72.8 ± 5.5 pmol/nmol cytochrome P-450/min. The presence of liver disease caused a significant degrease in AHH activity and cytochrome P-450 content. (Auth. Abs. Mod.)

80-0713. Brand, G. **Karzinogene Wirkungsweise gewebseingelagerter Partikel. [Carcinogenic Mode of Action of Tissue-Embedded Foreign Particles.]** *Zentralblatt fuer Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene. Originale Reihe B* 166(2/3): 159-168, 1978, German.

Foreign materials such as asbestos fibers, schistosomal eggs, or artificial implants may cause cancer. Physical factors such as size, shape, and surface properties are of decisive importance. Parent cells of later sarcomas are demonstrable in foreign body reactive tissue within 4 weeks following experimental implantation of chemically inert glass or plastic films. The parent cells soon expand into clones of preneoplastic daughter cells. Their maturation into autonomous, invasively growing cancer cells is indispensably associated with three sequential conditions: (1) macrophages losing functional and structural signs of phagocytic activity, (2) fibrous conversion of the foreign body reaction, and (3) the preneoplastic cells making firm contact with the foreign body surface (usually during the 5th to 7th month following foreign body implantation). Such processes may explain the carcinogenicity of asbestos fibers. The role that smoking plays in the development of asbestos cancer may be due to the chemical action of tar condensates or to shortened precancerous maturation time. These findings open new perspectives toward an understanding not only of asbestos-carcinogenesis and the cocarcinogenic role of tobacco smoking, but also of the cancer phenomenon in general. (Auth. Abs. Mod.)

80-0714. Bridges, B. A.; Clemmesen, J.; Sugimura, T. **Cigarette Smoking—Does It Carry a Genetic Risk?** *Mutation Research* 65(1): 71-81, March 1979.

This report from the International Commission for Protection Against Environmental Mutagens and Carcinogens contains information from various studies concerned with mutagens (e.g., benzopyrenes, dibenzanthracenes, and nitrosamines) in cigarette smoke, and the effects of these mutagens on sister chromatid exchange frequencies in lymphocytes; and on sperm number, genetic abnormalities, and motility. One study included in the report found that the frequency of stillbirth increased with heavier paternal smoking habits ($p<0.01$). In another study, spontaneous abortions were slightly more frequent among the wives of smokers. Further studies on the relationship between paternal smoking habits and perinatal mortality are urged.

80-0715. Busch, F. W.; Seid, D. A.; Wei, E. T. **Mutagenic Activity of Marihuana Smoke Condensates.** *Cancer Letters* 6(6): 319-324, May 1979.

Smoke condensates prepared from marihuana cigarettes were mutagenic in strain TA98 of the Ames *Salmonella*/microsome test, a short-term bioassay which estimates the mutagenic and carcinogenic potential of some chemicals. The mutagens in marihuana smoke condensates required liver enzymes to be activated. The specific mutagenic activity of marihuana smoke condensates was similar to that of tobacco smoke condensates prepared from American cigarettes. Fractionation studies of the marihuana smoke condensates showed that basic components accounted for 76 percent of the recovered mutagenic activity. (Auth. Abs.)

80-0716. Campbell, M. J.; Wagner, M.M.F.; Scott, M. P.; Brown, D. G. **Sequential Immunological Studies in an Asbestos-Exposed Population. II. Factors Affecting Lymphocyte Function.** *Clinical and Experimental Immunology* 39(1): 176-182, January 1980.

As part of an immunological study being carried out on a population exposed to asbestos, the effect of two mitogens, phytohemagglutinin (PHA) and pokeweed mitogen (PWM), on lymphocyte transformation has been assessed on three separate occasions for each mitogen. Both mitogens were assayed using fetal calf (FCS) or autologous serum (AS). Those men with pulmonary fibrosis (as measured by the presence of small opacities on X-ray) had a decrease ($p<0.05$) using PHA (FCS) when compared with those men with normal chest X-rays. Allowance has had to be made for the significant decline with age for both PHA (FCS and AS) transformation. There is a significant increase in PHA (FCS) and PWM (FCS) in ex-smokers and a further increase in smokers ($p<0.05$). These trends were consistent (at a lower level of significance) from survey to survey. There was no interaction between X-ray category and smoking. The lymphocyte antibody dependent cytotoxic test was measured on four occasions; the only observable effect was a nonsignificant increase in relation to smoking. (Auth. Abs.)

80-0717. Catena, E.; Marmo, E.; Marcatili, S.; Caputi, A. P. **Alterations Anatomiques et Fonctionnelles Precoces de l'Arbre Tracheo-Bronchique Apres Inhalation de Diverses Substances Exogenes. [Early Anatomic and Functional Alterations of the Tracheobronchial Tree Following Inhalation of Exogenous Substances.]** *Broncho-Pneumologie* 27(2): 154-168, 1977, French.

Ten guinea pigs were exposed in an inhalation chamber to smoke from 40 Italian cigarettes burning continuously for 1 hour. Ten more guinea pigs were exposed under identical conditions for 1 hour on each of 3 consecutive days. At the end of each experiment, the animals were sacrificed and their thoracic viscera examined. Histologic examination revealed significant lesions in both groups of animals. The principal lesion was hypersecretion of glandular epithelium with proliferation of caliciform cells. In the case of a single session of smoke inhalation, hypersecretion was most important in the proximal section of the tracheobronchial tree, decreasing toward its periphery, and disappearing completely in distal and bronchiolo-alveolar sections. In the case of repeated smoke inhalation, hypersecretion was observed at approximately the same levels. In addition, moderate vasodilatation of the chorion capillaries and intensive phagocytosis were observed increasing from proximal to distal sections. Also, bronchospasm characteristics (i.e., quasitotal obstruction of lumen) are much more pronounced in the case of repeated inhalation of cigarette smoke. It is concluded that exogenous contaminants like tobacco smoke cause relatively moderate reversible lesions in the bronchial wall. The functional alterations are distributed along the tracheobronchial tree in proportion to the exposure period. Histological aspects are interpreted in terms of activation of three different natural defense mechanisms: hypersecretion, phagocytosis, and muscular reactivity. The stimulation of reactivity in the nonstriated muscle tissue of the bronchi and the trachea is attributed to tobacco smoke, which may act indirectly on the bronchial wall.

80-0718. Chuong, B. T.; Benarie, M. **Retention of Nitrosamines Within the Respiratory Tract.** *Atmospheric Environment* 12(8): 1803, 1978.

To determine nitrosamine retention in the respiratory tract, the amount of nitrosamines in cigarette smoke, with and without inhalation, was measured in four male subjects each of whom smoked two packs of 20 cigarettes. Non-inhalation and inhalation experiments were carried out by the same person. Results indicated that 100 percent of the nitrosamines in the smoke, whether from a filter or nonfilter cigarette, were retained in the respiratory tract with inhalation, while the percentages were 68 and 62, respectively, without inhalation.

80-0719. Dalbey, W. E.; Nettesheim, P.; Griesemer, R.; Caton, J. E.; Guerin, M. R. **Chronic Inhalation of Cigarette Smoke by F344 Rats.** *Journal of the National Cancer Institute* 64(2): 383-388, February 1980.

Specific-pathogen-free female F344 rats were exposed by inhalation to what was considered a maximal tolerated dose of cigarette smoke. Total pulmonary deposition of smoke particulates from a single cigarette was 0.25 mg in young rats. Rats were exposed to smoke from seven cigarettes/day for as long as 2.5 years, at which time 30 percent of the rats remained alive. Mortality of smoke-exposed animals was not different from that of untreated or sham-exposed controls. Hyperplastic and metaplastic areas in the epithelium of the nasal turbinates, larynxes, and tracheas of exposed animals were observed at death. The lungs of exposed rats contained areas of focal alveolitis consisting of accumulated pigmented macrophages, epithelial hyperplasia, fibrosis, and disrupted alveolar structure. Smoke exposure did not change the total number of tumor-bearing animals relative to controls; however, exposed rats had significantly fewer tumors in the hypophyses, hematopoietic-lymphoid systems, uteri, and ovaries but an increased number of tumors in the respiratory tracts and dermes. The smoke-exposed animals developed more subcutaneous sarcomas and respiratory tract tumors ($p<0.05$) and tended to have more adrenal tumors and tumors orginating from the oral cavity, including the lips ($p<0.10$). Only 1 of 93 (1 percent) control rats had a tumor (an alveologenic carcinoma) in the respiratory tract as opposed to 7 of 80 (9 percent) exposed animals (nasal tumors: one adenocarcinoma and one squamous cell carcinoma; pulmonary tumors: five adenomas, two alveologenic carcinomas, and one squamous carcinoma). (Auth. Abs. Mod.)

80-0720. Davies, J. M.; Latto, I. P.; Jones, J. G.; Veale, A.; Wardrop, C.A.J. **Effects of Stopping Smoking for 48 Hours on Oxygen Availability From the Blood: A Study on Pregnant Women.** *British Medical Journal* 2(6186): 355-356, August 11, 1979.

The effects of stopping smoking for 48 hours on carboxyhemoglobin (COHb), hemoglobin—oxygen (HbO_2) affinity, and hemoglobin concentration were measured in venous blood and blood from the radial artery of women in the last trimester of pregnancy. The study group was divided into smokers who continued to smoke during the period (11 women), smokers who agreed to stop (11), and nonsmokers (10). The 22 smokers had much higher initial COHb values and greater HbO_2 affinity than the 10 nonsmokers, but inasmuch as their total hemoglobin concentrations were also higher, oxygen availability was not significantly reduced. In the 11 smokers who stopped smoking, there was a significant 8 percent increase in available oxygen; this was due partly to a 4 percent increase in functioning hemoglobin after the loss of carbon monoxide and partly to the increase in P_{50}(the tension of oxygen required to half-saturate the hemoglobin). Fetal hypoxia may occur during pregnancy and labor; an 8 percent increase in maternal "available oxygen" should definitely improve fetal oxygenation. Stopping smoking should be encouraged for 48 hours before elective deliveries and before all elective surgical procedures performed under general anesthesia, especially in anemic patients.

80-0721. Feron, V. J. **Effects of Exposure to Acetaldehyde in Syrian Hamsters Simultaneously Treated With Benzo(*a*)pyrene or Diethylnitrosamine.** *Progress in Experimental Tumor Research* 24: 162-176, 1979.

Long-term inhalation and intratracheal instillation studies of acetaldehyde were carried out in hamsters. Some of the animals were treated with either benzo(*a*)pyrene (BP) or diethylnitrosamine (DENA). Exposure to acetaldehyde vapor

(1,500 ppm) resulted in epithelial hyper- and metaplasia accompanied by inflammation in the nasal cavity and trachea. Extensive peribronchiolar adenomatoid lesions often accompanied by inflammatory changes occurred in the lungs after intratracheal instillation of acetaldehyde. There was no evidence that acetaldehyde was carcinogenic. Respiratory tract tumors were found at each of the BP doses tested and also after treatment with DENA. The studies produced insufficient evidence to warrant acetaldehyde's being regarded as a definite cofactor in respiratory tract carcinogenesis. (Auth. Abs.)

80-0722. Hecht, S. S.; Chen, C. B.; Ohmori, T.; Hoffmann, D. **Comparative Carcinogenicity in F344 Rats of the Tobacco-Specific Nitrosamines, *N*'-Nitrosonornicotine and 4-(*N*-Methyl-*N*-Nitrosamino) -1- (3-Pyridyl) -1- Butanone.** *Cancer Research* 40(2): 298-302, February 1980.

The tobacco-specific carcinogens, *N*'-nitrosonornicotine (NNN) and 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK), were tested for carcinogenicity in F344 rats. Each nitrosamine in trioctanoin was administered by s.c. injection to 12 male and 12 female rats over a period of 20 weeks. The total dose of each nitrosamine was 3.4 mmol. The experiment was terminated after 12 months. NNK induced nasal cavity tumors in 83 percent of the males and in 83 percent of the females, liver tumors in 83 percent of the males and in 100 percent of the females, and lung tumors in 67 percent of the males and in 67 percent of the females. NNN induced nasal cavity tumors in 92 percent of the males and in 75 percent of the females. Only one liver tumor and no lung tumors were observed in the NNN-treated rats. These results indicate that, in the F344 rat, NNK is a more powerful carcinogen than is NNN. (Auth. Abs.)

80-0723. Hopkin, J. M.; Evans, H. J. **Cigarette Smoke-Induced DNA Damage and Lung Cancer Risks.** *Nature* 283(5745): 388-390, January 24, 1980.

Epidemiological studies have firmly established that cigarette smoking causes almost all cases of anaplastic and squamous cell bronchial carcinomas. It is equally well known, however, that many smokers do not develop lung cancer and, although the amount of tobacco smoked is undoubtedly the dominant risk factor, it seems possible that other factors may also be involved. There is increasing evidence to support the hypothesis that somatic mutation is an important event in the development of cancer and it has recently been shown that cigarette smoke condensate (CSC) produces many dose-related lesions in the cellular DNA of cultured human lymphocytes as measured by sister chromatid exchange (SCE) induction. Cytogenetic studies had previously shown an increase in chromosomal aberrations in blood lymphocytes of heavy smokers relative to nonsmokers, but little or no increase in SCEs. Basal and CSC-induced SCE rates in lymphocytes from different individuals have been measured and results indicate that smokers have higher rates ($p<0.05$) than nonsmokers and that smokers with untreated lung cancer have consistently higher rates than their matched heavy smoking controls ($p<0.01$). These results are in keeping with the somatic mutational hypothesis and the epidemiological evidence but also raise questions related to difficulties in smoking dosimetry and to possible variation among different individuals' responses to a common insult. (Auth. Abs.)

80-0724. Horster, M.; Koenig, T.; Schmid, H.; Schmidt, U. **Nephron Electrolyte Transport and Sodium-Potassium Adenosine Triphosphatase Activity: Influence of Nicotine in Rat and Rabbit.** *Journal of Physiology* 295: 353-363, 1979.

The influence upon mammalian renal epithelial transport of *l*-nicotine was studied. The rat kidney in situ, when infused with nicotine (1 mg/hr/kg i.v.), lowered its absolute and fractional K^+-absorption significantly ($p<0.001$) and reversibly, but Na^+-absorption did not change. Effects on glomerular function and an irreversible effect upon epithelial Na^+ and K^+ absorption were prevailing at higher infused amounts (10 or 50 mg/hr/kg). Single dissected nephron segments (i.e., collecting tubule, rabbit) were perfused in vitro, and the Na^+ and K^+ transtubular net flux was measured while *L*-nicotine (50 ng/ml) had been added to the contraluminal side of the epithelium. Both Na^+-absorption and K^+-secretion were decreased reversibly and significantly ($p<0.002$). The activity of the Na^+-K^+-activated adenosine triphosphatase was significantly ($p<0.002$) decreased in the cortical collecting tubule and in the proximal convoluted tubule of the rabbit after incubation of single in vitro dissected nephron segments with *l*-nicotine (50 or 100 ng/ml). In contrast, nicotine added to a homogenate of renal cortical tissue had no effect on the Na^+-transport enzyme or on key enzymes of glycolysis and gluconeogenesis. These observations on the kidney in situ and on defined, perfused, and nonperfused nephrons in vitro suggest that *l*-nicotine has dose-dependent, direct epithelial, and mediated systemic effects on mammalian renal ion transport. (Auth. Abs.)

80-0725. Ingenito, A. J. **Effects of Cigarette Smoke Inhalation on the Cat Electroretinogram: Comparisons With Nicotine and Carbon Monoxide.** *Journal of Pharmacology and Experimental Therapeutics* 211(3): 647-655, December 1979.

Previous studies have shown a significant electroretinographic b-wave depression by carbon monoxide (CO) in anesthetized cats at "smoking" concentrations. The present studies were done to establish whether cigarette smoke inhalation under similar conditions would also result in a b-wave depression. Cats inhaled the smoke diluted to varying degrees with air for 1 min every 30 min over a 2-hour experimental period. Comparative studies were also done with intravenous nicotine injections and infusions (5-100 μg/kg) and with simultaneous intravenous nicotine and CO inhalation (0.05-0.1 percent in air). Arterial blood pressure, heart rate, arterial oxyhemoglobin, carboxyhemoglobin

(HbCO), pO_2, and pCO_2 were routinely monitored along with the dark-adapted electroretinographic response to flashes of white light. The data show that cigarette smoke inhalation results in a significant decrement in mean b-wave amplitude (maximum 46 percent) roughly paralleling arterial HbCO (maximum 48 percent). The decrement is statistically significant at -17 percent, following 60-min exposure to the smoke from each of two cigarettes, at an HbCO of 9.4 percent. Intravenous nicotine alone had no significant effect on the b-wave amplitude and when combined with simultaneous CO inhalation, nicotine did not affect the ability of CO to decrease the b-wave. The results reveal effects of cigarette smoke on the preganglion cell retina which are similar to those of CO. The findings are discussed in terms of potential deleterious effects of smoking on the visual system and also the possibility of a role for CO in the etiology of tobacco amblyopia. (Auth. Abs.)

80–0726. Janardhana Rao, G.; Krishna Rao, A.; Pattabiraman, T. N. **Studies on 2,3-Diphosphoglycerate Levels in Blood.** *Indian Journal of Medical Research* 70: 483-486, September 1979.

Levels of 2,3-diphosphoglycerate in the blood of 15 chronic smokers (≥ 10 cigarettes/day for the last 5 years) and 15 athletes were compared with the levels present in 15 normal persons. A significant ($p<0.001$) increase in levels of 2,3-diphosphoglycerate was found in chronic smokers and athletes when compared to control subjects. A significant increase was also noted when the levels were expressed as a function of hemoglobin concentration and also as a function of packed cell volume. (Auth. Abs.)

80–0727. Kahl, G. F.; Klaus, E.; Legraverend, C.; Nebert, D. W.; Pelkonen, O. **Formation of Benzo(*a*)pyrene Metabolite-Nucleoside Adducts in Isolated Perfused Rat and Mouse Liver and in Mouse Lung Slices.** *Biochemical Pharmacology* 28(7): 1051-1056, April 1, 1979.

The formation of benzo(*a*)pyrene metabolite-nucleoside adducts in perfused rat and mouse liver and in mouse lung slices was studied by Sephadex LH20 chromatography. In liver from β-naphthoflavone-pretreated rats, four different deoxyribonucleoside complexes were observed; these are tentatively attributed to DNA modification by the 7,8-diol-9,10-epoxide(s), secondary metabolites of benzo(*a*)pyrene quinones, the 4,5-oxide, and secondary metabolites of benzo(*a*)pyrene phenols. The diol-epoxide-deoxyribonucleoside adduct was also detected in perfused liver and in lung slices from 3-methylcholanthrene-treated genetically responsive C57BL/6N mice, whereas no adducts were detectable in such samples from 3-methylcholanthrene-treated genetically nonresponsive DBA/2N mice. In perfused liver of phenobarbital-pretreated rats, the 4,5-oxide-deoxyribonucleoside adduct was present. These results suggest that some of the benzo(*a*)pyrene metabolite-nucleoside complexes generated by microsomes and deproteinized DNA in vitro also occur in the intact rodent liver and lung tissues. Furthermore, complexes with the diol-epoxide(s) were observed with RNA from perfused liver of β-naphthoflavone-treated, but not from untreated or phenobarbital-treated rats. Complexes between ribonucleoside(s) and the diol-epoxide(s) were also found in perfused liver or lung slices from genetically responsive but not from genetically nonresponsive mice. (Auth. Abs.)

80–0728. Kiss, I.; Walker, R. J. **The Action of Cholinergic Antagonists on the Effect of Acetylcholine, Nicotine and Benzoylcholine Applied Iontophoretically Onto Two Identified Neurones in the Brain of *Helix Aspersa*.** *Comparative Biochemistry and Physiology. Part C* 60(1): 89-99, 1978.

Intracellular recordings were made from two identified neurones in the brain of *Helix aspersa*. Cell E4 is inhibited by acetylcholine while cell F1 is excited by acetylcholine. Thirteen cholinolytic agents were tested for their blocking actions against iontophoretically-applied acetylcholine on the two cells. The compounds examined were strychnine, scopolamine, atropine, tubocurarine, pancuronium, gallamine, hexamethonium, pentolinium, mecamylamine, benzoquinonium, decamethonium, suxamethonium, and benzoylcholine. All the compounds were at least partial antagonists on cell E4 while only three, i.e., scopolamine, decamethonium, and suxamethonium, did not block the cholinergic receptors of cell F1. Four cholinolytics were further tested as antagonists against iontophoretically-applied nicotine and benzoylcholine; these were atropine, strychnine, tubocurarine, and hexamethonium. The only compound which did not block the actions of nicotine and benzoylcholine on both cells was strychnine which had no blocking action against benzoylcholine on cell F1. (Auth. Abs.)

80–0729. Klein-Szanto, A.J.P.; Topping, D. C.; Heckman, C. A.; Nettesheim, P. **Ultrastructural Characteristics of Carcinogen-Induced Dysplastic Changes in Tracheal Epithelium.** *American Journal of Pathology* 98(1): 83-89, January 1980.

Squamous metaplasias with various degrees of atypia, carcinomas in situ, and microinvasive carcinomas produced in rat tracheal transplants by a 4-week exposure to dimethylbenz(*a*)anthracene were studied 12 to 96 weeks after cessation of carcinogen exposure. In addition to the classical nuclear and cytoplasmic indicators of atypism, the squamous metaplasias with slight and moderate atypia were characterized by the presence of glycogen in the spinous layer, atypical composite keratohyalin granules in the granular layer, and in most cases an orthokeratinized horny layer. The squamous metaplasias with severe atypia showed an increased secretory activity and a decreased number of desmosomes. Carcinomas in situ were characterized by numerous intracellular and extracellular microcysts filled with secretory material identified by histochemical techniques as containing neutral glycoproteins and sialomucins. Microinvasive carcinoma showed similar characteristics, with an increase in polylobulated nuclei and appearance of multilaminar ergastoplasm. An outstanding feature in all these lesions was the presence of dark fusiform epithelial cells. They were observed in the middle layers in the squamous metaplasias with severe atypia and appeared in all layers of the carcinomas in situ. (Auth. Abs.)

80–0730. Le Bouffant, L.; Martin, J. C.; Daniel, H.; Henin, J. P.; Normand, C. **Action of Intensive Cigarette Smoke Inhalations on the Rat Lung. Role of Particulate and Gaseous Cofactors.** *Journal of the National Cancer Institute* 64(2): 273-281, February 1980.

The action of high doses of cigarette smoke alone or combined with coal dust or acrolein was investigated in noninbred Sprague-Dawley rats. Inhalation occurred up to six sessions per day, the animals being subjected to a treatment with oxygen after each inhalation session to reduce the carbon monoxide level in the blood. The amount of tar found in the lungs depended on the number of inhalation sessions per day rather than on the total number of inhalations. Smoke inhalation caused emphysematous lesions, the extent of which depended on the total dose of smoke inhaled. No lung tumors were observed. Only a hyperplasia of the alveolar lining involving type II alveolar cells and, at a later stage, areas of metaplasia generally limited to the bronchial area were found. The association of coal dust with cigarette smoke did not alter the specific effects of smoke and resulted in effects peculiar to the action of dust: alveolitis of the macrophage type, fibrosis of the reticulin type, limited hyperplasia, and ciliated metaplasia. When smoke was combined with acrolein, the effects due to smoke were not appreciably altered. (Auth. Abs.)

80–0731. Le Groumellec, A.; Duhamel, Y.; Simon, L. **Etude de l'Affinite de l'Hemoglobine Pour l'O_2 par la Methode de la P50 Chez les Fumeurs. [Study of the Affinity of Hemoglobin for O_2 in Smokers, Using the P50 Method.]** *Ouest Medical* 31(4): 181-184, February 25, 1978, French.

Hemoglobin affinity for oxygen (O_2) was studied in blood samples of 12 moderate smokers (10-20 cigarettes/day) and 13 nonsmokers of the same average age (40 years). The increase in oxygen affinity in the presence of carboxyhemoglobin (COHb) in blood of the smokers was evaluated by simultaneous measurements of COHb levels and variations in a 50 percent saturation of hemoglobin with a gaseous mixture containing 3.7 percent O_2 (the P50 method). The average COHb level was found to be 1.02 percent in nonsmokers and 4.42 percent in smokers. A small (0.73 torr) but statistically significant difference was detected between P50 in the two groups of subjects, without any correction for carbon monoxide effect. This difference became insignificant after the correction was made. However minimal is the increase in P50 in the smokers, it indicates an increased oxygen affinity which may be a non-negligible factor contributing to coronary disease, particularly in heavy smokers. Except in the extreme cases, an increase in O_2 affinity does not appear to be the cause of disorders generated by cigarette smoke. Instead, it appears that smoking acts at the lung level by making ventilation more difficult.

80–0732. Lewis, D. J.; Edmondson, N. A.; Prentice, D. E. **A Quantitative Ultrastructural Comparison of Macrophages From Rats Exposed to Smoke Derived From Conventional Tobacco and a Tobacco Substitute.** *Toxicology Letters* 5(1): 83-87, January 1980.

Using an image-analyzing computer, a variety of ultrastructural features from micrographs of alveolar macrophages have been quantified. Macrophages from rats exposed to smoke from conventional tobacco cigarettes for 6 months revealed statistically significant ($p<0.001$) changes when compared to controls. The macrophages were larger, rounded with fewer pseudopodia, and contained increased numbers of inclusions. There were no statistically significant changes in macrophages from rats exposed to smoke from a tobacco substitute. (Auth. Abs.)

80–0733. Lindenschmidt, R. R.; Persaud, T.V.N. **Effect of Ethanol and Nicotine in the Pregnant Rat.** *Research Communications in Chemical Pathology and Pharmacology* 27(1): 195-198, January 1980.

Treatment of pregnant Sprague-Dawley rats on day 9 of gestation with both ethanol (0.02 ml/gm of a 12.5 or 25 percent solution, intraperitoneally) and nicotine (5 mg/kg, subcutaneously) resulted in no significant adverse effects on embryonic development and fetal viability. There was a significant ($p<0.05$) difference in mean fetal weight, however, for two experimental groups. Administration of 5 mg/kg of nicotine, subcutaneously, and 0.02 ml/gm of 25 percent ethanol, intraperitoneally, to Group IV rats resulted in a mean fetal weight of 5.3 ± 0.18 gm as compared to 0.02 ml/gm of a 25 and 12.5 percent ethanol solution administered intraperitoneally to Group II rats. (Auth. Abs. Mod.)

80–0734. Matejevic, D.; Pavlovic, M.; Djukic, V. **Promene u Elektrokardiografskim Kararteristikama Pacova pod Dejstvom Razlicitih Doza Nikotina. [Electrocardiographic Changes in Rats Induced by Different Nicotine Doses.]** *Srpski Arhiv Za Celokupno Lekarstvo* 105(10): 821-827, October 1977, Serbo-Croatian.

The effect of a continuous infusion of 30 to 200 mg/kg of a nicotine solution on heart rate and electrocardiographic (ECG) characteristics has been studied in seven male rats weighing 250 to 300 g. Nicotine hydrogen tartarate in a physiological saline solution was infused into the external jugular vein of the rats at a 33μl/min rate over a 1-hour period. A non-nicotine saline solution was administered to a control group of rats. During the first 15 to 30 min of nicotine infusion, statistically significant decreases in heart rate were observed: the decreases were 37.0 and 46.4 percent with doses of 40 and 50 mg/kg nicotine, respectively. Only two animals survived infusion of a 100 mg/kg dose and none survived infusion of a 200 mg/kg dose. There were several notable auricular and ventricular complex effects. The P-Q interval of the ECG increased significantly with a 30 mg/kg nicotine dose. The Q-T interval increased significantly at all nicotine doses. The T-P interval began increasing at a 50 mg/kg dose. The negative chromotropic effect of nicotine on heart rate appears dependent on lengthening all ECG intervals, particularly Q-T and T-P intervals. The complex effects of nicotine are discussed with respect to the autonomous nerve ganglions and ionic permeability of cell membranes.

80–0735. Pettersson, B. **Isolated Hamster Brown Fat Cells: A Bioassay for Toxicity Tests at the Cellular Level. Effect of Cigarette Smoke Condensates From High and Low Tar Yield Cigarettes.** *Chemico-Biological Interactions* 29(1): 95-102, January 1980.

A test system for the detection of substances with cell-irritating or cell-damaging effects using isolated brown fat cells is presented, and the effects of cigarette smoke condensates (CSC) from high- and low-tar yield cigarettes on this system are examined. The condensates inhibited noradrenaline-stimulated respiration in a dose-dependent manner. Ethanol solutions of the condensates had a stronger inhibitory effect than dimethylsulfoxide solutions. The CSC strongly inhibited the mitochondrial function but other cellular functions were also impaired. (Auth. Abs.)

80–0736. Post, C. **Studies on the Pharmacokinetic Function of the Lung With Special Reference to Lidocaine.** *Acta Pharmacologica et Toxicologica* 44(Supplement 1): 29-53, 1979.

A general discussion of the study of pharmacokinetic functions of the lung, with special reference to the effects of lidocaine, is presented. In healthy cardiopulmonary volunteers, the pattern of lung uptake of lidocaine was essentially the same as observed in anesthetized pigs. In the healthy volunteers (5 smokers, 6 nonsmokers), the maximal arterial concentration after the bolus injection of lidocaine was about one-third of what it would have been if the lungs had not exerted their trapping effect. The smokers had a significantly higher lung uptake of lidocaine than the nonsmokers ($p<0.05$). Benzo(*a*)pyrene has been reported to be covalently bound in rat lungs, the degree of binding being increased by exposing the rats to cigarette smoke. Observed changes in lidocaine pharmacokinetics in smokers' pulmonary circulation may be an early sign of changes in pulmonary function.

80–0737. Radford, E. P.; Martell, E. A. **Polonium-210: Lead-210 Ratios as an Index of Residence Times of Insoluble Particles From Cigarette Smoke in Bronchial Epithelium.** In: Walton, W. H. (Editor). *4th International Conference on Inhaled Particles and Vapors*, Elmsford, New York, Pergamon Press, 1977, pp. 567-581.

Lead-210 (^{210}Pb) and its granddaughter polonium-210 (^{210}Po) are both natural constituents of cigarette smoke, the ^{210}Pb being enriched in insoluble particles derived from sintered tobacco trichome tips. These particles are stripped of the polonium on combustion, and thus the polonium begins ingrowth at the time of inhalation. Polonium-210 is found in bronchial tissues of smokers, and evidence shows that ^{210}Pb is present at these sites in excess of the polonium. On the assumption that all polonium arises from ingrowth from the insoluble particles, one may calculate from the polonium-lead ratio the mean residence time of these particles. The half-life of polonium (138 days) is almost ideal for this purpose, and its alpha radiation makes measurements of very low concentrations possible. This technique is the first available to assess residence time for inhaled particles in the bronchial epithelium, an important datum because of the vulnerability of bronchial tissues to disease. Measurements from three smokers and two nonsmokers show that ^{210}Pb from natural aerosols also is concentrated at bronchial bifurcations, but little ^{210}Po is associated with this soluble lead. This fact makes estimates of residence time in bronchial epithelium of smokers (3-5 months in these preliminary data) likely to be low. (Auth. Abs.)

80–0738. Rees, P. J.; Chilvers, C.; Clark, T.J.H. **Evaluation of Methods Used to Estimate Inhaled Dose of Carbon Monoxide.** *Thorax* 35(1): 47-51, January 1980.

Estimation of alveolar carbon monoxide measured either as end-expired or mixed-expired carbon monoxide tension with two spectrophotometric estimations of venous blood carboxyhemoglobin (COHb) has been compared. Estimation of mixed-expired carbon monoxide proved more convenient for patients than the end-expired method, and this estimation of carbon monoxide exposure discriminated between nonsmokers and smokers as well as measurements of COHb. The technique is bloodless, economical, and simple to perform. It should prove a valuable method for validating reported smoking habits of people attending smoking advisory centers and for epidemiological surveys of smoking. (Auth. Abs.)

80–0739. Sarantos-Laska, C.; McCulloch, M. W.; Rand, M. J. **Facilitatory Effect of Nicotine on Adrenergic Neuroeffector Transmission in the Isolated Ear Artery of the Rabbit.** *Journal of Pharmacy and Pharmacology* 32(2): 92-96, February 1980.

The effects of nicotine on perfusion pressure and vasoconstrictor responses to sympathetic nerve stimulation in the isolated ear artery of the rabbit were studied. Infusions of nicotine (50μM) produced a transient increase in perfusion pressure and potentiated responses to nerve stimulation. These effects of nicotine were unaffected by atropine (0.3μM) and abolished or significantly reduced by hexamethonium (300μM) or mecamylamine (1μM), respectively. In experiments with ear arteries previously labeled with (^{3}H)noradrenaline, an infusion of nicotine (50μM) produced a transient increase in tritium efflux and the potentiation of responses to nerve stimulation in the presence of nicotine was accompanied by a statistically significant increase in stimulation-induced tritium efflux. These effects of nicotine were abolished by hexamethonium (300μM) or mecamylamine (1μM). (Auth. Abs.)

80-0740. Severson, R. F.; Snook, M. E.; Akin, F. J.; Chortyk, O. T. **Correlation of Biological Activity With Polynuclear Aromatic Hydrocarbon Content of Tobacco Smoke Fractions.** In: Jones, P. W.; Freudenthal, R. I. (Editors). *Carcinogenesis. Volume 3: Polynuclear Aromatic Hydrocarbons.* New York, Raven Press, pp. 115-130.

The study findings indicate that the polynuclear aromatic hydrocarbons (PAH) are the primary tumor initiators in cigarette smoke condensate (CSC) and that they must interact with other condensate components to exert their tumorigenic effect. The most refined and characterized PAH fraction from CSC that was tested, F-55B, failed to induce tumors in dimethylbenzanthracene-treated mice unless it was combined with other CSC fractions. When combined with other nonidentified components of the neutral fraction or with two other active fractions from the neutral and acidic portion of CSC, F-55B exhibited a synergistic tumorigenic effect. The multialkylated four-ring and larger PAH appear to be primarily responsible for the tumor-initiating activity of CSC. However, the PAH constituents of F-54 still remain to be tested for tumor-initiating activity before they can be classified as nontumorigenic. Each individual gel fraction of F-55B could be tested to determine which PAH components are the most effective in initiating tumor growth. (Auth. Abs.)

80-0741. Steele, V. E.; Marchok, A. C.; Cohen, G. M. **Transformation of Rat Tracheal Epithelial Cells by Benzo(*a*)pyrene and Its Metabolites.** *Cancer Letters* 8(4): 291-298, February 1980.

Benzo(*a*)pyrene (BaP) and its 7,8-dihydrodiol form were metabolically activated to malignantly transform a nontumorigenic rat tracheal epithelial cell line in culture. The proximal carcinogenic metabolite of BaP, diol-epoxide I, was more efficient in transforming the epithelial cell line than the intermediate or parent compound, even at a 50- or 100-fold lower concentration, respectively. Inoculations of the transformed cell lines into immunosuppressed isogenic recipients produced differentiated carcinomas similar to those which occur in humans. (Auth. Abs.)

80-0742. Teel, R. W.; Douglas, W.H.J. **Aryl Hydrocarbon Hydroxylase Activity in Type II Alveolar Lung Cells.** *Experientia* 36(1): 107, January 15, 1980.

Data reported suggest that type II alveolar pneumonocytes (L-2) from rat lung contain aryl hydrocarbon hydroxylase (AHH) and that they have the capability of metabolizing polycyclic aromatic hydrocarbons. Following a 24-hour exposure to benzo(*a*)pyrene, the AHH activity induced was twice that of dimethylbenzanthracene and 13 times that of 3-methylcholanthrene.

80-0743. Tompa, A.; Langenbach, R. **Culture of Adult Rat Lung Cells: Benzo(*a*)pyrene Metabolism and Mutagenesis.** *In Vitro* 15(8): 569-578, August 1979.

A method is described for obtaining and culturing large numbers of lung cells from normal adult male rats. The lungs were perfused in situ to remove blood cells and then perfused via the trachea with a trypsin-collagenase solution to initiate tissue digestion. The tissue was further digested in the enzyme solution and approximately 2 x 10^8 viable lung cells were obtained per animal. Primary cultures contained a mixed cell population. Through eight subcultures about 70 percent of the cell population possessed an epithelial-like morphology, whereas the remaining 30 percent was fibroblast-like. Three clones of epithelial-like cells were isolated at the fourth subculture. The mass culture lung cells and the epithelial-like clone that was studied retained a normal karyotype and did not grow in soft agar. Both the mass culture cells and the epithelial clone metabolized the lung carcinogen benzo(*a*)pyrene (BP) to water-soluble products. Furthermore, the mass culture lung cells metabolized BP to intermediate(s) which mutated Chinese hamster V79 cells from ouabain sensitivity to ouabain resistance. These lung cell cultures have potential use in cell transformation, mutation, and carcinogen metabolism studies. (Auth. Abs.)

80-0744. Uchida, E.; Hirono, I. **Effect of Phenobarbital on Induction of Liver and Lung Tumors by Dimethylnitrosamine in Newborn Mice.** *Gann; Japanese Journal of Cancer Research* 70(5): 639-644, October 1979.

The effect of phenobarbital on simultaneous induction of liver and lung tumors was examined in inbred DDD mice. Group 1 of newborn mice received a single intraperitoneal (ip) injection of dimethylnitrosamine (DMN) and after weaning they were given 0.05 percent phenobarbital solution to drink. Group 2 received an injection of DMN like Group 1 but were then given normal water. Group 3 were injected ip with 0.9 percent NaCl solution and then given phenobarbital solution to drink as in Group 1, and Group 4 were injected with 0.9 percent NaCl solution like Group 3, and then given tap water to drink. The animals were examined 16 weeks after birth. In Group 1, 27 of 35 mice (77 percent) had liver tumors and 15 (43 percent) had lung tumors. In Group 2, 8 of 24 mice (33 percent) had liver tumors and 16 (67 percent) had lung tumors. Animals in Groups 3 and 4 did not develop tumors. The difference in the incidences of liver tumors, but not lung tumors, in Groups 1 and 2 was statistically significant ($p<0.01$); that is, a promoting effect of phenobarbital was observed in induction of liver tumors, but not lung tumors. (Auth. Abs.)

80-0745. Warr, G. A. **The Biology of Normal Human Bronchoalveolar Cells.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(3): 23-24, 1979.

The free lung cells recovered from healthy humans by endobronchial lavage consist primarily of pulmonary alveolar macrophages (PAM) (78.8 ± 3.8 percent) and lymphocytes (16.7 ± 3.0 percent). In response to cigarette smoking, there is a fourfold increase in the number of PAM while the number of lymphocytes remains fairly constant. Suspensions of PAM from nonsmokers have a highly undulating surface membrane. PAM from cigarette smokers consistently show a reduction in the undulation of the membrane surface with focal areas of the cells' surface being smooth and devoid of membranous projections, or covered with small blebs and vesicles resulting in a cobblestone-like appearance. These alterations effectively reduce the amount of surface area exposed to the PAM environment. In modified Boyden chambers, PAM from smokers demonstrate higher random migration and migration in the presence of a chemotactic attractant. Using a capillary-tube assay method, the PAM were assayed for direct and indirect responsiveness to the lymphokin "MIF." Cells from smokers in control assays migrate at a rate three times that of cells from nonsmokers. PAM from nonsmokers respond to MIF in both assays demonstrating the ability of the free lung cell populations to actively participate in cell-mediated immune responses. Cells from smokers were unresponsive in these assays. Nonsmokers' and smokers' PAM populations exhibit similar distributions of cellular avidities for IgG-antigen complexes, while complement receptors are significantly reduced in PAM from smokers. Alterations in PAM receptor activity can occur, and activity may be increased during certain pathologic conditions which result in stimulation of the PAM. The ability of PAM to ingest and kill microorganisms in vitro varies with the organism employed in the assay and the environment from which the PAM are recovered. Phagocytosis of *Staphylococus aureus* by PAM from smokers is reduced, although more than compensated for in the lung by the fourfold increase in total numbers of PAM present. In contrast, phagocytosis and killing of *Candida krusei* is comparable between PAM populations from nonsmokers and smokers. As an example of the influence of pathologic changes in the lung environment on the PAM, pulmonary alveolar proteinosis has been shown to be associated with a reduction in PAM microbicidal activity, abnormal morphology, and decreased chemotactic responsiveness.

80-0746. Wehner, F. C.; Van Rensburg, S. J.; Thiel, P. G. **Mutagenicity of Marijuana and Transkei Tobacco Smoke Condensates in the Salmonella/Microsome Assay.** *Mutation Research* 77(2): 135-142, February 1980.

Extracts and smoke condensates of marihuana, Transkei home-grown tobacco, and also commercial cigarette tobaccos were assayed for their mutagenic activity to *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538, both with and without metabolic activation. No mutagenic activity was detected in dichloromethane extracts of marihuana and tobacco, but all the smoke condensates exhibited mutagenicity with metabolic activation. The only strain not mutated by any of the pyrolyzates was TA1535. Transkei tobacco pyrolyzate proved to be the most mutagenic, followed by marihuana, pipe, and cigarette tobacco. Mutagenicity was positively associated with the nitrogen content of the various products. The potent mutagenic action of marihuana smoke condensate, coupled with a condensate yield of more than 50 percent higher than that of cigarette and pipe tobacco, indicates a high carcinogenic risk associated with marihuana smoking. (Auth. Abs.)

80-0747. World Health Organization. **Nitrates, Nitrites and *N*-Nitroso Compounds.** Environmental Health Criteria 5, World Health Organization, Geneva, 1978, 107 pp.

Environmental health criteria for nitrates, nitrites, and *N*-nitroso compounds were reviewed by a WHO Task Group in February 1976. Chemistry and analytical methods, environmental sources, metabolism, effects on experimental animals and man, and evaluation of health risks are discussed, and recommendations for further studies are made. Nitrosonornicotine has been found in unburned smoking tobacco, chewing tobacco, and snuff. The same compound has been identified in the mainstream smoke of a popular nonfilter cigarette in the United States. Evidence suggesting the presence of dimethylnitrosamine, nitrosopyrrolidine, methylbutylnitrosamine, and nitrosopiperidine in tobacco smoke has also been obtained. Since more than 80 percent of over 100 *N*-nitroso compounds tested proved to be carcinogenic in animal experiments, it is suggested that exposure to these compounds be kept as low as practically achievable.

80-0748. Yoshida, K.; Kato, Y.; Imura, H. **Nicotine-Induced Release of Noradrenaline From Hypothalamic Synaptosomes.** *Brain Research* 182(2): 361-368, January 27, 1980.

In order to elucidate the functional role of nicotinic receptors in the hypothalamus, the drug-induced release of noradrenaline from hypothalamic synaptosomes was studied utilizing (^{3}H)noradrenaline ((^{3}H)NA). The release of (^{3}H)NA from synaptosomes was significantly ($p<0.01$) increased with an increase of the dose of nicotine, carbamylcholine chloride, reserpine, or tyramine hydrochloride added to the medium, whereas arecoline, atropine sulfate, or mecamylamine hydrochloride had no significant effect. Mecamylamine hydrochloride completely inhibited the nicotine- or carbamylcholine-induced release of (^{3}H)NA at the concentration of 10^{-4}M, but had no effect on the reserpine- or tyramine-induced release of (^{3}H)NA. A high concentration of potassium in the medium which depolarizes the synaptosome membrane significantly ($p<0.01$) enhanced the release of (^{3}H)NA. These results strongly suggest that there exist nicotinic cholinergic receptors in brain synaptic regions which play an important role in the function of the hypothalamus by releasing noradrenaline and that the release mechanism of noradrenaline induced by nicotine is different from that induced by reserpine and tyramine. Although the existence of postsynaptic nicotinic receptor sites could not be ruled out, the present studies indicate the importance of presynaptic cholinergic receptors in the brain. (Auth. Abs.)

80-0749. Zielhuis, R. L.; del Castilho, P.; Herber, R.F.M.; Wibowo, A.A.E. **Levels of Lead and Other Metals in Human Blood: Suggestive Relationships, Determining Factors.** *Environmental Health Perspectives* 25: 103-109, August 1978.

Epidemiological studies on metal levels in blood were carried out. Lead in blood (PbB) levels were regarded as an independent variable. The other metals studied were cadmium, manganese, zinc, copper, and iron. Evidence suggested the existence of a different trend in serum iron levels in relation to blood lead levels in nonoccupationally exposed males (negative trend) and females (positive trend). There was a positive relation between blood lead levels and blood manganese levels in children and occupationally exposed male workers. Blood metal levels increased with age in young children. There was evidence for an influence of socioeconomic class on blood lead levels in children. A direct and an indirect effect of smoking was found on blood lead levels and on the activity of δ-aminolevulinic acid dehydratase in erythrocytes in adult nonoccupationally exposed females. An increase of PbB levels with the number of cigarettes smoked per day was observed. In addition, a large influence of smoking on blood cadmium levels was found: in nonsmoking females, the mean was 0.41 ppb; in those smoking 1-9 cigarettes per day, 0.62 ppb; and in those smoking ≥10 cigarettes per day the mean was 0.70 ppb. (Auth. Abs. Mod.)

80-0750. Zuspan, F. P.; Davis, N. **The Effect of Smoking and Oral Contraceptives on the Urinary Excretion of Epinephrine and Norepinephrine.** *American Journal of Obstetrics and Gynecology* 135(8): 1012-1015, December 15, 1979.

Eight smokers and seven nonsmokers between the ages of 20 and 38 years were studied. None of these patients was pregnant. Three smokers and two nonsmokers were not taking oral contraceptives, as contrasted by three nonsmokers and four smokers taking oral contraceptives. Results approached statistical significance for urinary norepinephrine ($p = 0.15$) and epinephrine ($p = 0.07$) when the groups were compared. The values for nonsmoking/noncontraception tended to be low but, unexpectedly, the values for smoking/contraception also were low. When the smoking subjects (six of seven) stopped smoking, there was a significant decline in norepinephrine ($p = 0.02$) and the epinephrine decline approached significance ($p = 0.08$). The study does not show any synergistic effect of oral contraceptives and smoking but demonstrates that the smoker can diminish stimulation of the adrenal gland (epinephrine) and sympathetic nervous system (norepinephrine) if she will stop smoking. (Auth. Abs.)

See also, **80-0799, 80-0805**

MORTALITY AND MORBIDITY

80-0751. Collan, Y.; Asp, S.; Hernberg, S. **Laakarien Kuolleisuus Vuosina 1953-1972. II. Mieslaakarien Kuolemansyyt. [Mortality Among Physicians in 1953-1972. II. Causes of Death Among Male Physicians.]** *Suomen Laakarilehti* (35): 2797-2799, December 10, 1978, Finnish.

In comparing the mortality rate of male physicians to that of the general population in Finland, it was found that male physicians died less frequently of malignant tumors. The difference was due to a lower rate of tumors in the respiratory tract. In the 25-64 age group the probability of death due to respiratory cancer was 2.1 for male physicians and 3.7 for males in general. The corresponding figures in the 25-74 age group were 4.0 and 8.8, respectively. The lower mortality rate among male physicians can be partly explained by the fact that they smoke less. Of this group, 45 percent had never smoked, and 20 percent were daily smokers. The corresponding figures for the average population were 30 percent and 50 percent. Only two-thirds of the physicians who smoked used cigarettes, compared to 90 percent of the general population. Another reason for the lower mortality rate could be more efficient early diagnosis and treatment.

80-0752. Gillum, R.; Blackburn, H. **The Recent Decrease in Cardiovascular Mortality in the U.S.A.: Hard Facts and Soft Interpretations.** *Revue d'Epidemiologie et de Sante Publique* 26(6): 435-439, 1978.

Mortality rates for all major cardiovascular diseases in the U.S. have declined consistently since 1940 in white men, since 1949 in nonwhite men, and since 1958 in white and nonwhite women. Among the factors reportedly responsible for these favorable trends are (1) decreases in cigarette smoking among adults, (2) altered dietary intake of fats, (3) improved control of hypertension, and (4) improved care for cardiovascular disease patients. These factors, however, do not satisfactorily explain the changed mortality rates. For example, marked decreases in cigarette smoking have been found among middle-aged men, but similar changes have not been documented in women despite their rapidly falling ischemic heart disease mortality. Since there is difficulty in explaining trends in cardiovascular disease mortality in the United States, it is suggested that there is need for an efficient program to monitor simultaneously the influences, e.g., changes in social, economic, and environmental factors and availability of preventive health services, which might explain, or even predict, mortality trends.

80–0753. Godbold, J. H., Jr.; Tompkins, E. A. **A Long-Term Mortality Study of Workers Occupationally Exposed to Metallic Nickel at the Oak Ridge Gaseous Diffusion Plant.** *Journal of Occupational Medicine* 21(12): 799-806, December 1979.

This study was undertaken to determine whether mortality from respiratory cancer among workers occupationally exposed to metallic nickel at the Oak Ridge Gaseous Diffusion Plant differed from that of workers at the same plant with no record of occupational exposure to metallic nickel or any nickel compound. A cohort of 814 nickel-exposed workers and one of 1,600 controls were identified. The members of both cohorts had a minimum followup period of 19 years. Mortality from respiratory cancer and from other causes was examined in both groups. The data showed no evidence of an increased risk of mortality due to respiratory cancer among the nickel-exposed workers. Since smokers have an increased risk of developing diseases of the circulatory system, the standard mortality ratio of 0.65 for this cause of death in the nickel workers, as compared to 0.76 in the controls, was consistent with the presence of fewer smokers in the nickel-exposed cohort. However, the difference between nickel workers and controls in mortality due to diseases of the circulatory system did not appear to be large enough for smoking patterns to explain all the deficit in observed deaths due to respiratory malignancies in the nickel workers. The exposed cohort experienced lower mortality than the controls for all causes of death. However, this difference was not statistically significant. (Auth. Abs. Mod.)

80–0754. Preston, S. H. **Older Male Mortality and Cigarette Smoking. A Demographic Analysis.** Westport, Connecticut, Greenwood Press, 1976, 163 pp.

Older male mortality since World War II has consistently exceeded the values projected on the basis of earlier experience, and this excess has grown continuously. Possible causes of excessive older male mortality, such as genetic factors, past and current environmental factors, and several risk factors in adult male mortality identified in epidemiological studies, were examined. Although obesity, lack of exercise, stress, cholesterol consumption, and cigarette smoking have all been implicated in adult male mortality, by far the most promising explanation of recent excesses in older male death rates is cigarette smoking behavior. Epidemiological studies of this factor show consumption has increased two- or three-fold since 1930 in almost every country, and even more dramatically among older males. National levels of cigarette consumption correspond very closely to the excess in older male death rates.

80–0755. Wald, N. J. **Smoking as a Cause of Disease.** In: Bennett, A. E. (Editor). *Recent Advances in Community Medicine*. Volume I. Edinburgh, Churchill Livingstone, 1978, pp. 73-96.

This paper reviews epidemiological and mortality data concerning the following diseases associated with smoking: coronary heart disease, lung cancer, chronic bronchitis, chronic obstructive airways disease, cerebrovascular and peripheral vascular diseases, and oral cancer. Results from study data indicated that lung cancer mortality rates increased in proportion to the duration of smoking raised to the fourth power, i.e., (years smoked)^4, whereas the amount smoked was approximately linearly related to lung cancer mortality. Various types of "less harmful" cigarettes (e.g., filter, tobacco substitute) are also discussed.

NEOPLASTIC DISEASES

80–0756. Beard, C. M.; Noller, K. L.; O'Fallon, W. M.; Kurland, L. T.; Dockerty, M. B. **Lack of Evidence for Cancer Due to Use of Metronidazole.** *New England Journal of Medicine* 301(10): 519-522, September 6, 1979.

Experimental studies have shown that metronidazole is carcinogenic in rodents and mutagenic in bacteria. In 771 women given metronidazole for the treatment of vaginal trichomoniasis, more cancers developed than had been expected after exclusion of one cervical carcinoma. However, the excess was not statistically significant ($p>0.05$). The observed and expected numbers of breast cancer cases were the same, but four lung cancer cases were observed, whereas 0.6 would have been expected. Interpretation of the excess of lung cancers is difficult because all four subjects were smokers, and three were over 60 years of age. The expected number of cases is based on age-specific rates and thus provides an age "adjustment." However, age-specific rates were not available for smokers as compared with nonsmokers, and, furthermore, the smoking status of most women could not be determined. Thus, it was not possible to determine whether the excess of lung cancers was due to smoking, exposure to metronidazole, or synergism between these two factors. Overall, no appreciable increase in cases of cancer was observed. (Auth. Abs. Mod.)

80-0757. Bernard, J. P. **Epidemiologie des Cancers Primitifs Broncho-Pulmonaires. [The Epidemiology of Primitive Broncho-Pulmonary Cancer.]** *Cahiers Medicaux* 5(7): 433-436, November 5, 1979, French.

This is a brief survey of evolution of the broncho-pulmonary cancer incidence and its correlation with smoking, occupational exposure, and atmospheric pollution. In general, epidemiology of the disease is correlated with daily smoking rate, increasing with age, duration of smoking, cumulative consumption, and smoke inhalation. The risk is stabilized upon smoking cessation. Among histological cancers observed in smokers, epidermal cancer (adenocarcinoma type) occurs most frequently, whereas it occurs only rarely in nonsmokers. The incidence becomes noticeable at about age 55 in nonsmokers and 35 in smokers. It is concluded that smoking is the main risk factor for bronchial cancer (in over 90 percent of all cases) and a contributing factor in cases of throat cancer, chronic bronchitis, cardio- and arteriopathies. The risk from occupational exposure is estimated to be less than 5 percent of all primitive cancers. It is probable that X-rays and smoking act synergistically, as do some atmospheric pollutants (e.g., coal and gas combustion residues, and benzopyrene) to increase the risk from smoking.

80-0758. Chalon, J.; Tang, C.-K.; Gorstein, F.; Turndorf, H.; Katz, J. S.; Klein, G. S.; Patel, C. **Diagnostic and Prognostic Significance of Tracheobronchial Epithelial Multinucleation.** *Acta Cytologica* 22(5): 316-320, September-October 1978.

In 955 patients suffering from a wide variety of malignant tumors, multinucleated tracheobronchial epithelial cells in bronchial aspirate smears were found to be approximately twice as numerous as in a control group comparable in sex, age, and smoking habits but without malignancies ($p<0.0005$). When smoking habits were considered (822 of the 1,910 patients and controls were smokers), the statistical significance remained at $p<0.0005$ when comparisons were made between cancer and control subgroups with the exception of very heavy smokers (30 or more cigarettes/day) in which it fell to $p<0.01$. Patients with all types of invasive and noninvasive carcinomas had approximately a 2.3 times greater percentage incidence of high degree multinucleation (≥ 2.5 percent) than the controls (39 and 17 percent respectively). (Auth. Abs. Mod.)

80-0759. Fortson, J. K.; Janfaza, P. **Endotracheal and Tracheostomy Tube Cytology. A Noninvasive Technique for Early Detection of Carcinoma of the Lung and Aerodigestive Tract.** *Annals of Otology, Rhinology and Laryngology* 88(6, Part 1): 809-812, November-December 1979.

Sputum cytology has been of value in the early diagnosis of carcinoma of the tracheobronchial tree. In a preliminary study, the cytology examination of the secretions adherent to 50 endotracheal tubes suggested that endotracheal and tracheostomy tube cytology will prove to be a reliable, noninvasive, and cost-effective means of screening high-risk patients for occult primary carcinomas of the lung and upper aerodigestive tract. The subjects of this study included 50 patients (17 females, 33 males); those with known head and neck carcinoma or lung carcinoma were excluded. There were 15 nonsmokers and 35 smokers. In the smoking group which was composed of 9 females and 26 males, the number of packs of cigarettes smoked per day ranged from 0.5 to 3.5 packs per day for 20 years or more. These study patients were 40 years of age or older and had been either intubated for general anesthesia or airway distress, or required tracheostomy for airway obstruction. High-risk patients were defined as patients over 40 years of age who had smoked one- half or more packs of cigarettes per day for 20 years or more. Nonsmoking patients were used as controls. In the nonsmokers, all cytology returned either as class 1 or 2 (normal to atypical nonmalignant cells) and was considered negative. In the smokers, 2 were returned as class 1, 23 were class 2, 8 were class 3 (dyskeratotic cells, suspicious), and 2 were class 4 (cells and cell clusters, positive).

80-0760. Laurence, D.J.R.; Stevens, U.; Bettelheim, R.; Darcy, D.; Leese, C.; Turberville, C.; Alexander, P.; Johns, E. W.; Neville, A. M. **Role of Plasma Carcinoembryonic Antigen in Diagnosis of Gastrointestinal, Mammary, and Bronchial Carcinoma.** *British Medical Journal* 3(5827): 605-609, September 9, 1972.

Our studies have confirmed that raised plasma levels of carcinoembryonic antigen (CEA) occur with many but not all malignant tumors, particularly those of the gastrointestinal tract, breast, and bronchus. However, the incidence of raised values may reach 30 percent in diseases associated with inflammation, regeneration, or both. For example, while a high proportion (66 percent) of patients with carcinomas of the bronchus and upper respiratory tract had CEA in excess of 12.5 ng/ml, high values were often found in patients with inflammatory chest diseases. Examination of the data from patients with pulmonary infections showed that of the 14 patients falling into the false-positive group, 10 smoked cigarettes. None of the healthy control group of subjects who smoked cigarettes had levels in excess of 12.5 ng/ml, and of 60 patients with a variety of benign or inflammatory conditions who also smoked cigarettes, only 18 had levels over 12.5 ng/ml. The rest had normal values. It may be the combination of chest infection and smoking which is primarily responsible for the raised false-positive levels in such patients. Consequently, CEA cannot yet serve as a routine screening test for cancer. Effective surgical therapy results in high plasma CEA levels returning to normal. Subsequent rises appear to develop with tumor recurrence or spreading. At present the most useful role for CEA seems to be in monitoring patients during the post-therapeutic followup period. (Auth. Abs. Mod.)

80-0761. Riotton, G. **Introduction: Cigarette et Cancer du Poumon. [Introduction: Cigarette and Lung Cancer.]** *Medecine et Hygiene* 35(1260): 3611-3612, November 2, 1977, French.

The retrospective and prospective epidemiologic studies of lung cancer and cigarette consumption of the past 25 years are briefly reviewed. The main conclusions are cited from the parallel pathological studies of the precancerous lung lesions caused by cigarette smoking. Cancer control techniques such as the initial prevention measures (e.g., stopping smoking), precancerous asymptomatic cytologic and radiologic examinations, and early symptomatic diagnosis are reviewed. Early symptomatic diagnosis is believed to be the most beneficial technique leading to early treatment and rehabilitation of the patients. World Health Organization lung cancer mortality rates during the period 1970 to 1972 are shown for men in several European countries, the United States, and Canada.

See also, **80-0751, 80-0753**

80-0762. Stevens, R. G.; Moolgavkar, S. H. **Estimation of Relative Risk From Vital Data: Smoking and Cancers of the Lung and Bladder.** *Journal of the National Cancer Institute* 63(6): 1351-1357, December 1979.

Mortality data from cancers of the lung and bladder in England and Wales were analyzed. With the use of detailed information on cigarette consumption, a nonlinear least-squares analysis showed that the differences between males and females in the rates of these cancers could be explained on the basis of differences in smoking habits. Furthermore, estimates of the relative risk due to smoking were obtained. The relative risks of smoking 146,000 cigarettes (equivalent to 20 cigarettes/day for 20 years) were 4.3 for lung cancer and 2.9 for bladder cancer. These estimates agree with those obtained by other types of epidemiologic studies. (Auth. Abs.)

NON—NEOPLASTIC RESPIRATORY DISEASES

80-0763. Bagnato, A.; Colle, B.; De Marchi, D.; Talmassons, G. **Pollution du Milieu et Tabagisme: Recherches Effectuees sur Trois Populations de Travailleurs Exposes aux Differents Risques Pulmonaires. [Environmental Pollution and Smoking: Research on Three Population Samples of Workers Exposed to Different Pulmonary Risks.]** *Broncho-Pneumologie* 28(4): 272-277, July-August 1978, French.

Smoking as a risk factor for bronchopulmonary disorders was studied in three groups of workers exposed to various kinds of environmental pollution. Workers were selected from textile (71), machine (40), and metallurgy (561) industries; of these workers, the percentages of smokers were 40, 63, and 56, respectively. A smoking index based on the percent of smokers, number of cigarettes smoked per day, and years of smoking was calculated for each group. This index varied from 209 for the workers in the metallurgy industry to 51 for those in the textile industry. The metallurgy employees were subgrouped by machine shop, rolling mill, and foundry, and respiratory data were collected. In the first two subgroups, the difference in chronic bronchitis severity between smokers and nonsmokers was statistically significant. However, this difference was not significant in foundry workers who were exposed to the maximum pollution. Similar results were obtained from comparison of the objective indices of bronchopulmonary lesions, namely, maximum expiratory volume in 1 sec (MEV_1) and residual volume (V_R), and Tiffeneau and Motley indices. The MEV_1 percentage of the theoretical value for smokers vs. nonsmokers in the rolling mill subgroup was -5.423 vs. 6.211, respectively. This difference, as well as the difference in MEV_1 between smokers and nonsmokers of the machine shop subgroup, was significant. The difference between smokers and nonsmokers increased in significance with the smoking index which varied from 203 for the machine shop to 310 for the rolling mill. Nonsignificant values were found for smokers and nonsmokers in the foundry. The smoking hazard was not evident in the workers exposed to a very polluted environment.

80-0764. Brown, P.; Gajdusek, D. C. **Acute and Chronic Pulmonary Airway Disease in Pacific Island Micronesians.** *American Journal of Epidemiology* 108(4): 266-273, October 1978.

The populations of Mogmog and Falalop Islands of Ulithi Atoll, and of Ifaluk Atoll, in the Western Caroline Islands (481 people of all ages) were questioned and examined for the presence of asthma, chronic bronchitis, and chronic obstructive airway disease (COAD). Adults were also given spirometry tests. Of the children, 75 percent under 5 years of age were found to have asthma, declining to an adult level of 20 to 30 percent by late adolescence, with attacks predominantly linked to periodic epidemics of upper respiratory viral infections. Over one-third of adults, and almost one-half of adults over the age of 40, were affected by chronic bronchitis, often with an asthmatic component, and typical COAD occurred in almost one-third of the population over 50, mostly in males. Although 90 percent of the adult population smoked an average of one pack of cigarettes daily (as many women smoked as men), respiratory disease occurred in nonsmokers as frequently as in smokers. Alpha-1 antitrypsin deficiency was excluded in the finding of 100 percent homozygosity for the Pi M allele. Infection by *Mycobacterium* *tuberculosis* is common, but active pulmonary tuberculosis, while previously a serious problem, is at present only occasionally found on these islands, and atypical mycobacterial and fungal pulmonary infections do not appear to occur. Pulmonary airway diseases constitute the most important causes of morbidity and mortality in this population. (Auth. Abs. Mod.)

80–0765. Semple, P. d'A. **Alpha-1-Antitrypsin Deficiency and Chest Disease. A Clinical and Physiological Study.** *Scottish Medical Journal* 23(4): 281-285, October 1978.

Four families with alpha-1-antitrypsin deficiency have been identified. Of seven homozygous subjects five smoked and had chest disease while the two nonsmokers were unaffected. Productive cough was a feature in four of the five affected homozygotes and symptoms commenced earlier than is usual with chronic bronchitis and emphysema. Of 11 heterozygous subjects only 5 who smoked had symptomatic or laboratory evidence of obstructive airways disease, in each case less marked than that of their homozygous smoking relatives. Severely affected subjects were underweight, the degree of weight loss being related to the measured severity of airways disease. A survey of emphysematous patients screened for alpha-1-antitrypsin deficiency is described. The natural course and significance of this enzyme deficiency are examined with reference to previous literature. (Auth. Abs.)

80–0766. Field, G. B.; Owen, P. **Respiratory Function in an Australian Cotton Mill.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(3): 455-468, 1979.

A survey was conducted in an urban cotton mill employing 135 males and 236 females. A stratified random sample comprising 252 employees was allocated to 21 time slots defined by hours 1-7 of the work shift and Monday, Wednesday, and Friday of the working week. Each slot contained 12 employees balanced for sex and length of service. Data were provided by standard questionnaires, spirometry, and flow-volume curves from breathing air and a helium-oxygen mixture. After adjustment for height, age, and smoking habit, the data revealed linear and curvilinear decreases in respiratory function values over hours 1-7 of the work shift. These trends were more marked in the males. A tachyphylactic type of response was observed, the decline in respiratory function being greatest on Monday and least on Friday. The magnitude of the Monday fall in respiratory function in the males was greater than has been reported in other surveys with comparable dust levels, but the prevalence of byssinosis was only 0.8 percent. The trends in respiratory function over the work shift suggested that the reaction to cotton dust commenced in the distal airways and spread to involve the proximal airways as bronchoconstriction increased. Respiratory function in employees with a history of chest tightness was materially less impaired than in current cigarette smokers or employees with regular cough and sputum. Current smokers had a significantly lower forced expiratory volume in 1 sec and peak expiratory flow rate than nonsmokers ($p<0.001$). (Auth. Abs. Mod.)

80–0767. Helsing, K. J.; Comstock, G. W.; Speizer, F. E.; Ferris, B. G.; Lebowitz, M. D.; Tockman, M. S.; Burrows, B. **Comparison of Three Standardized Questionnaires on Respiratory Symptoms.** *American Review of Respiratory Disease* 120(6): 1221-1231, December 1979.

A new questionnaire (ATS-DLD) developed by the American Thoracic Society (ATS) for the Division of Lung Diseases (DLD) of the National Heart, Lung, and Blood Institute, designed to elicit more details about various symptoms and factors associated with chronic obstructive pulmonary diseases, was compared with two previous questionnaires, those developed by the British Medical Research Council (MRC) and by the National Heart and Lung Institute (NHLI). From a private census list of residents of Washington County, Md., six similar population samples of white men and women 25 to 64 years of age were selected, one each for administration of the three questionnaires by telephone and by mail. The six groups were similar with respect to sex and smoking habits in 1975 when the subjects were selected. Heavy cigarette smokers (≥40/day) constituted 21.5 to 25.4 percent of each of the six groups, moderate smokers (20-39/ day) 21.5 to 25.1 percent, light smokers (10-19/day) 24.1 to 27.5 percent, and nonsmokers 25.1 to 27.7 percent. A total of 1,004 interviews were obtained. Although the study groups were nearly identical with respect to their smoking status in 1975, this did not guarantee similarity with respect to smoking at the time the questionnaires were completed. The questionnaire responses indicated that 1 percent of the nonsmokers in 1975 were light-to-moderate smokers in 1977, and 8 percent of the smokers had quit. Indicated prevalences of chronic cough and chronic phlegm were essentially the same for all three questionnaires; the ATS-DLD elicited significantly more symptoms of any wheeze, whereas the NHLI elicited more mild breathlessness. The MRC and ATS-DLD were virtually identical in indicated prevalences of chronic wheeze, asthma, and hay fever, conditions not covered in the NHLI. All three questionnaires showed similar increases in symptoms with increased cigarette smoking and with decreased forced expiratory volume in 1 sec. Among the interviews conducted by telephone, the NHLI showed a greater variation in symptomatology by interviewer than either the ATS-DLD or MRC, although sampling variation could not be ruled out as a contributory factor. Among the questionnaires self-administered by mail, the ATS-DLD showed the lowest percentage of misunderstood questions, with NHLI next, and MRC significantly higher. (Auth. Abs. Mod.)

80–0768. Higenbottam, T.; Hamilton, D.; Feyerband, C.; Clark, T.J.H. **Acute Effects of Smoking a Single Cigarette on the Airway Resistance and the Maximal and Partial Forced Expiratory Flow Volume Curves.** *British Journal of Diseases of the Chest* 74(1): 37-46, January 1980.

The acute airway response to smoking a single cigarette was assessed in eight symptom-free male smokers. The changes in airway function were determined using a computer analysis of maximal and partial expiratory flow volume curves together with measurement of airway resistance. The amount of smoke inhaled was assessed by measuring levels of nicotine in venous blood. When the results of all studies were analyzed for the group as a whole, only small and inconsistent changes in airway function were observed after smoking a cigarette, although all the subjects appeared to inhale the smoke. Although the group showed inconsistent changes, one subject exhibited a change on all four occasions in the studies using the maximal and partial expiratory flow volume curves. This was seen particularly for maximal flow at 50 and 40 percent of vital capacity obtained from the maximal and partial expiratory flow volume curves, respectively. There appeared to be no difference between the two maneuvers in their sensitivity to smoke-induced change. One subject showed a response to smoking when airway resistance was measured, but this was on only one or two occasions. It is therefore possible that certain smokers are more susceptible to inhaled cigarette smoke, and such individuals may be those who develop chronic airflow obstruction. (Auth. Abs. Mod.)

80–0769. Hocking, W. G.; Golde, D. W. **The Pulmonary-Alveolar Macrophage. (Second of Two Parts).** *New England Journal of Medicine* 301(12): 639-645, September 20, 1979.

This part of a two-part review summarizes the present knowledge of pulmonary alveolar macrophages, with emphasis on function. The pulmonary alveolar macrophage (PAM) has important immunologic functions in pulmonary defense: participation in specific immunologic reactions and interaction with other immune system components. Elastase activity, the essential proteolytic component in the production of emphysema, has been demonstrated both in PAMs and granulocytes; PAMs from smokers have elevated elastase and lysosomal hydrolase activities. Studies have shown an increased glucose utilization in PAMs from smokers. Other effects of chronic cigarette smoking on PAM function include increased adherance to glass, increased random migration, diminished response to macrophage migration-inhibitory factor, and impairment of the ability to enhance lymphocyte proliferation. Smoking was also found to induce aryl hydrocarbon hydroxylase in PAMs.

80–0770. Jones, J. G.; Lawler, P.; Crawley, J.C.W.; Minty, B. D.; Hulands, G.; Veall, N. **Increased Alveolar Epithelial Permeability in Cigarette Smokers.** *Lancet* 1(8159): 66-67, January 12, 1980.

Two indices of alveolar epithelial permeability were derived in man by a noninvasive method. The rate of transfer of ^{99m}Technetium-labeled diethylenetriamine penta-acetate from the lung into the blood was measured and a significantly greater ($p<0.001$) transfer rate was found in symptomless cigarette smokers compared with nonsmokers. Although carbon monoxide is the most likely cause of the increase in permeability of the alveolar epithelium in smokers, other possible mechanisms include the action of nicotine, catecholamines, nitrogen oxide, nitrogen dioxide, and other constituents of cigarette smoke. The effect on the alveolar epithelium was slowly reversible in one subject: the clearance half-time rose from 21 min to 51 min within 3 months of stopping smoking. Smoking 20 cigarettes in 4 hours did not change permeability in the one subject studied. (Auth. Abs. Mod.)

80–0771. Jones, R. N.; Diem, J. E.; Glindmeyer, H.; Dharmarajan, V.; Hammad, Y. Y.; Carr, J.; Weill, H. **Mill Effect and Dose—Response Relationships in Byssinosis.** *British Journal of Industrial Medicine* 36(4): 305-313, November 1979.

Four hundred and eighty-six textile workers in three cotton mills and one wool/synthetic mill were studied for symptoms and functional effects of workroom exposure to dust. Byssinosis was found in 5.7 percent of 386 cotton workers, with an apparent threshold level of 0.5 mg cotton dust/m^3 of air. Smoking status (smoker and nonsmoker) and observed and estimated rates of byssinosis by current dust exposure category were analyzed by hierarchical log-linear modeling. In this analysis the prevalence of byssinosis is significantly ($p=0.005$) affected by exposure level beyond the effect of smoking. Smoking has a significant effect on the forced expiratory flow at 25 to 75 percent ($p=0.01$) and on the ratio of forced expiratory volume at 1 sec to forced vital capacity ($p=0.006$). Mean post-shift functional declines were greater in workers exposed to ≥ 0.2 mg/m^3. Workers with byssinosis were unequally distributed, however, with respect to job category and mill; and these variables, rather than current dust exposure levels, accounted for the observed distribution of byssinosis prevalence rates. Mill effect should be assessed in large-scale studies of byssinosis, most of which have analyzed biological response rates by combining mill and other variables to examine first-order effects of dust dosage. In such analyses, much of the observed variability may be due to factors other than dust dosage. (Auth. Abs. Mod.)

80–0772. Lowhagen, O.; Granerus, G.; Wetterqvist, H. **Studies on Histamine Metabolism in Intrinsic Bronchial Asthma.** *Acta Allergologica* 34(6): 395-404, December 1979.

Histamine metabolism was investigated in patients with intrinsic asthma over a 7-day period, during the acute phase and in remission, by determination of the urinary excretion of histamine metabolites. During acute attacks the excretion of methylhistamine (MeHi) and 1-methyl-4-imidazoleacetic acid (MeImAA) was significantly increased compared with healthy subjects. The excretion of MeHi was significantly increased during acute attacks compared with remission. The excretion of MeImAA was also increased, but the difference was not significant. The correlation between the excretion of MeHi and MeImAA was highly significant. Corticosteroids, given to some patients during remission, did not affect the excretion of histamine and its metabolites. Smoking and respiratory infection during the acute attacks were both accompanied by increased excretion of MeHi. The mean values of MeHi on days 2 to 7 were higher than in nonsmokers, but the differences were not significant. The mean values of MeHi in patients who were both smokers and infected, were higher than in those who were nonsmokers and noninfected during days 2 to 6. The difference was significant during days 3 and 4. The mean values of MeHi in both nonsmoking and noninfected patients were still significantly higher than the control values (where 7 out of 20 subjects were smokers) on days 1 to 5. The results of the study indicate that there is an increased histamine turnover in acute attacks of intrinsic bronchial asthma. (Auth. Abs. Mod.)

80-0773. Meadows, J. A., III.; Rodarte, J. R.; Hyatt, R. E. **Density Dependence of Maximal Expiratory Flow in Chronic Obstructive Pulmonary Disease.** *American Review of Respiratory Disease* 121(1): 47-53, January 1980.

Pulmonary mechanics were studied in 22 patients (4 women and 18 men) with chronic obstructive pulmonary disease (COPD) who had no evidence of asthma. Eleven of the 22 patients had a density dependence of maximal expiratory flow within the reported normal range. Patients with normal density dependency (responders) were not different from those with decreased density dependence (nonresponders) in mean age, smoking history (although the nonresponders tended to have smoked more), symptoms of cough or dyspnea, maximal expiratory flow at 75, 50, and 25 percent of the vital capacity during air breathing, total lung capacity, or static elastic lung recoil at 50 percent of the vital capacity. The nonresponders had smaller vital capacity values, larger ratios of residual volume to total lung capacity, higher resistance, and lower static recoil at total lung capacity. There was a correlation between diffusing capacity for carbon monoxide and density dependence. There was an inverse relationship between the change in density dependence after inhalation of isoproterenol and initial density dependence. Dysanaptic lung growth and increased compliance of the central airways are potential mechanisms for normal density dependence in these patients. The prevalence of normal density dependence in these patients with established COPD raises serious questions concerning the usefulness of density dependence in the early detection of COPD. The differences in density dependence may indicate differences in pathogenesis of the airflow obstruction and hence may be of prognostic significance. (Auth. Abs. Mod.)

80-0774. Mimica, M.; Saric, M.; Malinar, M. **Pusenje Duhana kao Uzrok Kronicne Nespecificne Bolesti Pluca. [Tobacco Smoking as a Cause of Chronic Nonspecific Lung Disease.]** *Arhiv za Higijenu Rada i Toksikologiju* 29(4): 289-304, 1978, Serbo-Croatian.

The effects of cigarette smoking on respiratory symptoms and ventilatory impairments were studied in a sample of 1,575 men and 1,690 women, aged 38-57 years. There were 490 nonsmokers, 263 ex-smokers, and 822 smokers among the men, and 1,386 nonsmokers, 54 ex-smokers, and 250 smokers among the women. The clinical and epidemiological study was done twice over a 3-year interval. The answers to a questionnaire on respiratory symptoms revealed that chronic cough incidence in both men and women increased steadily with the number of cigarettes smoked daily up to 30. This incidence, however, decreased slightly in the subjects who smoked more than 30 cigarettes/day. Cough was nearly five times more frequent in smokers of 21 to 30 cigarettes/day than in nonsmokers (52.3 and 36.5 percent for men vs. 10.8 and 7.4 percent for women). Dyspnea during exertion and asthma symptoms were only twice as frequent in smokers of the same group than in nonsmokers. Bronchial rales and prolonged expiration were 3-5 times more frequent in heavy smokers than in nonsmokers. Ventilatory tests showed that the mean forced expiratory volume in 1 sec (FEV_1) and forced vital capacity (FVC) values decreased with the increase in daily cigarette consumption. However, the difference was only 5 to 10 percent between the heaviest smokers and nonsmokers. Over a 3-year period, the difference in FEV_1 of the expected normal value between smokers and nonsmokers increased regularly with increased daily cigarette consumption. In men FEV_1 increased by a factor of 1.6 from nonsmokers to smokers of over 30 cigarettes/day. Male smokers who inhaled deeply complained about chronic cough more often than those who did not inhale (47.5 vs. 30.9 percent). Unexpectedly, FEV_1 and FVC values were better in smokers who inhaled than in those who did not. Also, the mean ventilatory functions were slightly but significantly lower in smokers of low-quality oriental tobacco than in those who smoked "blend" cigarettes. It is concluded that long-term smoking contributes, although not dramatically, to the impairment of respiratory organs in most smokers. However, smoking may stimulate respiratory functions to some degree in some smokers.

80-0775. Palmer, A.; Renzetti, A. D., Jr.; Gillam, D. **Respiratory Disease Prevalence in Cosmetologists and Its Relationship to Aerosol Sprays.** *Environmental Research* 19(1): 136-153, June 1979.

A randomized, cross-sectional epidemiologic study of the prevalence of respiratory disease from exposure to hair spray in cosmetologists was conducted in the State of Utah in all but the sparsely populated western regions of the State. The sample selected for survey consisted of 262 student cosmetologists, 213 graduate practicing beauticians, and 569 females not exposed to hair sprays as part of their occupation (except that of housewife), matched for age, smoking history, and geographic location. Of the cosmetologists, 72.7 percent were nonsmokers, 16.6 percent smokers, and 10.7 percent ex-smokers. Study methods included a modified British Medical Research Council respiratory symptom questionnaire, chest X-ray, forced expiratory spirogram, and measurements of individual and environmental hair spray particulate exposure concentrations. Additionally, for a subgroup in the study population closing volume, slope of phase III and closing capacity of the lung and carbon monoxide diffusing capacity were measured, and sputum cytology was studied. Study findings indicated that, in comparison to the control population, cosmetologists have an increased prevalence of chronic respiratory disease symptoms, small airway obstruction, and atypical sputum cytology and that these increases were significantly related to duration of occupational exposure. The findings with regard to thesaurosis, although suggestive of a positive relationship with hair spray exposure of long duration, were not conclusive. Significantly more respiratory symptoms were found in smokers as compared to ex-smokers and nonsmokers ($p<0.001$). Atypical sputum cytology was also most prevalent in smokers (38.8 percent), followed by nonsmokers (29 percent), and by ex-smokers (22.5 percent). (Auth. Abs. Mod.)

80–0776. Rossiter, C. E.; Harries, P. G. **UK Naval Dockyards Asbestosis Study: Survey of the Sample Population Aged 50-59 Years.** *British Journal of Industrial Medicine* 36(4): 281-291, November 1979.

As part of a general morbidity study of all civilian employees in the four Royal Naval Dockyards, the clinical, radiological, and physiological effects of exposure to asbestos in 1,200 men aged 50-59 years were studied in detail. The sample included all men on the Register of Asbestos Workers, one in three of those currently in occupations where intermittent exposure to asbestos may occur, and 1 in 30 of the remainder. The conclusions are mainly in accord with those of the comprehensive morbidity study of all the civilian dockyard workers, and show that smoking played a large part in increasing prevalence rates of radiographic, clinical, and physiological abnormalities in this population. A subgroup of 39 men, who were working as asbestos laggers or sprayers before 1957, was identified. These men showed much more extensive disease than any other subgroup, and the smokers were worse than the nonsmokers. Of these smokers, 48 percent had small opacities of category 1/1 or more, 76 percent reported that they coughed during the day, and 53 percent had crepitations; forced expiratory volume and transfer factor were also markedly reduced in these 21 smokers. Although asbestos exposure had been intermittent for the majority of the population, the prevalences of pleural thickening, small opacities, current respiratory symptoms and crepitations were shown to be related to duration of exposure to asbestos. (Auth. Abs.)

80–0777. Samet, J. M.; Epler, G. R.; Gaensler, E. A.; Rosner, B. **Absence of Synergism Between Exposure to Asbestos and Cigarette Smoking in Asbestosis.** *American Review of Respiratory Disease* 120(1): 75-82, July 1979.

To assess both independent and synergistic effects of exposure to asbestos and cigarette smoking on the development of asbestosis, survey data from four groups of workers exposed to asbestos were analyzed with multivariate statistical models. Survey methods were standardized and included for the 383 subjects a respiratory symptoms questionnaire, occupational history, physical examination, pulmonary function testing, and a chest radiograph. Exposure to asbestos and cigarette smoking were assessed by questionnaire. Synergism between the two exposures was not present for previously identified manifestations of asbestosis including bilateral fine crackles, clubbing, dyspnea, radiographic abnormality, decreased forced vital capacity, and decreased single-breath diffusing capacity of the lung for carbon monoxide. However, independent additive effects of these two exposures were present for each of these parameters. (Auth. Abs.)

80–0778. Stanhope, J. M.; Rees, R. O.; Mangan, A. J. **Asthma and Wheeze in New Zealand Adolescents.** *New Zealand Medical Journal* 90(645): 279-282, October 10, 1979.

Two epidemiological surveys of New Zealand adolescents are reported. The prevalence of asthma was 7.5 percent in Rotorua and 7.9 percent in Wairoa. Current cigarette smoking was more prevalent in inactive asthma and wheeze groups than others ($p<0.003$). Father's and elder sibling's smoking had no significance, but mother's smoking showed an interaction effect with subject's smoking such that inactive asthma was 2.3 times more common in smoking children of smoking mothers than in all other subjects. When this group was excluded, no significant association between asthma/wheeze and smoking remained. (Auth. Abs. Mod.)

80–0779. Vezzoli, F.; Calienno, A.; Longhini, E. **Small Airways Disease: A Trial of an Easy Functional Discrimination of Preclinical Emphysema.** *Respiration* 37(5): 282-290, 1979.

In the genesis of small airways obstruction (SAO) two alterations with different prognoses have to be considered: the first, in which the obstruction depends on a reduction of the diameters of the small bronchi, is reversible; the second, probably irreversible, is due to pulmonary emphysema in its preclinical stage. To distinguish between these two mechanisms, 5 normal, male, nonsmokers (N) and 10 male smokers considered to be affected by SAO were studied by means of lung function tests. All of the tests showed a significant difference between SAO subjects (smokers) and nonsmokers. It is suggested that among various respiratory function tests there are some which have a prognostic value and seem to be coupled to a qualitative meaning: this is the case for the single-breath nitrogen test which is significantly greater in subjects with irreversible abnormality. Other tests are very sensitive to detect SAO subjects but are lacking in specificity and in prognostic meaning (closing volume, maximum expiratory flow, frequency dependence of compliance). Finally, other tests (maximal mid-expiratory flow and residual volume) seem to have a prognostic value only because they indicate an advanced stage of the illness independent of the kind of impairment. (Auth. Abs. Mod.)

80–0780. Vraney, G. A.; Pokorny, C. **Pulmonary Function in Patients With Gastroesophageal Reflux.** *Chest* 76(6): 678-680, December 6, 1979.

Gastroesophageal reflux may play a causal role in a variety of chronic pulmonary disorders. In an effort to determine whether patients with reflux as a group demonstrated a significant degree of pulmonary dysfunction, 100 patients (48 women and 52 men, with 93 percent of the group in excess of 40 years of age) with reflux were compared with another group of 100 control patients, matched for age and sex and without demonstrable reflux. Standard pulmonary function studies were used for this comparison. No difference could be detected between the two groups, which suggested that the isolated findings of reflux in patients with pulmonary disorders should not be considered etiologic by virtue of the association alone. Of the patients with reflux, 29 percent were cigarette smokers; in the nonreflux group, 39 percent were smokers. The difference in abnormal flow rates noted between the smoker and nonsmoker groups was slightly greater than that between the reflux and nonreflux groups, but no statistically significant difference was observed among the groups. (Auth. Abs. Mod.)

80–0781. Walter, S. **The Immediate Airway Response to Cigarette Smoking.** *Indian Journal of Medical Research* 70: 819-824, November 1979.

The immediate effects of smoking two cigarettes, on the peak flow rates and forced expiratory spirograms, were studied in 26 young, asymptomatic male smokers. The subjects had smoking histories of 2.4 ± 1.7 years and had smoked a total of 1045.8 ± 872.3 packs of cigarettes (one pack containing 10 cigarettes) during their lifetime. Among the subjects, 14 smoked non-filtertipped and 12 smoked filter-tipped cigarettes. There was a significant decrease in flow rates at high lung volumes but not in those at low lung volumes. In the 14 subjects who smoked non-filtertipped cigarettes, there was also a significant fall in forced vital capacity ($p<0.01$) and in the forced expiratory volume in one second (FEV_1) ($p<0.05$). The fall in peak expiratory flows (PEF) observed in the 26 subjects had a significant correlation ($p<0.05$) with the presmoking FEV_1; those who showed a greater fall in PEF had a lower presmoking FEV_1. These findings indicate that the immediate response of the large airways to smoking is constriction. The constrictor effect on the small airways is probably masked by other opposing mechanisms. The data suggest that (1) the immediate response to smoking may help to identify smokers who are more prone to develop chronic obstructive pulmonary disease, and (2) although cigarettes are filtered, significant amounts of bronchoconstrictor agents remain. (Auth. Abs. Mod.)

See also, **80–0736, 80–0801**

CARDIOVASCULAR DISEASES

80–0782. Aronow, W. S. **Effect of Non-Nicotine Cigarettes and Carbon Monoxide on Angina.** *Circulation* 61(2): 262-265, February 1980.

Non-nicotine Mint Bidis cigarettes made from Indian herbal leaves were used to study the effect of smoking five non-nicotine cigarettes and of breathing carbon monoxide on exercise-induced angina in 12 male angina patients, all of whom smoked one package of cigarettes daily. Smoking increased venous carboxyhemoglobin from 1.71 to 5.35 percent, decreased exercise duration until angina 45 percent, increased ischemic ST-segment depression at angina from 1.33 to 1.52 mm, and decreased systolic blood pressure times heart rate at angina. Breathing carbon monoxide increased venous carboxyhemoglobin from 1.73 to 5.37 percent, decreased exercise duration until angina 35 percent, increased ischemic ST-segment depression at angina from 1.31 to 1.50 mm, and decreased systolic blood pressure times heart rate at angina. Greater decreases in exercise duration until angina and in systolic blood pressure times heart rate at angina ($p<0.001$) were observed after smoking than after breathing carbon monoxide. Tobacco components other than nicotine or carbon monoxide are responsible for a small decrease in exercise performance until angina. (Auth. Abs. Mod.)

80–0783. Carlson, L. A.; Bottiger, L. E.; Ahfeldt, P.-E. **Risk Factors for Myocardial Infarction in the Stockholm Prospective Study. A 14-Year Follow-up Focussing on the Role of Plasma Triglycerides and Cholesterol.** *Acta Medica Scandinavica* 206(5): 351-360, 1979.

A 14-year followup of the Stockholm prospective study is reported. A number of 130 new myocardial infarctions (MI) were found in a prospective group of men ($n=3,189$) and another 46, i.e., a total of 176 MI, in the total group ($n=3,486$). The rate of MI/1000 in relation to smoking was 56 ($p<0.001$). In a stepwise multiple regression analysis, age was the most important single independent risk factor followed by systolic blood pressure (BP), smoking, plasma triglycerides, erythrocyte sedimentation rate, and hemoglobin level. Elevated BP became an important risk factor only after the age of 50. When only age, BP, smoking, and the two plasma lipids were entered into the logistic multivariate analysis, plasma triglycerides were more important as a risk factor than cholesterol. (Auth. Abs. Mod.)

80–0784. Estrada, M. L.; Anastacio, R. V. **Premature Coronary Artery Disease and Smoking.** *Philippine Journal of Internal Medicine* 16: 151-167, October-December 1978.

In an effort to identify the potential young coronary patient and, in particular, to ascertain the role of smoking as a coronary disease risk factor, 56 healthy men (both smokers and nonsmokers) aged 22-28 years underwent cardiopulmonary function tests. Other coronary risk factors of the subjects, such as hypertension, diabetes mellitus, and coronary artery disease were also identified. To determine the acute effects of smoking on cardiopulmonary function, each subject was asked to smoke a cigarette. Among the risk factors for coronary artery disease, the incidence of smoking was highest (68 percent). Eighteen (31 percent) were identified as potential coronary artery disease patients, as evidenced by ischemic changes in the exercise electrocardiogram. The majority of them (77 percent) were chronic smokers (more than 5 years) and heavy smokers (≥20 cigarettes/day). There was no statistically significant difference in blood pressure and heart rate between smokers and nonsmokers before or after smoking. Analysis of the electrocardiogram after smoking revealed an earlier appearance of ischemic changes at a lower work load, especially among smokers with initially abnormal ST segments.

80–0785. Garg, J. P.; Chaturvedi, M. K.; Singh, V. **Immediate Effects of Cigarette Smoking on Pulse, B.P., and E.C.G. in Healthy, Young, Asymptomatic Smokers.** *Clinician* 42(9): 342-345, September 1978.

The immediate effects of cigarette smoking on pulse, blood pressure, and electrocardiogram (ECG) were studied in 183 asymptomatic smokers and 23 ex-smokers, all of whom were healthy young men between 19 and 30 years of age. Blood pressure, pulse, and ECG were taken before and 10 min after the subjects had smoked two cigarettes. After smoking, there was a highly significant increase in pulse rate ($p<0.001$) which was greater in light smokers (5-15 cigarettes/day) than in heavy smokers (>15 cigarettes/day). A highly significant rise in blood pressure ($p<0.001$) was observed in all subjects, irrespective of smoking habits. The ECG showed an increase in heart rate immediately after smoking. Generalized decrease in amplitude of T-wave with or without S-T segment depression was noted in 7 light smokers (6.36 percent), 13 heavy smokers (17.78 percent), and 1 ex-smoker (4.34 percent).

80–0786. Hall, R. G.; Sachs, D.P.L.; Hall, S. M. **Medical Risk and Therapeutic Effectiveness of Rapid Smoking.** *Behavior Therapy* 10: 249-259, 1979.

To assess the potential medical risks of rapid smoking, 24 healthy, male cigarette smokers were examined under the following conditions: baseline (after 12-hour tobacco abstinence), normal smoking, and rapid smoking. After normal and rapid smoking standard 12-lead electrocardiogram (EKG), arterial blood gases, and vital signs were evaluated, and during rapid smoking each subject was monitored electrocardiographically throughout the session. During or after rapid smoking there were no EKG abnormalities. Heart rate at normal and rapid smoking differed significantly from baseline and from each other ($p<0.05$). Both smoking conditions (normal and rapid) increased respiratory rate and systolic blood pressure compared to baseline ($p<0.05$), but differences between normal and rapid smoking were not significant for either variable. Diastolic blood pressure was not affected by either smoking condition. Rapid smoking produced significant differences in serum potassium concentrations (compared to normal smoking, $p<0.05$), arterial blood, pH, and arterial carbon dioxide tension (compared to baseline and normal smoking, $p<0.05$). Changes in measured arterial oxygen saturation were minimal and not significant, whereas there was a significant rise in carboxyhemoglobin from baseline to normal to rapid smoking ($p<.05$). Many subjects developed lowered arterial serum potassium after rapid smoking, and two-thirds of the subjects developed relative hypoxemia. Among those who completed treatment, 61 percent were still abstaining from cigarettes after 6 months. Rapid smoking was found to be safe for healthy subjects. However, the safety of the technique among persons with cardiopulmonary disease cannot be predicted from the results of this study. (Auth. Abs. Mod.)

80–0787. Haynes, S. G.; Feinleib, M. **Women, Work and Coronary Heart Disease: Prospective Findings From the Framingham Heart Study.** *American Journal of Public Health* 70(2): 133-141, February 1980.

This study examined the relationship of employment status and employment-related behaviors to the incidence of coronary heart disease (CHD) in women. Between 1965 and 1967, a psychosocial questionnaire was administered to 350 housewives, 387 working women (women who had been employed outside the home over one-half their adult years), and 580 men participating in the Framingham Heart Study. The respondents were 45-64 years of age and were followed for the development of CHD over the ensuing 8 years. Mean levels of six risk factors (age, systolic and diastolic blood pressure, serum cholesterol, cigarette smoking, and glucose intolerance) were measured. Working women did not have significantly higher incidence rates of CHD than housewives (7.8 vs. 5.4 percent, respectively). However, CHD rates were almost twice as great among women holding clerical jobs (10.6 percent) as compared to housewives. The most significant predictors of CHD among clerical workers were suppressed hostility, having a nonsupportive boss, and decreased job mobility. CHD rates were higher among working women who had ever married, especially among those who had raised three or more children. Among working women, clerical workers who had children and were married to blue collar workers were at highest risk of developing CHD (21.3 percent). (Auth. Abs. Mod.)

80-0788. Lowe, G.D.O.; Drummond, M. M.; Forbes, C. D.; Barbenel, J. C. **The Effects of Age and Cigarette-Smoking on Blood and Plasma Viscosity in Men.** *Scottish Medical Journal* 25(1): 13-17, January 1980.

Blood viscosity and its major determinants (hematocrit, plasma fibrinogen, and plasma viscosity) were measured in 90 apparently healthy men aged 16-80 years. Cigarette smokers ($n=45$) had higher levels of blood viscosity, hematocrit, fibrinogen ($p<0.001$), and plasma viscosity ($p<0.025$) than nonsmokers ($n=45$). Blood viscosity was still higher in smokers after correction to a standard hematocrit ($p<0.02$). Fibrinogen, corrected blood viscosity, and plasma viscosity rose with age in both groups, but young smokers had prematurely elevated levels of these variables and less pronounced rises with age. These results show that age and cigarette smoking must be considered in studies of blood and plasma viscosity as they provide further evidence for an association between viscosity and arterial disease. (Auth. Abs.)

80-0789. Matzdorff, F. **Kriterien fur die Spatprognose nach Herzinfarkt. [Criteria for Long-Term Prognosis Following Myocardial Infarct.]** *Lebensversicherungs Medizin* 29(5): 129-132, September 1977, German.

Results of three studies of patients who survived a myocardial infarct are published. In the first study, 476 male patients aged 30-64 years were examined at least 6 months after the first infarct and followed up for 3 years. In this group, 61 patients died within 3 years (a mortality rate of 4.3 percent per year), and 47 of them died from a second infarct. The incidence of reinfarction was 5 percent/year with a 67 percent mortality rate. Among the known risk factors of a first infarct only smoking appeared to increase significantly the risk of reinfarction. For smokers vs. nonsmokers, 20.9 vs. 12.9 percent, respectively, experienced a second infarct within 3 years. The incidence of a second infarct was no higher in patients who stopped smoking immediately after the first infarct than in those who did not smoke even before the first infarct. The larger the heart volume in these patients, the more frequent the second infarct and the smaller the chance of survival. In the second study, the prognostic significance of the stress test was studied over a 2- to 4-year period in 447 male patients aged 40- 59 years. It was determined that the incidence of a second infarct increases with decreasing cardiac reserve. Therefore, heart enlargement and ischemia reaction in the stress test were the most decisive criteria of reinfarction risk. In the third study, the duration of disability following a myocardial infarct was evaluated in a sample of 200 patients referred to the clinic within 3 months after the infarct and 200 other patients who started therapy after 12 months or later.

80-0790. Poston, R. N. **Immunological Aspects of Atheroma: A Review.** *Journal of the Royal Society of Medicine* 72(9): 674-682, September 1979.

The role of immune complexes as atherogenesis potentiators in experimental animals is well-established. However, it has been difficult to investigate the role of tobacco allergies in coronary disease even though several studies have shown that tobacco allergy incidence is clearly associated with thromboangiitis obliterans (69- 80 percent greater incidence above that in control smokers). The possible pathogenic mechanism of tobacco allergy involvement with thromboangiitis obliterans is release of platelet activating factor from basophils sensitized with cytophilic IgE antibody, on contact with the allergen. Thrombosis can be directly induced by this mechanism, and an increased ease of platelet aggregation has been seen after cigarette smoking. Smoking may also enhance thrombosis by activating clotting factors and by affecting prostaglandin metabolism.

80-0791. Romo, M.; Ruosteenoja, R. **Sudden Coronary Death: Incidence and Common Risk Factors.** *Advances in Cardiology* 25: 1-8, 1978.

Sudden death is very common in coronary heart disease. At least 40-50 percent (in males over 50 percent) of all deaths occur within 1 hour of onset of acute heart attack symptoms, and about one-third of deaths occur within 15 min. Sudden instantaneous death, within min or 1 hour, is more common in males than in females. The tendency to sudden death during an acute heart attack is greater at younger ages than in the elderly. Male preponderance is evident in all age groups, being least in the oldest age groups. Female incidence lags 15-20 years behind that of males, and so sudden coronary death appears to be particularly common in middle-aged men. Common risk factors for coronary heart disease are encountered more often among the fatal than the nonfatal cases of acute heart attack. To determine those specific for sudden death, one study compared three groups of cases, i.e., sudden deaths, nonsudden deaths, and nonfatal cases. Among males, hypertension and heavy smoking (>25 cigarettes/day) — least frequent among survivors and most common in the group of sudden deaths—became risk factors for sudden death ($p<0.05$). In female cases of sudden death, hypertension, heavy smoking, lack of physical activity during leisure time, and obesity were more common, but the numbers were small and the differences did not reach statistically significant levels.

80-0792. Shurtleff, D. **The Framingham Study. An Epidemiological Investigation of Cardiovascular Disease. Section 30.**

Some Characteristics Related to the Incidence of Cardiovascular Disease and Death: Framingham Study, 18-Year Follow-up. In: Kannel, W. B.; Gordon, T. (Editors). The Framingham Study. An Epidemiological Investigation of Cardiovascular Disease, DHEW Publication No. (NIH) 74-599, February 1974, 316 pp.

This section of the Framingham Study is composed of tables of data for 14 events including myocardial infarction, intermittent claudication, coronary insufficiency, coronary heart disease, cardiovascular disease, angina pectoris, atherothrombotic brain infarction, and death. For each event there are 18 characteristics considered, e.g., blood pressure, serum cholesterol, pulse rate, cigarettes smoked, hypertension, heart enlargement, and diabetes mellitus. Statistical information includes smoothed, crude, and age-adjusted rates for men and women, and regression coefficients of the logistic function.

80–0793. Steinmetz, J.; Panek, E.; Siest, G. **Personal and Familial Factors in Cholesterolemia: Criteria for Selection of a Reference Population.** *Clinical Chemistry* 26(2): 219-226, 1980.

Factors affecting cholesterolemia in a healthy population of 23,300 subjects, aged 20-59 years, were studied. Multiple-contingency tables were established to quantify the information obtained by means of a factorial correspondence analysis. Cholesterolemia paralleled alcohol and tobacco consumption and arterial blood pressure. Use of oral contraceptives did not influence cholesterol values. An increase in cholesterol concentration of 0.21 mmol/l, to 5.81±0.98 mmol/l, was found for smokers (>10 cigarettes/day) who were not overweight (weight<115 percent of ideal weight) and who consumed small amounts of alcohol (<44 g/day). Male smokers of >20 cigarettes/day had high cholesterol values whereas consumption of >20 cigarettes/day was accompanied by relatively low cholesterol values in the 40- to 59-year-old female smokers. (Auth. Abs. Mod.)

80–0794. Sutorius, D. J.; Majeski, J. A.; Miller, S. F. **Vascular Complications as a Result of Intra-Aortic Balloon Pumping.** *American Surgeon* 45(8): 512-516, August 1979.

Intra-aortic balloon conterpulsation (IABP) was used to assist 109 patients with extensive myocardial infarcts, unstable angina, cardiogenic shock, and unstable cardiodynamic states after cardiopulmonary bypass over a 6-year period. Severe vascular occlusion occurred in three patients (3 percent) which required an above the knee amputation. Each patient had a long history of smoking (≥1 pack of cigarettes/day for 25 to 30 years). Obesity, atherosclerotic disease of the femoral vessels, and extensive coronary artery disease were additional contributing factors. Two of the three patients survived, but both survivors had extensive postoperative myocardial infarctions. A low flow cardiac state and the presence of atherosclerotic changes in the legs must be precipitating factors for the vascular complications. Several possible methods to minimize complications of this nature include (1) angiographic examination of the lower aorta and femoral arteries at the time of cardiac catheterization, (2) frequent monitoring with ultrasound equipment, and (3) use of anticoagulation during and after the period of conterpulsation. (Auth. Abs. Mod.)

80–0795. Ueda, K.; Howard, G.; Toole, J. F. **Transient Ischemic Attacks (TIAs) and Cerebral Infarction (CI): A Comparison of Predisposing Factors.** *Journal of Chronic Diseases* 33(1): 13-19, 1980.

Fifteen clinical characteristics (including family history of stroke, hypertension, diabetes mellitus, and smoking) thought to be contributors to cerebral infarction (CI) were analyzed in 42 patients with transient ischemic attacks (TIAs) due to cervical arterial lesions and 52 with cerebral infarction who had neither previous TIAs nor detectable lesions of neck arteries. By using linear discriminate function analysis, the characteristics which differ between these groups were determined. The importance of such characteristics is to establish which patients will develop an infarction without TIAs as warning signals. In the case of smoking, 36 percent of the TIA patients and 27 percent of the CI patients were smokers. Smoking did not discriminate significantly between the two groups of patients. Blood pressure, atrial fibrillation, myocardial infarction, diabetes mellitus, serum lipid level, and family history of stroke seemed to discriminate between patients with infarction without history of TIAs and those who had TIAs only. (Auth. Abs. Mod.)

80–0796. Wennmalm, A. **Cigarette Smoking, Prostaglandins and Reactive Hyperemia.** *Prostaglandins and Medicine* 3(6): 321-326, December 1979.

Forearm blood flow was recorded at rest, and following forearm ischemia before and after cigarette smoking in healthy male and female volunteers. The experiments were also repeated after pretreatment of the subjects with indomethacin, a prostaglandin (PG) synthesis inhibitor. Before pretreatment with indomethacin, ischemia induced a reactive hyperemia amounting to 20±4 ml/100 ml tissue. This hyperemia was significantly ($p<0.01$) reduced by cigarette smoking, to 12±3 ml/100 ml tissue. After indomethacin, which in itself lowered the reactive hyperemia to 8±2 ml/100 ml tissue, smoking did not elicit any effect. It is suggested that smoking counteracts reactive hyperemia in tissues by interfering with the same physiological mechanism as indomethacin, i.e., with the vascular formation of PG. The possible significance of this observation in relation to cigarette smoking and ischemic heart disease is stressed. (Auth. Abs. Mod.)

See also, **80–0824**

PREGNANCY AND INFANT HEALTH

80-0797. Erb, L.; Andresen, B. D. **The Fetal Alcohol Syndrome (FAS): A Review of the Impact of Chronic Maternal Alcoholism on the Developing Fetus.** *Clinical Pediatrics* 17(8): 644, 646-649, August 1978.

A review of the impact of maternal alcoholism on the developing fetus, referred to diagnostically as the Fetal Alcohol Syndrome (FAS), is presented. The FAS is characterized by prenatal and postnatal growth deficiency, microcephaly, and developmental delay or mental deficiency; its most critical manifestation is the degree to which the child's intelligence is impaired. Several alternate or contributory causes of the FAS have been suggested, e.g., maternal malnutrition, lead poisoning, and genetic causes, as well as smoking and drug abuse.

80-0798. Miller, H. C.; Hassanein, K.; Hensleigh, P. A. **Maternal Factors in the Incidences of Low Birthweight Infants Among Black and White Mothers.** *Pediatric Research* 12(10): 1016-1019, October 1978.

Black mothers (1,077) and white mothers (2,333) were placed in one of three groups, depending on whether they had medical complications of pregnancy, one or more of seven behavioral conditions of pregnancy, or had no medical complications and no behavioral conditions. The seven behavioral conditions included (1) cigarette smoking (any cigarettes in any part of pregnancy), (2) low maternal weight gains in pregnancy, (3) being under 17 years of age at delivery or (4) over 35 years of age at delivery, (5) absence of any prenatal care, (6) being underweight for height at conception, and (7) use of addicting drugs or large amounts of alcohol during pregnancy. The seven behavioral conditions were not all of equal frequency and importance. Cigarette smoking had the highest incidence. Approximately 40 percent of all the mothers smoked, with whites smoking a little more than blacks. The incidences of low birthweight (LBW) infants (<2500 g) were not significantly higher in the lowest than in the highest of the socioeconomic groups of either black or white mothers when mothers were matched either for medical complications, for behavioral conditions, or for neither. The higher incidences of LBW infants in the lowest socioeconomic groups of black and of white mothers were most probably related to the higher incidences of mothers with behavioral conditions and the lower incidence of mothers who had neither medical complications nor behavioral conditions in the lowest socioeconomic groups. The incidences of LBW infants were low (1-3 percent) in both whites and blacks irrespective of socioeconomic status in the absence of medical complications and behavioral conditions of pregnancy. Socioeconomic status apparently had no significant effect on the incidence of medical complications of pregnancy, but was associated with an increasing incidence of mothers with behavioral conditions as socioeconomic circumstances worsened. (Auth. Abs. Mod.)

80-0799. Mitev, P.; Peychev, P.; Atanasova, D.; Atanasova, Zh.; Peeva, I.; Nikiforov, N. **Kum Vuprosa za Vliyanieto na Kisloroda i Tsigareniya Dim Vurkhu Roda i Novorodenoto. [The Effect of Oxygen and Cigarette Smoke on Fetus and Newborn Infant.]** *Akusherstvo i Ginekologiya* 17(2): 111-113, 1978, Bulgarian.

One study of pregnant Wistar rats and two studies of pregnant women are described. In the rat experiments, four groups were exposed to either oxygen in concentrations of 216 or 36 ml/l air, or to smoke from two cigarettes or one-half cigarette. Each group received oxygen or cigarette smoke two or four times per day, for 10 min. One-half of each subgroup was exposed for 19 days following conception and then killed. The other half was exposed during the entire gestational period. Most of the embryos were found dead or resorbed in the rats exposed to cigarette smoke for 19 days. Control group embryos were only occasionally resorbed. The 6-day-old newborn rats which were exposed to cigarette smoke throughout gestation weighed an average of 6.8 to 11.8 g as compared with the control group newborns who weighed 15.3 g. In one study of women, clinical examinations of 2,122 housewives and tobacco industry employees revealed that the menstrual cycle was often retarded in smokers (3.1 percent) as compared to nonsmokers. Also, spontaneous abortions, premature delivery, and gynecologic and general disorders were more frequent in smokers. It was concluded that cigarette smoke unfavorably affects fetal embryonic development. In the second human study, an experimental transcutaneous treatment with oxygen of 17 pregnant women was successful in preventing spontaneous abortions.

80-0800. Paganelli, R.; Ramadas, D.; Layward, L.; Harvey, B.A.M.; Soothill, J. F. **Maternal Smoking and Cord Blood Immunity Function.** *Clinical and Experimental Immunology* 36(2): 256-259, May 1979.

As it seems possible that increased vulnerability to chest infection of children of smoking parents is due to intrauterine secondary immunodeficiency, measurements were made of hemoglobin, leukocyte counts, immunoglobulins (IgG, IgG_1, IgM), transferrin, lymphocyte phytohemagglutinin (PHA) response, and neutrophil mobility in the cord blood of six mothers who smoked during pregnancy and of eight mothers who did not. There was no significant difference in levels of hemoglobin, immunoglobulin and transferrin concentrations, neutrophil and lymphocyte counts, the E-rosette percentage and count, and leukocyte mobility, with and without bacterial endotoxin stimulus, between the smoking and nonsmoking groups. The cord blood lymphocytes from the smoking mothers showed a significantly higher thymidine uptake in PHA-stimulated cultures than did those from nonsmoking mothers when the test was performed on whole blood cells in either autologous serum ($p<0.05$) or serum from healthy adults ($p<0.05$), but there was no difference between the groups when separated lymphocytes were studied. There was also a significantly greater thymidine uptake by the unstimulated separated cells in smokers cultured in adult serum than in nonsmokers ($p<0.01$); a similar trend with autologous serum was not significant.

80-0801. Vangen, O. **Morbiditet hos Barn og Modre ved Aktiv og Passiv Roking. [The Effect of Passive and Active Smoking on the Morbidity of Children and Mothers.]** *Tobakken og Vi* (3): 10-15, 1979, Norwegian.

The effect of passive smoking on the development and the morbidity of infants and children under 2 was studied. Premature births were four times more common among smoking ($n=201$) than nonsmoking ($n=98$) mothers (19.4 and 4.1 percent, respectively. Diabetic and pre-eclamptic individuals were not included in the study. At week 40 of pregnancy, the average weight of the infant was 425 g more for nonsmoking mothers than for smoking mothers. Colds occurred in 21.3 percent of the children when both of their parents smoked, 60 percent more frequently than for children of nonsmoking parents. Serious respiratory infections occurred in 25.3 percent of the children with smoking parents, 70 percent more frequently than in the group of children from nonsmoking families. The morbidity among mothers, related to smoking habits, was studied for the 2-year post-partum period after the last birth. Even though adults had a higher tolerance for tobacco smoke, the difference between smoking and nonsmoking groups was large. Allergy-related illnesses occurred in 12 percent of the mothers from homes where both spouses were smokers, as compared to 4 percent for nonsmokers. The corresponding figures for uncomplicated respiratory infections were 26.7 and 13.0 percent, respectively; and for serious respiratory infections, 12.0 and 2 percent, respectively.

See also, **80-0720, 80-0733, 80-0818**

OTHER DISEASES AND CONDITIONS

80-0802. Boulton, T.J.C.; Rowley, M. P. **Nutritional Studies During Early Childhood. III. Incidental Observations of Temperament, Habits, and Experiences of Ill-Health.** *Australian Paediatric Journal* 15(2): 87-90, June 1979.

The mothers of 391 normal 3-month-old and 266 1-year-old babies were asked standard questions about their child's motor activity, sleeping, and thumb-sucking habits, and whether the child had had colic, regurgitant vomiting, or an episode of ill health during infancy. By 1 year 36.5 percent of the mothers considered their babies to be very active, compared to only 4 percent considering them placid. At 3 months of age 20 percent of babies were waking at least once a night, and significant ethnic differences were present, with babies of Italian-born parents waking more frequently. Colic was reported in 40 percent of babies, occurring frequently in 24.5 percent. It occurred slightly more frequently in babies of mothers with higher educational attainment, and in first-born. In 59 percent of the babies there were no episodes of ill-health, or only one or more episodes of upper respiratory infection. Prevalence figures are reported for wheezing episodes, bronchitis, and vomiting with diarrhea. The number of cigarettes smoked each day was inversely related to socioeconomic factors such as the mother's educational level and the type of housing afforded by the family ($p<0.002$), and there was a significant increase ($p<0.05$) in the frequency of wheezing episodes in those babies whose mothers were heavy smokers. The percentage frequency of light (0-1 cigarettes/day) smokers was 10 percent higher in mothers than in fathers (47 vs. 37 percent, respectively); however, for heavy (>20 cigarettes/day) smokers, the percentage frequency was 10 percent in fathers and 3 percent in mothers. (Auth. Abs. Mod.)

80-0803. Bressler, R.; Durand, J. L. **Oral Contraceptive Risks: A Realistic Appraisal.** *Drug Therapy* 9: 81-85, 87, 91-94, October 1979.

Much has been learned about the safety of oral contraceptives after 20 years of widespread use. The major concerns about these agents have been focused mainly on their role in enhancing the risk of cancer, thromboembolic disease, stroke, myocardial infarction, and hypertension. Although oral contraceptives appear to contribute to the development of cardiovascular disease and cancer, other risk factors, such as cigarette smoking, age, and family history, must be taken into account. It has been shown that smoking more than one pack of cigarettes per day does not potentiate the effect of oral contraceptive use on the development of thrombotic stroke. However, a marked enhancement in the relative risk of hemorrhagic stroke does occur in patients who smoke heavily and who also use oral contraceptives. There is no doubt, however, that oral contraceptives should not be used in patients with a history of cancer or cardiovascular disease, undiagnosed genital bleeding, or known or suspected pregnancy. Caution is also advised in prescribing them to women over 40 years of age or to women with a history of migraine, hypertension, epilepsy, leiomyomas of the uterus, or idiopathic jaundice in pregnancy. (Auth. Abs. Mod.)

80-0804. Christiansen, J. S. **Cigarette Smoking and Prevalence of Microangiopathy in Juvenile-Onset Insulin-Dependent Diabetes Mellitus.** *Diabetes Care* 1(3): 146-149, May-June 1978.

In order to evaluate a possible relation between cigarette smoking and prevalence of diabetic microangiopathy, a series of 180 consecutive patients suffering from insulin-dependent juvenile-onset diabetes mellitus with different durations of disease (60 patients each with diabetes for 10 to 19 years, 20 to 29 years, and 30 to 39 years, respectively) were examined for clinical signs of retinopathy, nephropathy, and peripheral neuropathy. The results were compared with the patients' previous and present smoking habits. Cigarette smoking was defined as daily smoking of at least 10 cigarettes for 1 year or more. Smoking habits of the total diabetic sample were not significantly different from those of a nondiabetic control sample. However, a decline in the number of cigarette smokers and a rising number of ex-smokers were noted with increasing duration of diabetes. In comparing smokers and nonsmokers, no difference was found in the prevalence of peripheral neuropathy, background retinopathy, and proliferative retinopathy. However, the prevalence of nephropathy (persistent proteinuria) was significantly higher ($p<0.05$) among those patients who were or had been cigarette smokers. Thus, cigarette smoking might be considered a risk factor for the development of diabetic nephropathy. (Auth. Abs.)

80-0805. Hollo, I.; Gergely, I.; Boross, M. **Influence of Heavy Smoking Upon the Bone Mineral Content of the Radius of the Aged and Effect of Tobacco Smoke on the Sensitivity to Calcitonin of Rats.** *Aktuelle Gerontologie* 9(8): 365-368, August 1979.

Using Norland-Cameron Bone Mineral Analyzer BMA 178 Type, it has been found that bone mineral content of the radius is considerably less in male heavy smokers aged 61-75 years and 76-90 years than in nonsmokers of similar ages. Differences within the male age groups are considerable and statistically highly significant: $p<0.01$ in the group of 61- to 75-year-old men, and $p<0.02$ in the group of 76- to 90-year-old men. The difference among women is considerably less ($p<0.05$). Under the influence of equal doses of calcitonin, the decrease in the calcium level of rats treated with tobacco smoke was less in each series than that of nontreated rats. Consequently, tobacco smoke reduced the sensitivity to calcitonin, resulting in a certain resistance to calcitonin. Differences found are statistically highly significant in each series ($p<0.01$). In contrast to calcitonin resistances caused by castration, adrenalectomy, or aminoglutethimide treatment, tobacco smoke-induced decreased sensitivity to calcitonin was not alterable by pretreatment with dehydroepiandrosteron. No calcitonin resistance resulted from cadmium (Cd) tested in two different doses; the following serum calcium concentrations were found under calcitonin action: control ($n=11$): 7.5 ± 0.47 mg/100 ml, treated with 0.005 mg Cd ($n=10$): 7.8 ± 0.56 mg/100 ml, and treated with 0.05 mg Cd ($n=10$): 7.6 ± 0.42 mg/100 ml. It is possible that smoking promotes the decrease in mineral content of bones, i.e., the aggravation of osteoporosis, by the means of calcitonin resistance. (Auth. Abs. Mod.)

80-0806. Oddsson, E.; Binder, V.; Thorgeirsson, T.; Jonasson, T. A.; Gunnlaugsson, O.; Wulff, M.; Jonasson, K.; Wulff, H. R.; Bjarnason, O.; Riis, P. **A Prospective Comparative Study of Clinical and Pathological Characteristics in Icelandic and Danish Patients With Gastric Ulcer, Duodenal Ulcer, and X-Ray Negative Dyspepsia. II. Histological Results.** *Scandinavian Journal of Gastroenterology* 13(4): 489-495, 1978.

The occurrence of gastritis in antral and body mucosa is compared in an Icelandic and a Danish group of patients with gastric ulcer, duodenal ulcer, and X-ray negative dyspepsia. In all, 93 Icelandic and 88 Danish patients were examined, of whom 120 were smokers. All signs of antral gastritis were more frequent in Icelandic than in Danish patients, but only the incidence of superficial inflammation and decreased mucus content in surface and crypt epithelium differed significantly. In body mucosa pseudopyloric metaplasia was more frequent in Iceland and occurred equally frequently in all three diseases. A statistically significant correlation ($p<0.05$) was found between macroscopic gastritis and occurrence of antral superficial inflammation and between smoking and superficial inflammation and decreased mucus content in the pyloric biopsy specimen. The presence of histological gastritis was not correlated to the intake of alcohol and salicylic acid, nor to the presence of pain at the time of investigation. (Auth. Abs.)

80-0807. Soldatos, C. R.; Kales, J. D.; Scharf, M. B.; Bixler, E. O.; Kales, A. **Cigarette Smoking Associated With Sleep Difficulty.** *Science* 207(4430): 551-553, February 1, 1980.

A group of 50 smokers (31 males, 19 females) experienced greater sleep difficulty than a group of 50 nonsmokers matched by age and sex. The smokers were awake for a significantly longer time than the nonsmokers (92.7 vs. 73.9 min, $p<0.05$), primarily because they had greater difficulty in falling asleep (43.7 vs. 29.8 min, $p<0.05$). The two groups did not differ in personality patterns or drug consumption. Sleep patterns significantly improved in a group of eight chronic smokers (1.5-3.0 packs/day for 2 years) when they abstained from cigarette smoking. Total time spent awake decreased by 45 percent on the first three nights of abstinence from 75.9 to 42.0 min ($p<0.01$); sleep latency decreased notably, but there was little change in time spent awake after sleep onset. On nights 8 and 9 of withdrawal, the total time the subjects were awake continued to be less than during the baseline nights (51.0 vs. 75.9 min; a 33 percent decrease, $p<0.05$). These findings are consistent with reports on the stimulant effects of nicotine.

80-0808. Sweet, J. B.; Butler, D. P. **The Relationship of Smoking to Localized Osteitis.** *Journal of Oral Surgery* 37: 732-735, October 1979.

The relationship between smoking and localized osteitis was studied in 200 patients who had 400 mandibular third molars removed. Among these patients, 23.9 percent of the females and 22 percent of the males were identified as smokers. Localized osteitis occurred at 11 of 92 (12 percent) surgical sites of patients identified preoperatively as smokers, but at only 8 of 308 (2.6 percent) extraction sites of nonsmokers. Patients identified as heavy smokers (≥0.5 packs/day) had a significantly greater postoperative incidence of localized osteitis. Localized osteitis occurred at 11 of 76 (14.5 percent) extraction sites of patients identified as postoperative smokers, whereas in patients who did not smoke postoperatively, there was an incidence of localized osteitis of 8 of 324 (2.5 percent). Localized osteitis occurred in 10 of 38 patients (26.3 percent) who smoked postoperatively. However, there was no occurrence in seven patients identified as smokers preoperatively who did not smoke postoperatively. Of the 10 patients in whom localized osteitis developed, 40 percent smoked on the day of surgery and all 10 smoked the day after surgery. These results indicate that there is a significant difference in the incidence of postoperative localized osteitis at extraction sites of mandibular third molars between smokers and nonsmokers. Smoking after removal of mandibular third molars will cause a definite increase in the incidence of localized osteitis. A patient should, therefore, be cautioned against smoking cigarettes for an indetermined period of time after oral surgery.

80-0809. Weber, A.; Fischer, T.; Grandjean, E. **Passivrauchen: Reizwirkungen des Gesamtrauches und der Gasphase. [Passive Smoking: Irritating Effects of the Total Smoke and the Gas Phase.]** *Sozial-und Praventivmedizin* 24(4): 280-281, August 1979, German.

Annoyance and irritation of eyes, nose, and throat were measured during exposure to total cigarette sidestream smoke (particulate and gas phases) or to gas phase smoke alone. In both cases annoyance increased sharply within the first 10 min of exposure, then continued to increase very slightly during further exposure to total sidestream smoke or remained at the same level during exposure to the gas phase. Both eye irritation and blinking rate increased continuously to a maximum over 50 min of exposure to total sidestream smoke. It is concluded that annoyance was caused primarily by the gas phase of cigarette smoke, while irritating effects were due mainly to the particulate phase of smoke.

See also, **80-0780**

BEHAVIORAL AND EDUCATIONAL RESEARCH

80-0810. Beck, K. H., III. **The Effects of Positive and Negative Affect-Arousing Communications Upon Attitudes, Belief Acceptance, Behavioral Intention, and Actual Behavior.** Doctoral Dissertation, Syracuse University, University Microfilms 78-11,632, 1978, 341 pp.

The effectiveness of emotionally arousing persuasive communication (e.g., fear arousal) was examined in this dissertation. Emotionally based physiological responses to slides depicting some of the health consequences of smoking were measured. The study considered whether smokers would show more attitude change in the direction advocated in response to increased positive affect-arousing rather than increased negative affect-arousing persuasive communications, and whether nonsmokers would show less overall attitude change than smokers in response to both types of affect-arousal. In addition to the subjects' attitudes toward smoking, measures of belief acceptance, behavioral intention, and actual behavior were also taken. A total of 240 subjects (120 smokers and 120 nonsmokers) participated in the experiment. Analysis of the effect of the arousal manipulation upon comprehension, attitude, belief acceptance, and behavioral intention showed that smokers comprehended more of the message than nonsmokers and that nonsmokers held more negative attitudes toward smoking than did smokers. As the type of arousal became more negative, the attitude toward smoking became more negative.

80-0811. Botvin, G. J.; Eng, A.; Williams, C. L. **Preventing the Onset of Cigarette Smoking Through Life Skills Training.** *Preventive Medicine* 9(1): 135-143, January 1980.

The effectiveness of a 10-session social psychological approach to the prevention of cigarette smoking was tested on 8th, 9th, and 10th graders (n=281) in suburban New York. The program was designed to address both the social and psychological factors promoting the onset of smoking by attempting to (a) increase students' ability to cope with direct pressures to smoke, (b) decrease their susceptibility to indirect pro-smoking social influences, and (c) improve their ability to cope with anxiety. The program was conducted by allied health professionals and utilized group discussion, modeling, and behavior rehearsal. Results indicate significant differences between the experimental and control groups in terms of the proportion of new "experimental smokers" ($p<0.01$); the onset rate for the control group was 16 percent, compared with only 4 percent for the experimental group. Furthermore, there were significantly greater pre-test—post-test changes for the experimental group on several of the knowledge and psychological measures, suggesting that the decrease in the onset of smoking behavior among the experimental subjects did occur for the hypothesized reasons. (Auth. Abs. Mod.)

80–0812. Carney, R. E. (Editor). **Risk-Taking Behavior. Concepts, Methods, and Applications to Smoking and Drug Abuse.** Springfield, Illinois, Charles C. Thomas, 1971, 219 pp.

Basic concepts and theories of risk-taking and the role of variables such as personality and social factors, as well as types of and attitudes toward risk-taking behavior are discussed. In addition, the management of risk-taking behaviors, such as smoking and drug abuse, is examined. A number of procedures (e.g., punishment consisting of aversive stimulation or removal of positive reinforcers) utilizing essentially counter-conditioning may be used to modify smoking behavior. Restriction of the range of stimuli controlling smoking operants, reduced availability or total elimination of stimuli in the smoker's environment, and satiation may also be used. The smoker can perhaps be trained by others to use some of these procedures (especially coverant control), but because "others" often present reinforcing and discriminative stimuli maintaining and controlling smoking, they can best assist the smoker to make significant changes in his environment demanded by the procedures. (Auth. Abs. Mod.)

80–0813. Center for Disease Control. **Smoking Behavior and Attitudes of Physicians, Dentists, Nurses, and Pharmacists, 1975.** *Morbidity and Mortality Weekly Report* 26(23): 185, June 10, 1977.

The proportion of physicians, dentists, and pharmacists who smoke cigarettes has decreased markedly, according to a 1975 national survey of the smoking behavior and attitudes of professional health personnel. Results of the survey, which were compared with earlier surveys conducted between 1967 and 1969, also indicate that smoking among nurses is at about the same level as it was in 1969 (39 percent in 1975 vs. 37 percent in 1969). By contrast, the proportion of smokers dropped among (1) physicians from 30 percent in 1967 to 21 percent in 1975, (2) dentists from 34 percent in 1967 to 23 percent in 1975, and (3) pharmacists from 35 percent in 1968 to 28 percent in 1975. Although a greater proportion of nurses are smoking compared to the other professional groups surveyed, nurses smoke fewer cigarettes per day than physicians, dentists, and pharmacists who smoke. People in the four professions generally and increasingly see cigarette smoking as a cause of heart disease and oral cancer, and as either a major or contributing cause of lung cancer, chronic bronchitis, and pulmonary emphysema. Most of the professionals reported that they believe it is their role to set a good example by not smoking, that it is their responsibility to convince people to stop smoking, and that they should be more active in speaking to lay groups about cigarette smoking. Health professionals were also found to be increasingly optimistic about their ability to affect change in their patients' cigarette smoking behavior.

80–0814. Coe, R. M.; Cohen, J. D. **Cigarette Smoking Among Medical Students.** *American Journal of Public Health* 70(2): 169-171, February 1980.

A survey of first and second year medical students at Saint Louis University revealed that only 6 percent were current cigarette smokers, and even fewer (both nonsmokers and current smokers) expected to be smoking in the near future. The students feel strongly that physicians have a major responsibility to encourage nonsmoking among patients and that smoking in public places should be severely restricted. There were no statistically significant differences in attitudes between the smokers and nonsmokers. The largest difference was in attitude toward regulation of smoking in public places. Current smokers were less favorably inclined toward increased regulation. Current smokers were less emphatic than nonsmokers about the physicians' responsibility. Nonsmokers also scored better on knowledge of health hazards of smoking than did current smokers. Students who presently smoke reported having more friends who also smoke than did nonsmokers. There were no differences among the groups with respect to parents who currently smoke.

80–0815. Food and Drug Administration. **A Study of Health Practices and Opinions. Final Report.** U.S. Department of Commerce, National Technical Information Service Publication No. PB-210 978, June 1972, 456 pp.

This national survey investigated beliefs and/or practices concerning areas such as (1) use of vitamins or other nutritional supplements, (2) weight reduction, (3) self-diagnosis, (4) self-medication, (5) health practitioners used, and (6) "aids" to quitting smoking. Among the total sample 6 percent had bought something without a prescription that was advertised "to help you quit smoking or to cut down smoking." For this group, 80 percent said it was something other than "ordinary candy or gum." Purchase of these products was more likely among higher income people. Only 1 percent of those with incomes under 3,000 dollars reported it, as compared to 3 percent of those with incomes from 3,000 to 6,999 dollars, 6 percent of those with incomes of 7,000 to 11,999 dollars, and 6 percent of those with incomes of 12,000 dollars or more. When shown a list of possible influences upon them, 53 percent of the users of a special product said TV or radio commercials affected their purchase, 24 percent cited newspaper or magazine advertisements, and 15 percent cited store displays. "Friends" were cited by 12 percent, magazine articles by 7 percent, "relatives" by 5 percent, and medical columns by 4 percent. Those persons who bought these products tended to have higher education and income than the total population, and more of them were male than in the total population.

80-0816. Frederiksen, L. W.; Simon, S. J. **Modification of Smoking Topography: A Preliminary Analysis.** *Behavior Therapy* 9(5): 946-949, 1978.

Instructions were used to modify topographical characteristics of smoking behavior in a single-case multiple-baseline design experiment. The subject was a 24-year-old male hospital employee who had smoked approximately one pack/day for 7 years. Topographical components of puff frequency and puff duration were modified. Generalization to natural environment situations was unobtrusively assessed. During baseline the subject averaged 9.0 puffs/cigarette during the videotaped sessions and 8.1 puffs during generalization probes. After instructions, puff frequency was reduced to criterion (i.e., six puffs or less) over the next 17 sessions and during all but three generalization probes. Mean puff duration decreased from a terminal baseline performance of 1.50 and 1.40 sec (lab sessions and generalization probes, respectively) to 0.78 and 0.87 sec following instructions. Carbon monoxide (CO) levels, subjective enjoyment, and daily smoking rate also were observed. Self-monitoring data revealed a gradual decreasing trend in smoking rate during topography changes. The baseline average of 26.2 cigarettes/day decreased to 18.6 cigarettes during the last week of training. Mean pre-CO levels during baseline were 28.7 ppm with a mean CO boost of 4.2 ppm. These were reduced to mean pre-CO levels of 24.0 ppm during puff-frequency training and 14.4 ppm during puff-duration training. Carbon monoxide boosts were reduced to below 1 ppm during both training phases. Mean subjective enjoyment ratings on a 0 to 10 scale (10 = maximal enjoyment) were 4.5 during baseline, 5.4 during puff-frequency training, 1.1 during puff-duration training, and 7.0 during followup. Results showed that instructions modified topographical components of smoking behavior in clinically significant directions and that these changes generalized. (Auth. Abs. Mod.)

80-0817. Grosz, H. J. **Nicotine Addiction: Treatment With Medical Hypnosis. Part 3.** *Journal of the Indiana State Medical Association* 72(1): 43-45, January 1979.

This report deals with those factors that seem to have influenced the outcome of a previously reported single hypnosis treatment session of cigarette smokers. First, the most important prognostic factor in the treatment of smokers was the time elapsed since the cessation of treatment. For both men and women, the success rate declined with time, from 59.1 and 57.6 percent 1 month after treatment, to 31.1 and 33.5 percent, respectively, after 6 months ($p<0.0001$). Second, the amount smoked at the time treatment began was clearly of importance in determining outcome. Although the mean difference in the number of cigarettes smoked between those who quit and those who failed to do so was less than half a pack of cigarettes a day, it was statistically significant ($p<0.001$). Third, personality and attitude were evaluated on the basis of the patients' response as to the intensity of their smoking enjoyment, their motivation to quit smoking, their confidence in their unaided ability to quit smoking, their confidence in hypnosis as an aid to quit, and the intensity of their general feeling of nervousness. Only in the case of nervousness was there a close to statistically significant difference between those who maintained abstinence from smoking for 3 months and those who did not ($p<0.062$).

80-0818. Hackett, D. R. **Smoking Habits During Pregnancy.** *Irish Medical Journal* 72(11): 483-486, November 30, 1979.

All obstetrical inpatients ($n=90$) in the public wards of a hospital were interviewed about their smoking habits during pregnancy using a standard questionnaire. Among the patients, 55 admitted to smoking before the start of pregnancy, and 49 admitted to smoking after the fourth month. There were 33 patients who stated that they had never smoked. Almost all patients were aware that smoking might be harmful to the fetus, and many cut down their habit during the first half of pregnancy. Nevertheless, a substantial number of patients (14) increased their smoking habit during the second half of pregnancy, at a time when the effects of smoking have been shown to be of greatest risk to the fetus. (Auth. Abs.)

80-0819. Houpt, J. L.; Orleans, C. S.; George, L. K.; Brodie, H.K.H. **Cigarette Smoking.** In: Houpt, J. L.; Orleans, C. S.; George, L. K.; Brodie, H.K.H. *The Importance of Mental Health Services to General Health Care.* Cambridge, Massachusetts, Ballinger Publishing Company, 1979, pp. 55-99.

In this report requested by the Institute of Medicine, National Academy of Sciences, for submission to the President's Commission on Mental Health, a behavioral approach to the smoking and health problem was presented in one chapter. The following analyses were presented: sociodemographic characteristics of smokers, organizational and staffing patterns of existing health care delivery systems, and requirements for effective treatments. Recommendations for a cost-effective health care delivery system were made. It was estimated that smoking was responsible for about 325,000 premature deaths each year. The effects of passive smoking also received increasing attention. Surveys showed that (1) in 1970 and 1975 there were decreasing proportions of male smokers, except in the over 65 group; (2) there was a 23 percent increase in teenage girls smoking between 1969 and 1975; and (3) smoking rates among white adults were 38 percent compared with 55 percent among blacks in 1974. Socioeconomic analyses found that smoking rates were lowest for persons who never went to high school (37 percent adult men, 18 percent adult women); that among men increasing income was associated with decreased smoking but among women with increased smoking; and that higher smoking rates were observed for large cities as compared with small cities and rural areas. The use and effectiveness of smoking cessation strategies were emphasized. Public information campaigns, mass-media educational campaigns, and interpersonal communications were judged important but insufficient methods for alteration of smoking behavior. Socioeconomic status strongly correlated with attitudes toward utilization of voluntary mental health care services, especially in the lower socioeconomic stratum. A four-level intervention model was outlined as follows: Level I (minimal intervention), education; Level II (intermediate intervention), audiovisual materials and instructions; Level III (maximal intervention), instruction in behavioral self-control techniques and a structured support system with therapists; and Level IV (intensive programing), multicomponent intervention.

80–0820. Howe, M.; Summerfield, A. B. **Orality and Smoking.** *British Journal of Medical Psychology* 52(1): 85-90, 1979.

This study was designed to investigate the relation of orality and smoking, smoking motivation, and anticipatory pleasure in smoking. The subjects were 46 males and 51 females (54 smokers and 43 nonsmokers) who completed questionnaires designed to elicit information about their motivation and behavior. Results indicated that nonsmokers compared to smokers did not differ significantly on orality. Difficulty in stopping smoking was positively related to cigarette-sucking ($p<0.001$), adult pencil-sucking ($p<0.001$), and nail-biting ($p<0.05$). Smoking for psychological as opposed to social reasons was positively related to cigarette-sucking ($p<0.01$) and adult pencil-sucking ($p<0.02$). Anticipation of pleasure in smoking before becoming a smoker was negatively related to nutriment orality ($p<0.001$), positively related to smoking because of a craving ($p<0.01$), and, for females only, positively to cigarette-sucking ($p<0.025$). The results are discussed in relation to the role of oral ingestion and the reaction of the central nervous system to the drug. (Auth. Abs. Mod.)

80–0821. Hutchinson, R. R.; Pierce, G. E.; Emley, G. S.; Proni, T. J.; Sauer, R. A. **The Laboratory Measurement of Human Anger.** *Biobehavioral Reviews* 1(4): 241-259, 1977.

A method was developed for the unobtrusive measurement of the frequency, duration, and intensity of nonfunctional jaw contractions in humans. This method was used to study the environmental causes of jaw clenching (bruxism). Subjects included normal males and females, ranging in age from 17 to 59 years. The six experiments included (1) measurement of jaw contractions in humans, (2) the effects of intense noise upon jaw contractions, (3) jaw clenching in smokers following cessation of cigarette smoking, (4) effects of cessation of monetary delivery upon jaw contractions, (5) effects of several fixed-ratio schedules of reinforcement and of extinction upon jaw contractions, and (6) the effects of changes in response-independent pay upon jaw contractions. Jaw contractions were differentially increased by the delivery of noise, the cessation of smoking, and the termination or reduction of both response-dependent and response-independent money, and of points convertible into money. Three females and five males comprised the normal smoker volunteers. They were aged 22-59 years, and were selected from a community smoking clinic. The subjects smoked from 3 to 46 years, and were consuming 10-40 cigarettes/day at the beginning of the studies. During the course of the experiment, two subjects stopped for 10 and 4 days, one stopped for 20 days and then started again, and five subjects stopped for the duration of the experiment (31-33 days). It was demonstrated that interruption of consumption of a positive primary reinforcer (smoking) resulted in jaw contractions.

80–0822. Hyland, M.; Birrell, J. **Government Health Warnings and the "Boomerang" Effect.** *Psychological Reports* 44(2): 643-647, April 1979.

The presence of a government health warning on cigarette advertisements can "boomerang," leading to an increased desire to smoke. In this study, 24 housewives saw a series of 25 cigarette advertisements; for 12 subjects the health warning was present and for 12 it was absent. Half the subjects in each group were smokers and half nonsmokers. The presence of the warning increased the desire to smoke but the increase seems to be greater for smokers than for nonsmokers. The presence of the warning decreases the perceived goodness of the advertisement but does not affect its perceived familiarity. (Auth. Abs.)

80–0823. Jones, J. W. **Adverse Emotional Reactions of Nonsmokers to Secondary Cigarette Smoke.** *Environmental Psychology and Nonverbal Behavior* 3(2): 125-127, 1978.

Exposure to secondary cigarette smoke is one of the most frequently mentioned annoyances of Americans. Two studies are reviewed which examine the impact of ambient smoke on the emotional reactions of nonsmokers. The results indicate that exposure to secondary cigarette smoke significantly increased the anxiety levels of the subjects. They were more worried, tense, high-strung, and emotionally upset than they were after exposure to clean air. Exposure to secondary smoke was also shown to result in more aggressive behavior of the subjects.

80–0824. Koslowsky, M.; Croog, S. H.; La Voie, L. **Perception of the Etiology of Illness: Causal Attributions in a Heart Patient Population.** *Perceptual and Motor Skills* 47(2): 475-485, October 1978.

This study examined perceived causes of myocardial infarction in a patient population of 345 men (between 30 and 60 years of age) previously free from significant medical problems. Investigation of their perceptions following the life-threatening illness crisis indicated that stress and tension factors were the causes most commonly cited. The most important individual perceived cause was "being under tension at work," which was expressed by nearly 65 percent of the subjects in the study. This was followed closely by "nerves" (60 percent), "worry" (59.7 percent), and "smoking" (59.1 percent). Possible social and psychological correlates are analyzed using an attribution theory framework, and their implications are discussed. (Auth. Abs. Mod.)

80-0825. Lando, H. A. **Toward a Clinically Effective Paradigm for the Maintenance of Nonsmoking.** *Behavior Therapy* 9(4): 666-668, 1978.

Three research studies are proposed toward investigation of maintenance of nonsmoking. The first study will compare a two-stage treatment program consisting of aversive conditioning and self-management and a three-stage treatment which includes booster sessions. In the second study, subjects will be assigned to treatment involving (1) three phases (preparation, aversion, maintenance), (2) two phases (preparation, aversion; preparation, maintenance; or aversion, maintenance), or (3) one phase (preparation, aversion, or maintenance). The third study will focus on specific treatment elements. The efficacy of minimal contact treatment and of a tracking program which attempts to place nonabstinent subjects on a sharply reduced schedule of smoking will also be addressed.

80-0826. Marvit, R. C.; Rigney, K. B.; Yost, F. N. **A Good Neighbor Policy: Controlled Smoking Areas and Health Department Attitudes.** *Hawaii Medical Journal* 39(1): 7-10, January 1980.

A need to regulate smoking in public areas has been mandated by public concern. To determine the most effective policy, 3,000 Health Department employees were surveyed statewide. Results overwhelmingly support some form of restriction in the Department's facilities regardless of the respondents' own smoking behavior. Of the respondents, 77 percent favored no-smoking sections in common work areas (65.6 percent of the smoker respondents). No-smoking sections or tables in dining rooms were supported by 72 percent (61.5 percent of the smokers). A total of 44 percent favored banning smoking throughout the Department buildings statewide (17.8 percent smokers); about 60 percent in the clinics (37.6 percent smokers); and 57 percent in the waiting rooms (57 percent smokers). About 10 percent stated that they favored restrictions because the State Department of Health should not condone smoking and should set an example to others. A number pointed out the issue of smokers' and nonsmokers' rights, the need to designate smoking areas rather than outright banning, and the difficulties that air conditioning with its recirculation presented in public buildings. About 25 percent of the smokers said they would attend "quit smoking" clinics, if offered at their work site. Of the 556 respondents who said they were ex-smokers, 56 percent quit for fear of health consequences, 13 percent on their physicians' advice, 16 percent because of family pressure, and only 5.6 percent because of media pressure. A policy to create controlled smoking areas in the Hawaii State Health Department system will be based on this survey. The active participation of employees to control smoking may prove the most effective means of reducing tobacco consumption. A followup survey is planned 1 year from now to determine the effectiveness of the policy and the attitude and behavior of the workers. (Auth. Abs. Mod.)

80-0827. Miller, P. M. **Interactions Among Addictive Behaviours. (Letter).** *British Journal of Addiction* 74(2): 211-212, June 1979.

In addition to being linked in terms of health hazards, smoking, overeating, and excessive alcohol consumption appear to be interrelated on a behavioral level. Few controlled studies are, however, available which analyze the relationships among these habits, although the amount of literature on alcohol-food and particularly on alcohol-cigarette interactions is increasing. Health surveys indicate that a strong positive correlation exists between heavy drinking and heavy smoking. The author suggests that substance abuses be viewed as a totality and interactions between these habits be studied systematically.

80-0828. Thomas, C. B.; Santora, P. B.; Shaffer, J. W. **Health of Physicians in Midlife in Relation to Use of Alcohol: A Prospective Study of a Cohort of Former Medical Students.** *Johns Hopkins Medical Journal* 146(1): 1-10, January 1980.

The use of alcohol over time by a cohort of physicians was surveyed. The relationship of quantity of cigarettes smoked to health status in 1978 was also considered. Two cross-sectional measures were used: (1) quantity of cigarettes smoked daily In Medical School, and (2) maximum number of cigarettes smoked daily Up to Age 30. In comparing the Healthy Group with that of the Not Healthy Group on these two scales, the amount of cigarettes smoked was significantly higher for the Not Healthy Group in both instances ($p<0.01$). In addition, when subjects in each of the four main disorder groups were compared with their matched controls on these two scales, those in the Coronary Heart Disease Group and in the Hypertension Group were significantly heavier smokers both in the In Medical School and the Up to Age 30 comparisons (Coronary Heart Disease: In Medical School, $p<0.01$; Up to Age 30, $p<0.05$); Hypertension (<0.001 for both comparisons). Two longitudinal measures of smoking were also considered: (1) total years of cigarette smoking, and (2) total years of smoking heavily (20 or more cigarettes a day).

80-0829. Thompson, M. E.; Forbes, W. F. **Do People Smoke for Nicotine? (Letter).** *British Medical Journal* 2(6135): 502, August 12, 1978.

Canadian data are used to corroborate the observation that as average nicotine delivery of U.K. cigarettes declined, the annual consumption of cigarettes greatly increased (*British*

Medical Journal 1(6117): 922, April 8, 1978). Canadian survey data also indicate that the percentage of cigarette smokers has decreased and the light smoker class has diminished. British data indicate that both male and female smokers have been smoking more cigarettes, but that relatively more female ex-smokers are drawn from the light smoking class. See also 78-1392.

80-0830. Willis, K. A.; Wehler, R.; Rush, W. A. **MacAndrew Scale Scores of Smoking and Nonsmoking Alcoholics.** *Journal of Studies on Alcohol* 40(9): 906-907, September 1979.

The MacAndrew Scale (MAC), an objective personality measure, was utilized to establish that men alcoholics who smoke would score significantly higher on the MAC than would men alcoholics who do not smoke. As the MAC had been validated as an instrument for identifying alcoholics and drug addicts, it was considered likely that it could also be used to identify smokers. A sample of 141 men alcoholics at a State hospital was selected and self-reports of their smoking habits were recorded. Each man completed the Minnesota Multiphasic Personality Inventory, and each was scored for the MAC. Of the 141 patients, 19 (13.5 percent) were nonsmokers. It was found that smoking alcoholics scored significantly higher ($p<.01$) on the MAC (mean score = $29.08 \pm .38$) than nonsmoking alcoholics (mean score = $26.79 \pm .79$).

SMOKING CESSATION METHODS

80-0831. American Health Foundation. **Study of Smoking Intervention Techniques: American Health Foundation Final Report.** American Health Foundation, New York, 1977, 588 pp.

Research and data collected on minimal intervention and maximal intervention smoking programs were presented in this final project report. Minimal interventions were explained as those which required an oral and written message by a physician during a physical examination; maximal interventions were directed at smokers who were motivated to enter into structured smoking cessation programs. Also included were a variety of supplementary studies concerned with conceptual, methodological, and service issues intrinsic to conducting effective smoking cessation programs. The minimal intervention study was designed to develop the most cost-effective method for helping large numbers of smokers to stop smoking. It was predicted that enough smokers would quit as a result to make the effort cost-effective. Eleven treatment conditions were included. Three basic procedures comprised the treatment conditions, but they were combined in a variety of ways. The procedures were baseline questionnaire, intervention and take-home material, and followup questionnaire. Subjects were selected from patients of a Health Maintenance Center who were mailed a questionnaire, which they brought with them to their annual physical. Based on the way questions were answered, patients were assigned to either a control or experimental group. After the patient's physical examination, he was told to stop smoking. In all experimental groups but one the patient was also given take-home material. Doctors were given instructions for delivering the antismoking message to their patients. The general strategy in analyzing minimal intervention data was to select which independent variables, in addition to treatment conditions and type of smoker, would be important in predicting success. Age, sex, and education were selected as variables. The dependent variables were identified as primary success criteria and secondary success criteria. Primary criteria involved a comparison of quitting frequencies and dosage reduction. Secondary criteria involved change in a number of variables from baseline to followup. A total of 1,375 subjects provided the data base for analysis. Thirteen hypotheses were analyzed. As only 157 of the 1,375 subjects actually stopped smoking, it was concluded that minimal intervention was not successful in motivating subjects to stop smoking. However, substantial numbers of subjects in all conditions actually reduced their dosage, and in many cases the reduction was significant. This dosage reduction for subjects exposed to tailored messages suggests that these messages may have greater impact than messages in standard followup procedures, one-liner intervention, or incomplete treatment. The purpose of the maximal intervention project was to develop and improve effective and efficient clinical methods for reducing smoking. Researchers tried to develop new insight into psychological and behavioral factors affecting smoking and smoking cessation. Three relatively inexpensive methods were developed: group treatments, individual counseling, and individual counseling with hypnosis. A background questionnaire was utilized, and 1-month, 5-month, and 12-month followups were conducted. There were 2,094 smokers seen over a 2-year period. Group therapy was more successful than the other programs. An attempt was made to distinguish between successful quitters and nonquitters, and a 14-hour group treatment approach was studied, which provided maximal support through the initial day of quitting. Short-term quit rates (1 week) of 80 percent were obtained with two such pilot study groups.

80-0832. Amit, Z.; Sutherland, E. A.; Weiner, A. **Stop Smoking for Good.** New York, Walker and Company, 1976, 222 pp.

Behavior modification is used in a 7-week program for stopping smoking. The various phases are (1) become aware of your smoking pattern, (2) learn to relax, (3) learn an alternate response, (4) cut out the automatic cigarette, (5) progress review, (6) satiation smoking, (7) stop for good, and (8) stopping completely. Appropriate program form examples are included.

80-0833. Caldwell, E. **How You Can Stop Smoking . . . Permanently.** North Hollywood, California, Wilshire Book Company, 1959, 96 pp.

This book is written as a guide for persons who want to quit smoking. Its main theme is that most Americans who smoke are missing the pleasures of life as well as rapidly killing themselves. The book discusses the medical case against smoking as well as the benefits of quitting, i.e., "joy of living" and increased life span. Among the suggestions for stopping the smoking habit are (1) acquire the knowledge why smoking undermines health, (2) list reasons for quitting, (3) announce intention to quit, (4) choose a partner, (5) vary daily routines, (6) fix a deadline and get rid of smoking materials, and (7) permit no exceptions to the "no smoking" rule.

80-0834. Danaher, B. G.; Lichtenstein, E. **Become an Ex-Smoker.** Englewood Cliffs, New Jersey, Prentice-Hall, Inc. 1978, 237 pp.

This book has been written as a self-help workbook for persons wanting to stop smoking. It views smoking as a learned behavior which has to be unlearned and for which new behaviors have to be substituted. A step-by-step approach to stopping smoking, including four tested methods (both aversive and nonaversive), is described. Simple techniques for diverting, redirecting, and relearning thought behavior patterns that may lead back to smoking are outlined. To deal with the mental urge to smoke and to control the physical need for tobacco, self-rewarding techniques and deep muscle relaxation skills are suggested. Ways to avoid weight gain following cessation of smoking are also described. A resource appendix presents a look at new arenas for smoking control and behavioral treatment of smoking.

80-0835. Farebrother, M.J.B.; Pearce, S. J.; Turner, P.; Appleton, D. R. **Propranolol and Giving Up Smoking.** *British Journal of Diseases of the Chest* 74(1): 95-96, 1980.

A placebo-controlled double-blind clinical trial was conducted to determine whether propranolol (40 mg three times a day) helps cigarette smokers to stop smoking; 73 subjects entered the trial but at the end of 8 weeks only six had stopped smoking, three in the propranolol and three in the placebo group. There was no evidence of any helpful effect of propranolol in subjects trying to stop smoking. (Auth. Abs.)

80-0836. Hackett, G.; Horan, J. J. **Focused Smoking: An Unequivocably Safe Alternative to Rapid Smoking.** *Journal of Drug Education* 8(3): 261-266, 1978.

Rapid smoking is generally considered to be the most effective single treatment method for the behavioral control of smoking. However, its safety has recently been questioned. In lieu of rapid smoking, a new risk-free strategy called focused smoking has been developed. While smoking at a normal rate, subjects are asked to focus on the realistic aversive consequences of smoking (e.g., burning sensations in mouth and throat, stained fingers and teeth). This study presents the results of a 6-month followup of nine subjects (five males and four females with an average age of 20.2 years) exposed to a comprehensive treatment program which included focused smoking. At the end of treatment, all nine subjects (who had been smoking an average of 20.55 cigarettes per day) reported that they had stopped smoking. This initial 100 percent effectiveness declined to 77.8, 66.7, and 55.6 percent at 1, 3, and 6 months, respectively. Of the four subjects who relapsed prior to the 6-month followup, two had resumed smoking at their pretreatment levels and two had reduced those levels by half. The 55.6 percent success rate compares favorably with success rates previously obtained with rapid smoking.

80-0837. Powell, D. R. **The Effect of a Multiple Treatment Program and Maintenance Procedures on Smoking Cessation.** Doctoral Dissertation, University of Michigan, Ann Arbor, University Microfilms 79-16795, 1979, 191 pp.

There were 51 subjects (29 women, 22 men) aged 18-62 years in this 5-day smoking cessation program. Subjects smoked between 8 and 60 cigarettes/day with a mean of 29.8. The mean number of years smoked was 17.5, and an average of five previous attempts at quitting smoking had been made. All subjects were required to attend an explanatory meeting (Phase I) and four consecutive treatment meetings that were held 1 week later (Phase II). Meetings lasted approximately 2 hours. All participants were involved in Phase II as a group of 51 people. At the end of Phases I and II, subjects were blocked according to the number of cigarettes they smoked per day and their sex and were randomly assigned to one of three maintenance conditions (Phase III). Each Phase III condition contained 17 subjects. There were three Phase III groups: support, telephone contact, and no contact controls. Phase IV involved the sending and returning of the 2-month followup form. At the end of Phase II, 100 percent of the subjects reported that they were abstinent. Subjects were asked to rate their status as a nonsmoker, ranging from an extremely comfortable nonsmoker to a smoker. Thirty-five subjects (68.6 percent) indicated that they were somewhat comfortable nonsmokers. Asked to rate their current desire for a cigarette, 80 percent of the subjects reported either a very small desire or some desire for a cigarette. All subjects reported that they could control their urges. At the 2-month followup 43 subjects were abstinent and 8 were recidivists. Information concerning support received, changes in weight and alcohol consumption, desire for a cigarette, ability to control urges, and difficulty in quitting was obtained.

80–0838. Sachs, D.P.L.; Hall, R. G.; Pechacek, T. F.; Fitzgerald, J. **Clarification of Risk-Benefit Issues in Rapid Smoking.** *Journal of Consulting and Clinical Psychology* 47(6): 1053-1060, December 1979.

Several articles on rapid smoking have stressed the theorized danger of this smoking abatement procedure while providing little evidence to support the claim. Additionally, they have disregarded the benefits of not smoking. This consideration of safety without benefit is found to be simplistic and potentially harmful. An analysis comparing the known risks and benefits is made, and the current literature on the efficacy and safety of rapid smoking is reviewed. Risk—benefit analysis reveals that rapid smoking is safe and effective for healthy subjects, but additional research is required to determine its safety and efficacy for persons with cardiac and pulmonary disease. (Auth. Abs.)

See also, **80–0786, 80–0812, 80–0817, 80–0819**

TOBACCO MANUFACTURING AND PROCESSING

80–0839. Brinkmann, G. **Cigarette Smoke Filtration and Ventilation.** *Tabak Journal International* (2): 121-122, 125, April 1979.

Substances contained in tobacco smoke in the form of particles can be reduced by means of filtration. The gaseous constituents can be affected only by the admixture of additives (e.g., silicic acid or meerschaum) to the filter material. In the smoke flow, ventilation reduces not only the proportion of particulate, but also of gaseous constituents. Filter retention can be considerably increased by the use of special materials, e.g., myria or acetate. Filtration capacity is maximized when using various forms of filters. Selective filtration is obtainable by means of additives to the filter material. Higher retentions are possible by the use of multiple filters with comparable pressure drop. Furthermore, improved constancy of the condensate values in the smoke may be obtained with ventilation by means of diversified pressure drop of the filter components. (Auth. Abs. Mod.)

80–0840. Kameswara Rao, B. V.; Gnana Kumar, D.; Chakraborty, M. K. **Quantitative Changes in the Polyphenolic Constituents of Flue-Cured Tobacco From Harvesting to Ageing.** *Tobacco Research* 4(2): 10-12, December 1978.

Quantitative variations in the polyphenolic constituents of two varieties of flue-cured tobacco (Kanakaprabha and Virginia Gold) from harvest to aging stage were studied. Chlorogenic acid and rutin levels were highest at the end of the leaf-drying stage; during aging there was a reduction in the levels of both these constituents. Nitrogen fertilization did not have any effect on these constituents. While Kanakaprabha contained more chlorogenic acid, Virginia Gold was richer in rutin. (Auth. Abs.)

80–0841. Shambhu, M. B.; Digenis, G. A. **Functionalized Macroreticular Resins as Effective and Selective Traps for Gaseous Tobacco Smoke Components.** *Journal of Macromolecular Science. Part A* 11(12): 2293-2299, 1977.

Macroreticular polystyrene resins with polyamine functions and copper (II) ions in chelated form were synthesized, and their abilities to selectively remove hydrogen cyanide and volatile aldehydes from tobacco smoke were determined. Reductions of up to 80 and 40 percent, respectively, were observed. The functionalized fixed-pore macroreticular resins were more efficient than their solvent-swellable "popcorn" counterparts in the removal of these gas-phase components of smoke. (Auth. Abs.)

See also, **80–0685, 80–0856**

TOBACCO ECONOMICS

80-0842. Chakaravarthy, S. **Tobacco—How Sweet Is Its Economic Aroma?** *Tabak Journal International* (6): 434-435, December 1978.

Before the establishment of the Commodity Board in India in 1976 by an Act of Parliament, tobacco interests were essentially left to private enterprise, and the economic aroma of tobacco was not always pleasant due to natural, national, and international factors. However, in 1977, for example, the Board came to the rescue of the growers following an unprecedented cyclone by disbursing 35 million rupees for replanting the crop and repairing the barns. The Tobacco Board of India is endeavoring through various plans to make Indian tobacco economically viable to all segments of the industry, be it the grower, buyer, or trader. (Auth. Abs.)

80-0843. Maxwell, J. C., Jr. **Cigar Sales Still Falling; Now at Half of Mid-1960s Level.** *Tobacco International* 181(23): 7-8, November 16, 1979.

During the past 15 years, cigar sales have been halved and continue to fall, although some people in the industry believe the volume could bottom out at 4 to 4.5 billion units. The premium-priced cigar (50¢ or more), however, accounts for 2.5 percent of the industry's unit volume and 15 percent of the dollar volume, a 12 and 15 percent growth rate, respectively, compared with 1973 volumes. Untipped cigarillos increased in sales volume from 1977 to 1978, perhaps due to Dutch Masters Camaroon Wiff sales. The cigar industry is presently testing an aromaless cigar called Flites in the new smoker market. Marketing data from various cigar companies are tabulated.

80-0844. Muralidhara Rao, N. K. **The Indian Tobacco Board and Its Role.** *Tabak Journal International* (6): 449-450, 1976.

India produces over 360 million kg of tobacco and ranks third among tobacco-producing countries, next only to the United States and China. The share of Indian flue-cured tobacco in international trade is around 11 percent, and here India ranks next only to the United States. In view of the importance of tobacco in the Indian economy, the government of India introduced the Tobacco Board Act of 1975, providing for development of the tobacco industry under the control of the Union Government. The duties of the Board include (1) regulation of production and curing of tobacco; (2) evaluation of national and international tobacco markets; (3) improvement of existing markets and development of new markets; (4) recommending minimum prices for export; (5) disseminating information useful to tobacco growers, dealers, and exporters; and (6) purchasing tobacco from growers when necessary to protect their interests.

BILLS AND LEGISLATION

80-0845. Cobey, C. **The Resurgence and Validity of Antismoking Legislation.** *University of California Law Review (Davis)* 7: 167-195, 1974.

Concern over unrestricted smoking in public places has become more vocal in the past few years. This article examines whether Federal, State, and local governments have the authority to restrict smoking of tobacco products in public places. The questions posed by this problem are treated by first examining the antismoking crusade of the earlier part of this century, determining why that movement failed, and indicating what lessons can be learned from its failure. Next, current evidence on smoking and health is considered. A brief section is devoted to public opinion on the issue of smoking. Legal bases for governmental restrictions of smoking are studied, followed by a summary of existing and proposed government actions to restrict smoking. The article concludes with a comment on the efficacy of restrictions, and further recommendations for additional action.

80-0846. Lindesmith, L. A. **Clean Indoor Air—A Fight for Rights for Health—Not Unwarranted Government Intervention. (Letter).** *Wisconsin Medical Journal* 78: 12-13, November 1979.

The Environmental Health Committee of the Governmental Affairs Commission of the State Medical Society has tried to address the issue of whether or not clean air is worthwhile in relationship to the proposed Clean Indoor Air Bill, Senate Bill 80, and its watered-down counterpart, Assembly Bill 80, currently being considered in the Legislature. The Committee unanimously endorsed the concepts and proposals of Senate Bill 80 which would provide for areas for smoking and for nonsmoking in public places, including government buildings, hospitals, and large restaurants. The fiscal impact of asking businesses and government agencies to provide signs and areas for smoking and for nonsmoking separately is negligible for any one business and a drop in the bucket for government buildings. Yet in 1976 U.S. Government agencies willingly spent 200 million dollars on pollution control measures to try to control the outside air. In 1978 the tobacco industry spent 800 million dollars just in advertising tobacco products and the American public spent as much money buying tobacco as it did paying doctor bills. Urging our legislators to provide pollution control of our indoor air can result in good health effects at almost no public expense.

GENERAL

80-0847. Advisory Council on the Environment of Quebec. **L'Usage du Tabac Dans les Endroits Publics et la Protection des Non-Fumeurs. [The Use of Tobacco in Public Places and Protection of Nonsmokers.]** The Government of Quebec, Publication No. ISBN 2-550-00193-1, May 1979, 289 pp., French.

The Advisory Council issued this report after a lengthy study of (1) the effects of tobacco smoke on the well-being and health of nonsmokers and (2) the legislation which regulates smoking in public places in other countries. The primarily Canadian and U.S. data reviewed the effects of passive smoking on fetuses, children, adults, and animals. International legislative efforts to control tobacco consumption in public places were reviewed for countries such as the U.S.S.R., the United States, Canada, and for Quebec. The Advisory Council concluded that with the absence of appropriate Canadian legislation in Quebec, legislative intervention at the provincial level was necessary to protect nonsmokers. Adoption of a basic law concerning smoking in public was proposed. The law would prohibit smoking in public transports, elevators, schools, museums, libraries, lecture halls, sport and artistic show places, health establishments, gymnasiums, and government conference rooms. The law would further require adequate ventilation in other public places (e.g., restaurants, clubs, and air terminals).

80-0848. Brass, A. **Where There's Smoke. (Letter).** *British Journal of Hospital Medicine* 21(3): 316, March 1979.

Despite the latest Surgeon General's report, tax dollars continue to be used in price support of the American tobacco industry whose farmers, the President declared, depend upon that income. Just how far a self-righteous bureaucracy should go in ordering how we live is probably going to be the top sociological topic of the 1980s. Already across the country there are signs that to smoke or not has become the litmus test of being a good American. Those who light up in a lift or train are likely to find themselves victims of flying insults or even handbags hurled by furious citizens spluttering about "fouling my air" and clearly itching for a return to the law of the guillotine. In California in November a referendum that would have banned smoking in just about every building and space to which the public is admitted (including restaurants) was only narrowly defeated. And among the ranks of the newly enlightened, to smoke at all—marihuana excepted—sinks one automatically to a par with those who want to kill whales, cut down all the forests in America, and build nuclear power stations on every grassy knoll.

80-0849. California Fire Incident Reporting System. **1978 California Fire Incident Reporting System Annual Report.** Office of the California State Fire Marshal, Sacramento, California, March 1979, 95 pp.

This annual report tabulates information such as number of fires, time of day and type of fire incident, property classifications, financial losses, source and form of ignition heat, and type and form of material first ignited. Natural product is one subcategory of "type of material first ignited" and includes rubber, grass, grain, food, coal, cork, leather, and tobacco. These various ignition sources are primarily responsible for fires outdoors and in dwellings (totals of 1,952 and 29,852, respectively). Smoking material is a subcategory of "form of heat causing ignition." In 1978 smoking materials' heat caused ignition primarily in dwellings and secondly in passenger transportation vehicles (totals of 5,175 vs. 1,629, respectively).

80-0850. Charlewood, G. P. **How to Prevent Patients From Smoking. (Letter).** *South African Medical Journal* 53(15): 569, April 15, 1978.

A physician who persuades all his patients who smoke to give it up describes some of the successful methods he uses. For women, a persuasive argument seems to be that smoking causes premature aging of the face. An outright refusal to look after pregnant women who smoke unless they give up the habit has to date been persuasive in all but one case. Discussion of the risks of lung cancer and emphysema is apparently useless, because "that only happens to old people."

80-0851. Claus, D.; Kornhuber, H. H. **Vor allem das inhalierende Zigarettenrauchen reduzieren. [Above All Reduce Cigarette Smoking With Inhalation.]** *Munchener Medizinische Wochenschrift* 121(48): 1583-1584, November 30, 1979, German.

The most realistic goal of an antismoking campaign should be reduction of cigarette smoking, and more tolerant attitudes toward cigar and pipe smokers. These opinions are based on information that cigarette smoking and inhalation are more significant risk factors than cigar and pipe smoking without inhalation. Statistical data from literature on mortality rates from bronchial and lung carcinomas, and the relative percentages of cigarette smokers, nonsmokers, and pipe or cigar smokers in a sample of cardiovascular patients and controls are cited to support these views. Changing from cigarette to pipe or cigar does not reduce the mortality risk because the habit of inhaling and hence the increased risk remains. It is recommended that cigars, but not cigarettes, be made more accessible, particularly to young people, by installing self-service shops and vending machines. Danish women who are habitual cigar smokers are characterized as being more

best copy available

sensible than German women who continue to smoke cigarettes in steadily increasing numbers.

80-0852. Dutartre, D.; Sertain, G. **Le Tabac, Fleau Social, et ses Consequences en Pathologie Broncho-Pulmonaire. [Tobacco—A Social Scourge and its Effects on Broncho-Pulmonary Pathology.]** *Ouest Medical* 30(10): 689-696, May 10, 1977, French.

A survey of medical literature on bronchopulmonary pathology and tobacco smoking is made. Psychological, sociological, and physiological factors of the smoking habit are briefly discussed. Various experimental studies are used to explain the mechanisms of action of tobacco smoke at the bronchial level. The complex role of other carcinogenic factors (e.g., radioactivity, air pollution, hormones, age one started smoking, amount smoked) is stressed. It is concluded that bronchopulmonary cancers and chronic obstructive bronchopathies are among the principal factors of morbidity and mortality in developed countries. The importance of the physicians' role is stressed in prevention of smoking, particularly in young people.

80-0853. Fletcher, C. M. **Health Education and the Control of Cigarette Smoking.** In: Bennett, A. E. (Editor). *Recent Advances in Community Medicine*. Volume I. Edinburgh, Churchill Livingstone, 1978, pp. 97-115.

This paper is a general discussion of health education, stopping smoking, and disease prevention. The print and broadcast media were cited as main channels for communicating the health effects of smoking. However, it was noted that health professionals and other role models were also important the latter in preventing teenage smoking. Further, antismoking legislation, taxation, and restriction of smoking in public places may be effective in controlling smoking in prevention of disease.

80-0854. Fritschler, A. L. **Smoking and Politics. Policymaking and the Federal Bureaucracy.** Englewood Cliffs, New Jersey, Prentice-Hall, Inc., 1975, 191 pp.

Since January 1966, a health warning has been printed on all cigarette packages sold in the United States. This book is a political science study of the cigarette labeling controversy that occurred in the 1960's. It deals with the Surgeon General's Report and the subsequent efforts of the Federal Trade Commission (FTC) to alert the public to the dangers of smoking by labeling cigarettes with a warning message. The study focuses upon the policymaking process in the FTC, its administrative procedures, and the external relations of the agency to Congress, the President, the courts, interest groups, the general public, and other agencies. Agency policymaking through rulemaking, public hearings, and the promulgation of the rules are discussed. Developments related to the consumer politics phenomenon of the early 1970's are also included.

80-0855. Great Britain Laboratory of the Government Chemist. **Tobacco Examination.** In: Great Britain Laboratory of the Government Chemist Report, 1977, pp. 4-5.

A brief history of the Great Britain Laboratory of the Government Chemist and its role in examining tobacco is given. The moisture level was controlled originally in order to prevent the incorporation of excessive water in manufactured tobacco such as roll tobacco. Added oil (used in the spinning process) was also used as an adulterant. The quantity of oil was limited in 1900. The determination of the moisture and inorganic contents for the control of tobacco revenue assessment ceased at the close of 1977 because of a fundamental change in the basis of the assessment, completing a notable era in the history of the Laboratory.

80-0856. Great Britain Laboratory of the Government Chemist. **The Hunter Committee.** In: Great Britain Laboratory of the Government Chemist Report, 1977, pp. 96-97.

Two important decisions were taken by the Independent Scientific Committee on Smoking and Health (the Hunter Committee) in Great Britain during the year. The first was to advise the Secretary of State to permit the use, under certain conditions, of two synthetic tobacco substitutes, Cytrel and New Smoking Material, in the manufacture of cigarettes. The decision was taken only after the applicants had satisfied the Committee by submitting evidence of thorough testing of the products in accordance with the guidelines promulgated in the first report of the Committee in 1975. The evidence submitted included the results of analyses of the smoke for a large number of components considered to be of importance to the health of smokers, acute studies with animals, short-term clinical tests, carcinogenicity and teratological studies, and long-term inhalation studies on animals. The second decision by the Hunter Committee was to recommend that tobacco manufacturers be allowed to use certain additives in cigarettes, hand-rolling tobacco, and cigar and pipe tobaccos. As a result, the Department of Health and Social Security was able to publish a list of nearly 400 permitted additives and the maximum proportions for their use. Others have since been added to the original list. Most of these compounds were natural or synthetic chemical flavorings, sugars, or humectants. The general basis on which they had been judged to be acceptable in the proportions specified was that they had been in regular use in smoking products in Western Europe or North America for 20 years or more without any evidence of harmful effects attributable to their use.

80-0857. Lemoine, J. M. **Groupe d'Etudes Contre le Tabagisme. [An Antismoking Study Group.]** *Broncho-Pneumologie* 27(2): 89-95, 1977, French.

Results are reported of a survey of tobacco consumption, tobacco product commercialization, tobacco-associated diseases, and methods of smoking control in 16 European countries. The results were obtained by means of a questionnaire and were discussed by the smoking control study group during the 25th Congress of the International Association for Broncho-Pneumology in Novi Sad (Yugoslavia). The statistics illustrate a continuous general increase in tobacco consumption, except in Belgium where consumption decreased in 1975 by 3 percent. A comparison of the commercial benefit figures with estimated expenses caused by the principal tobacco-associated diseases revealed that the cost of smoking is at least double the alleged benefit. It appears that 95 percent of all bronchogenic cancers, 50 percent of all chronic bronchitis cases (85 percent in Belgium), and vascular atheromatous diseases can be attributed to smoking. In all countries surveyed, with the exception of East Germany, the incidence of these three diseases parallels tobacco consumption. The annual incidence of bronchogenic cancers increased by 1.33 to 24.32 percent over a period of 2 to 43 years depending on the country. In East Germany, this incidence decreased from 40.6 in 1969 to 40.0 cases in 1974 per 100,000 population. The incidence of cardiovascular diseases over 10- to 20-year periods increased by 50 to 180 percent, except in East Germany and in the Netherlands. A favorable response to the suggestion of professional conversion made to planters, manufacturers, and distributors of tobacco was received from five countries. Seven countries expressed interest in forbidding advertising of tobacco products (a policy which has been in effect for several years in Sweden, Yugoslavia, and parts of France). All countries were in favor of education of adults and youth by way of radio, TV, and the press. Seven countries recommend initial education at the elementary school. Several countries forbid smoking in certain public places and eight countries are in the process of extending interdiction to all public places.

80-0858. Loftus, J. J. **Back-Up Report for the Proposed Standard for the Flammability (Cigarette Ignition Resistance) of Upholstered Furniture, PFF 6-76.** U.S. Department of Commerce, National Bureau of Standards, Publication No. NBSIR 78-1438, June 1978, 240 pp.

This report brings together data, information, and reports generated by the Center for Fire Research (CFR) at the National Bureau of Standards (NBS) and by others during 4 years of work on the development of a viable and reproducible test method for measuring the flammability of upholstered furniture. All of this information was used in the preparation of a recommended Proposed Standard for the Flammability (Cigarette Ignition Resistance) of Upholstered Furniture, PFF 6-76. The objective of the proposed standard was to reduce the number of upholstered furniture fire incidents which occur every year in the United States as a result of careless smoking. According to the 1974 National Household Fire Survey, of the 4.6 million residential fires, fabric items were identified as first to ignite in an estimated 469,000. In 93,000 cases, approximately 20 percent of the 469,000, upholstered furniture was the first item ignited. In 88 percent (82,000) of the upholstered furniture fire incidents, the probable cause (or ignition source) was estimated to be cigarettes, cigars, and other smoking-related materials. A survey of fire deaths showed that the most frequent cause (27 percent) was the residential fire where furnishings were the first item to ignite, and the ignition source was smoking. (Auth. Abs. Mod.)

80-0859. Smith, D. A. **We Should Be Doing Both.** *Pennsylvania Medicine* 81(12): 29, December 1978.

The controversy of the "safe cigarette" and relevant articles are discussed. A previous article stated that cigarettes today are less hazardous than in the pre-1960 era and that the technology to further define and reduce the toxic constituents of cigarettes is available (*Journal of the American Medical Association* 240(12): 1255-1259, September 15, 1978). It is possible that by removing toxic constituents from cigarettes and by reducing the amount of smoke inhaled the risk of smoking-associated disease will greatly diminish. Less hazardous cigarettes may or may not be a partial answer to the smoking habit and smoking-related disease. See also 79-0369.

80-0860. Stepney, R. **Homo Fumens.** *World Medicine* 14(6): 49-50, December 13, 1978.

Smoking is a psychological tool for those who claim that it counters boredom and fatigue, aids concentration, reduces the effects of stress, and helps to avoid emotional extremes. There is also evidence that smoking can counter the decrement in task performance following alcohol consumption. In addition, various studies indicate that smokers are able to manipulate their psychological state toward sedative or stimulant effects. Nicotine has been shown to attenuate stress and suppress aggressive behavior in animals and man. It is clear that if smoking does confer psychological benefits, the problem of preventing smoking will not be advanced by ignoring them.

80-0861. Tobacco International. **FTC Attack Employs Industry's Own Study.** *Tobacco International* 182(1): 22-23, January 11, 1980.

The Federal Trade Commission's (FTC) release of a confidential study sponsored by the Tobacco Institute has deeply disturbed many of those in the tobacco industry. The study had been intended to help combat increasing attacks by antismoking groups, but backfired when the FTC obtained it and sent it to Congress as part of the annual report on smoking. The study was conducted by the Roper Organization in 1978 and included information on public attitudes toward the tobacco industry, labeling, and nonsmoker-protective legislation. The Commission's report also asked that Congress approve funding for an antismoking informational campaign to run in the mass media, and to order rotating health warnings for cigarette packs. This practice is now in use in Sweden where some 16 different messages are used. The FTC said the labels should name diseases potentially caused by smoking. Responding to the annual report, the managing director of the Tobacco Growers Information Committee noted that the FTC itself admits that more than 90 percent of the adults in the United States know the content of the warnings on cigarette packages, but that few notice the warning any longer.

80-0862. Tobacco Reporter. **Anti-Smoking Momentum Threatens Cigarette Industry.** *Tobacco Reporter* 107(1): 12, January 1980.

A confidential study of public attitudes on cigarette smoking conducted for the Roper Organization was mistakenly released by the Federal Trade Commission. The findings reveal that public antismoking sentiment is on the upsurge, posing a major threat to the industry. Majorities are in favor of segregating smokers and nonsmokers in workplaces, restaurants, and transportation vehicles. Only 27 percent of those surveyed rated the tobacco industry as "very interested" or "moderately interested" in the safety of persons using their products.

80-0863. World Health Organization, 31st World Health Assembly. **Health Hazards of Smoking.** *Bulletin Europeen de Physiopathologie Respiratoire* 14(4): 91p-92p, July-August 1978.

In view of the health hazards of smoking, the World Health Organization (WHO) in this resolution urges member states (1) to strengthen health education programs; (2) to adopt comprehensive measures to control smoking; (3) to protect the rights of nonsmokers; and (4) to seek alternatives to replace tobacco growing. WHO also requested the Director General to take certain actions, including efforts to continue to intensify WHO activities related to the control of smoking and to encourage research on the causes of smoking.

AGRICULTURE

80–0864. Merker, J.; Stoertzer, T. **Ein Beitrag zur chemischen Geiztriebhemmung nach dem Kopfen des Tabaks. [Contribution to the Chemical Inhibition of Sucker Growth Following Tobacco Topping.]** *Berichte des Instituts fuer Tabakforschung, Dresden* 24/25: 5-37, 1977-78, German.

Field trials were carried out from 1971 to 1977 in East Germany using systemic and various contact sucker control agents on different varieties of tobacco plants. The East German systemic sucker control agent Malzid 30 (Mz30), an aqueous emulsion of 30 percent maleic hydrazine (MH) and 30 percent diethanolomine, proved to be a good to very good inhibitor of tobacco sucker growth when sprayed at the rate of 15 l/ha or 2.5 percent immediately after topping. It is released for use in tobacco cultivation in East Germany, pending establishment of the legal MH residue limit. With use of Mz30 the yield is about 4 percent higher than with hand-suckered plants. Alkaloid levels (mainly nicotine) were increased up to 53 percent over the topped controls, depending on the year, weather conditions, and tobacco variety. Nicotine and nornicotine content of the topped and Mz30-suckered Burley tobacco plants varied from 1.46 to 2.70 and 0.09 to 0.18 percent, respectively, depending on location and harvest phase. These levels were only insignificantly higher than those in untreated topped plants. Other technological and chemical characteristics (volatile bases, total nitrogen, total protein) were not significantly affected by the use of Mz30. Average MH residues of Mz30-suckered plants were 40 ppm, if harvested 27-33 days after spraying. As a rule, MH residues were lower than 80 ppm, the legal limit in West Germany, provided the leaves were not harvested earlier than 14 days after spraying with Mz30. The shorter the time interval between spraying and harvest, the higher were the MH residues in the leaves. These residue levels decreased by an average of 19 to 32 percent after curing, depending on the harvest phase. The imported Off-Shoot-T(OST) product and the East German Cu 452 product were found to be the most effective contact agents tested for sucker control.

See also, **80–0874**

80–0865. Tobacco International. **Sucker Control, Reduced MH Residue Promised With FST-7.** *Tobacco International* 182(3): 34-35, February 8, 1980.

A new tobacco sucker product called FST-7 has been developed which contains a contact-type sucker control chemical and maleic hydrazide (MH). The product controls suckers by restricting cell division in new plant growth. When compared with the suggested rate of MH applied at the same time, FST-7 results in a higher degree of sucker control and lower residue of MH. FST-7 should be applied in solutions containing 6 percent of the product. According to the manufacturer, results can be expected within 1 hour after application from the contact portion of FST-7. The potassium salt MH in the product provides full-season systemic control of new sucker growth.

80–0866. Zilkey, B. F. **Effect of Seedbed Growing Medium and Number of Transplant Pullings on Certain Agronomic, Chemical, and Physical Leaf Measurements of Flue-Cured Tobacco in Ontario.** *Tobacco International* 182(4): 73-74, February 22, 1980.

Two seedbed growing materials, muck and a 50/50 mixture of muck-peat, and three sequential seedling pullings from each seedbed medium were studied in the greenhouse in a preliminary 2-year experiment to determine relationships of growth medium and time of pulling to certain seedling growth and agronomic, physical, and chemical characteristics of flue-cured tobacco. Seedling shoot and root dry weight nearly doubled over the 10- to 14-day period between pullings one and three from both seedbed materials. Seedlings from the muck-peat seedbed were easier to pull by hand than from muck alone. Yield, cash returns, and total alkaloid levels for cured leaves were higher for pulling one transplants than for other pullings from both seedbed media. However, this must be balanced against the higher field mortality of pulling one transplants. Price, filling value, lamina weight, and reducing sugar levels for cured leaves were unaffected by number of pullings or type of seedbed medium. (Auth. Abs.)

CHEMISTRY

80–0867. American Chemical Society. **Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke.** Proceedings of American Chemical Society Symposium, 173rd American Chemical Society Meeting, Agricultural and Food Chemistry Division, New Orleans, 1977, 597 pp.

This publication contains most of the papers delivered at the symposium "Recent Advances in the Chemical Composition of Tobacco and Tobacco Smoke" held under the sponsorship of the Agricultural and Food Chemistry Division of the American Chemical Society. The papers were given as a part of the 173rd American Chemical Society Meeting in New Orleans, Louisiana, March 20-25, 1977. Various chemical constituents of tobacco leaf (i.e., proteins, amino acids, terpenoids, carbonyls, lipids, phenols, nitrates) are discussed as is alkaloid biosynthesis. Mechanisms of smoke formation are among the physical properties discussed. In addition, topics such as genetic constituent control and agricultural practices (e.g., fertilization) are presented.

80–0868. Baker, R. R. **Combustion and Thermal Decomposition Regions Inside a Burning Cigarette.** *Combustion and Flame* 30(1): 21-32, 1977.

Cigarettes have been smoked under continuous draw conditions in an atmosphere containing oxygen-18, in order to locate the regions where carbon monoxide and dioxide are formed by thermal decomposition and combustion of tobacco. The carbon oxides found inside the burning zone (combustion coal) at temperatures above 500°C are formed largely by combustion of tobacco. Those found behind the coal at temperatures below 500°C are produced mainly by thermal decomposition of tobacco. The gas concentration at a given position inside the cigarette is dependent on the net chemical production of the gas, and the net rates of diffusive and convective flow through that region. Similarly, the temperature at any point inside the cigarette is determined by the net heat released by local chemical reaction, as well as by the heat transferred by conduction, convection, and radiation. The net rates of formation of heat and gases by chemical reaction at various positions inside the cigarette have been calculated from the empirical temperature and gas distributions, using heat and mass transfer equations. There is a strong exothermic region inside the coal where oxygen is consumed and the carbon oxides are formed by combustion. There is a strong endothermic region behind the coal where the carbon oxides are formed by thermal decomposition of tobacco. (Auth. Abs.)

80–0869. Canadian Council on Smoking and Health. **Tar, Nicotine, Carbon Monoxide Figures Released.** *Canadian Council on Smoking and Health Newsletter* 6(1): 10-15, January 1980.

The Health and Welfare Minister of Canada released a table of tar, nicotine, and carbon monoxide (CO) levels in cigarettes sold in Canada. For those brands with low tar and nicotine levels, carbon monoxide levels seem correspondingly low. For brands with higher tar and nicotine levels, however, CO levels vary. The Minister advised that those who cannot stop smoking should lessen their risk of smoking-related disease both by choice of cigarette brand and by reducing the amount of smoke inhaled. The latter can be accomplished by lengthening the period between cigarettes, lengthening the period between puffs, not inhaling, removing the cigarette from the mouth after each puff, and throwing away longer butts.

80–0870. Dalferes, E. R., Jr.; Webber, L. S.; Radhakrishnamurthy, B.; Berenson, G. S. **Continuous-Flow (AutoAnalyzer I) Analysis for Plasma Thiocyanate as an Index to Tobacco Smoking.** *Clinical Chemistry* 26(3): 493-495, 1980.

A procedure for determining thiocyanate in plasma with an autoanalyzer modified with a 50 mm tubular flow cell to increase the sensitivity of thiocyanate detection is described. The determination procedure is suitable for distinguishing smokers from nonsmokers, the mean values being 151 (S.D. 43)μmol/l and 62(S.D. 19)μmol/l, respectively. To determine how the number of cigarettes smoked affects thiocyanate concentrations, the data for smokers were divided into two groups: those smoking fewer than 20 cigarettes a day and those smoking 20 or more a day. The mean (±2 S.E.) thiocyanate value was insignificantly higher for persons smoking more than 20 cigarettes a day (178 ± 54μmol/l) than for individuals smoking 20 or fewer a day (148 ± 19μmol/l). (Auth. Abs. Mod.)

80–0871. Enzell, C. R.; Wahlberg, I.; Aasen, A. J. **Isoprenoids and Alkaloids of Tobacco.** *Fortschritte der Chemie Organischer Naturstoffe* 34: 1-79, 1977.

Tobacco isoprenoids and alkaloids and their degradation products are discussed. Since a large number of compounds belonging to these groups has been obtained from tobacco in recent years, some hypotheses as to the modes of formation of the nor-compounds can now be presented. This article is arranged according to the hypothetical biogenetic model. A comprehensive compilation of constituents of the groups selected is given. The major groups discussed are (1) carotenoids and acyclic isoprenoids, (2) diterpenoids, (3) triterpenoids and steroids, (4) cyclic sesqui- and monoterpenoids, (5) pyridine derivatives, (6) pyrrole and pyrrolidine derivatives, (7) pyrazine derivatives, and (8) miscellaneous nitrogen-containing constituents.

80–0872. Hamashima, T.; Koiwai, A. **Hatabako no Kokitsumi to Seiju Sanseibu Seibun ni Kausuru Kenkyu. [Tobacco Aroma and Taste and Acidic Constituents in the Essential Oil.]** *Bulletin of the Okayama Tobacco Experiment Station* 38: 97-104, December 1977, Japanese.

Steam-volatile acidic constituents of various bright tobacco samples which were judged as aromatic, semiaromatic, and aroma-deficient by sensory test were analyzed by gas-chromatography. About 70 clear peaks were obtained as major constituents in the acidic fraction. Thirteen peaks were high in the aromatic tobacco samples and low in aroma-deficient ones. The reverse was the case with 10 peaks. The rest of them had no clear relation to the judgment of sensory test. The ratios of the total values of 13 aromatic constituents of semiaromatic and aroma-deficient tobacco samples to those of aromatic ones were 0.5-0.2 and 0.2-0.1, respectively. The ratios of the total values of 10 aroma-retardant constituents of semiaromatic and aroma-deficient tobacco samples to those of aromatic ones were 0.9-6 and 2-4, respectively. As for the ratio of the value of the 13 constituents to those of the 10 aroma-retardant constituents, they were 99-129 in aromatic tobacco samples, 11-70 in semiaromatic ones, and below 10 in aroma-deficient ones. (Auth. Abs.)

80-0873. Hecht, S. S.; Chen, C.H.B.; Hoffmann, D. **Tobacco-Specific Nitrosamines: Occurrence, Formation, Carcinogenicity, and Metabolism.** *Accounts of Chemical Research* 12(3): 92-98, March 1979.

The results described in this review indicate that the tobacco-specific carcinogens, *N'*-nitrosonornicotine (NNN) and 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK), may be causative factors in the various cancers associated with tobacco usage. These nitrosamines are derived predominantly from the major tobacco alkaloid, nicotine, by nitrosation during the curing and smoking of tobacco. Other tobacco alkaloids may also be precursors to carcinogenic nitrosamines. Recently *N'*-nitrosoanatabine, another tobacco-specific *N*-nitrosamine, has been identified in tobacco (0.6-13 ppm), cigarette smoke (0.33-4.6 μg/cigarette), and cigarette sidestream smoke (0.15-1.5 μg/cigarette). Data on the carcinogenic activity of this nitrosamine are not yet available. Since NNN and NNK form during curing, it is feasible to reduce their concentrations in tobacco by appropriate management of the curing and related processes. Formation during smoking can also be inhibited. Another approach to reducing tobacco-related cancers begins with an understanding of the metabolic activation and detoxification of tobacco-specific nitrosamines. The enzymes that mediate these transformations can be induced or inhibited by environmental modifiers. Such modifiers may increase or decrease the carcinogenic effects of these nitrosamines, several of which undergo metabolic α-hydroxylation, a likely activation process.

80-0874. Higman, H. C., Severson, R. F.; Chortyk, O. T.; Arrendale, R. F. **Pyrolytic Evaluation of Close-Grown Tobaccos.** *Beitraege zur Tabakforschung International* 10(1): 65-72, December 1979.

A pyrolytic method for evaluating the smoke properties of tobacco materials for their potential to produce biologically significant smoke phenols and polynuclear aromatic hydrocarbons (PAH) has been applied to close-grown tobacco, in comparison to conventionally grown tobacco. Analyses were performed on the lipids of tobacco samples to attempt correlations with amounts of polynuclear aromatic hydrocarbons in pyrolyzates. Relative to a standard flue-cured tobacco, the close-grown tobacco samples contained lower nicotine, neophytadiene and hydrocarbon levels, and lower sterols, solanesol, and fatty acid levels. Close-grown tobacco pyrolyzates contained equivalent PAH levels and higher levels of phenols. Based on the pyrolysis data, the close-grown tobaccos do not offer a more desirable smoking product than conventionally grown flue-cured tobaccos. However, the lower levels of nicotine and higher amounts of smoke phenols may possibly make the close-grown materials suitable for inclusion into low-nicotine tobacco sheet products. (Auth. Abs.)

80-0875. Kung, S. D. **Isoelectric Points of the Polypeptide Components of Tobacco Fraction 1 Protein.** *Botanical Bulletin of Academia Sinica* 17(2): 185-191, July 1976.

Isoelectric focusing of *S*-carboxymethylated Fraction 1 protein from *Nicotiana tabacum* in polyacrylamide gel has resolved the subunits into their component polypeptides. There are three polypeptides in the large subunit and two in the small subunit. The isoelectric points are 6.10, 6.05, and 6.00 for the large subunit polypeptides and 5.35 and 5.31 for the small subunits. (Auth. Abs.)

80-0876. Matsushima, S.; Ishiguro, S.; Sugawara, S. **Composition Studies on Some Varieties of Tobacco and Their Smoke. I. Major Components in Smoke Condensate.** *Beitraege zur Tabakforschung International* 10(1): 31-38, December 1979.

The relative proportions of the major smoke components and N-containing components (e.g., pyridines) in the semivolatile phase and the nonvolatile phase from some varieties of tobacco, i.e., flue-cured, Burley, and Turkish tobacco, were determined. In flue-cured tobacco smoke, 2-furfural, 5-methylfurfural, 5-hydroxymethylfurfural, and other carbohydrate pyrolysates, catechols, and organic acids were present in higher concentrations than in the smoke from the other tobaccos. In Burley tobacco smoke, myosmine was present in much higher concentration than in the smoke from the other tobaccos. In Turkish tobacco smoke, glycerol and β-methylvaleric acid were both present in much higher concentrations. (Auth. Abs.)

80-0877. Michael, L. W.; Finelli, V. N.; Elia, V. J.; Petering, H. G.; Lee, S. D.; Campbell, K. I. **Interaction of Metal Binding Agents From Combustion Products With Trace Metals.** In: *Trace Substances in Environmental Health—9.* Proceedings of the University of Missouri's Annual Conference on Trace Substances in Environmental Health, University of Missouri, Columbia, 1975, pp. 355-360.

Metal binding agents present in the environment may react with trace metals, thus altering essential biological functions. Combustion products from tobacco, gasoline, and coal as well as from other pyrolytic processes are ubiquitous sources of such material. The potential for such interactions in biological systems has been demonstrated by comparing the copper binding activity of condensates from selected sources, such as tobacco smoke, coal, and auto exhaust. The reactivity with other metals was also examined. The isolated metal binding fractions have been tested for effects on metalloenzymes and mitochondrial respiratory activity. In addition, the efficacy of this concept was tested in vivo using a chelating agent, 3-ethoxy-2-oxo-butyraldehyde *bis* (thiosemicarbazone). This compound has been found to alter cholesterol metabolism in a manner similar to that observed in copper-deficient rats. (Auth. Abs. Mod.)

80-0878. U.S. Environmental Protection Agency. **Scientific and Technical Assessment Report on Nitrosamines.** U.S. Department of Commerce, National Technical Information Service, Publication No. PB-273 055, June 1977, 99 pp.

Nitroso compounds, as well as nitrates and amines, are found in a variety of pharmaceutical products, constituting another potential for human exposure. Reported values of nitroso compounds in cigarettes range from 0 to 180 ng/cigarette. A one pack/day smoker may then be exposed to a few micrograms per day. Nitrite formation in human saliva, particularly among smokers, has been estimated at about 6 to 10 mg/day. Human saliva in smokers also has been found to contain thiocyanate ion, which has been shown to catalyze the reaction of nitrite and secondary amines to form nitrosamines. Tobacco smoke contains several secondary amines and oxides of nitrogen. Preliminary estimates suggest that exposure to precursor amines and nitrogen oxides from food, water, and smoking is probably greater than that through inhalation.

PHARMACOLOGY AND TOXICOLOGY

80-0879. Altland, P. D.; Rattner, B. A. **Effects of Nicotine and Carbon Monoxide on Tissue and Systemic Changes in Rats.** *Environmental Research* 19(1): 202-212, June 1979.

Male rats were exposed to carbon monoxide (CO) (100, 250, or 500 ppm) and/or injected intraperitoneally at hourly intervals for 4 hours with a subconvulsive dose of nicotine (0.5 or 1.0 mg/kg) to determine systemic and tissue changes. Each agent alone induced only slight changes in blood glucose and lactic acid, and no change in plasma enzyme activities. Nicotine (1.0 mg/kg) plus CO (100-500 ppm) produced elevated blood glucose and lactic acid, and increased plasma aspartate aminotransferase activity. Plasma alanine aminotransferase activity was increased with 1.0 mg/kg nicotine and 500 ppm CO. Nicotine (1.0 mg/kg) induced hypothermia and a low incidence in neutral fat droplets in thigh muscles, whereas nicotine in conjunction with CO produced a slightly more pronounced hypothermia and a high incidence of neutral fat deposition in thigh muscles. Plasma corticosterone concentration was not consistently altered in any group. This study demonstrates that nicotine and CO, in combination, induce more profound systemic and tissue changes than either agent alone. These changes are probably caused by the direct action of catecholamines released by nicotine, and prolonged nicotine toxicity due to the inhibitory effects of CO on hepatic drug metabolism. (Auth. Abs.)

80-0880. Baker, E. L., Jr.; Peterson, W. A.; Holtz, J. L.; Coleman, C.; Landrigan, P. J. **Subacute Cadmium Intoxication in Jewelry Workers: An Evaluation of Diagnostic Procedures.** *Archives of Environmental Health* 34(3): 173-177, May-June 1979.

An outbreak of cadmium intoxication in a jewelry factory provided an opportunity for evaluating the usefulness of diagnostic procedures used to evaluate human cadmium toxicity. Blood cadmium levels in workers exposed to cadmium were higher (0.93 vs. 0.38μg/100 ml) than in unexposed workers. A dose-response relationship was noted between blood cadmium level and symptom prevalence in four symptoms (dyspnea, chest pain, dysuria, and dizziness). Segmental hair analysis revealed highest cadmium levels (up to 19μg/g) in segments formed prior to cadmium exposure, suggesting that extrinsic contamination was the primary source of cadmium in the hair. Blood cadmium levels were not related to cigarette smoking. Although mean blood cadmium levels were highest among workers who smoked one or more packs of cigarettes/day, cigarette consumption was not related to increased cadmium levels in other groups of workers. Urine cadmium levels were also unrelated to cigarette consumption rate. Levels of β_2-microglobulin were within normal limits. No significant renal or pulmonary dysfunction was noted. Symptoms ceased after a cadmium-containing brazing alloy used in jewelry production was replaced, yet urine cadmium levels remained persistently elevated in four workers. Blood cadmium determinations were found to be useful in evaluating symptoms potentially related to cadmium intoxication. (Auth. Abs. Mod.)

80-0881. Ball, L. M.; Plummer, J. L.; Smith, B. R.; Bend, J. R. **Benzo(*a*)pyrene Oxidation, Conjugation and Disposition in the Isolated Perfused Rabbit Lung: Role of the Glutathione S-Transferases.** *Medical Biology* 57(5): 298-305, October 1979.

The isolated perfused rabbit lung metabolized 7-11 percent of 20μmol of (^{14}C)-benzo(*a*)pyrene added in the perfusion medium in 1 hour. The major metabolite formed was 3-

hydroxybenzo(*a*)pyrene, both free (30-40 percent of the total metabolites) and conjugated (4 percent of total metabolites). Quinones comprised 15 percent of the total and metabolism at the 9, 10 position accounted for a further 10 percent. Forty percent of the water-soluble metabolites were chromatographically identical to the glutathione conjugate of benzo(*a*)pyrene 4,5-oxide. Sulphate and glucuronide conjugates were formed in small but detectable amounts, principally from phenols, but also from dihydrodiols. After 1 hour the more water-soluble conjugates had diffused from the lung into the perfusion medium, but the majority (60-90 percent) of the metabolic products were still concentrated within the lung. The lung's limited ability to conjugate its major metabolites of benzo(*a*)pyrene with sulphuric or glucuronic acid, coupled with slow elimination of the products formed, particularly dihydrodiols, may contribute to the susceptibility of this organ to polycyclic aromatic hydrocarbon-induced carcinogenesis. (Auth. Abs.)

80–0882. Basrur, P. K.; Basu, P. K. **Effect of Cigarette Smoke on the Surface Structure of the Conjunctival Epithelium.** *Canadian Journal of Ophthalmology* 15(1): 20-23, January 1980.

Syrian golden hamsters were exposed to cigarette smoke in an intermittent smoke exposure device, and the surface structure of the tarsal conjunctiva was compared with that of sham-smoked and cage-held controls. Scanning electron microscopy revealed distortion and loss of microvilli of the conjunctival cells in the smoke-exposed animals. (Auth. Abs.)

80–0883. Bernfeld, P.; Homburger, F.; Russfield, A. B. **Cigarette Smoke-Induced Cancer of the Larynx in Hamsters (CINCH): A Method to Assay the Carcinogenicity of Cigarette Smoke.** *Progress in Experimental Tumor Research* 24: 315-319, 1979.

An experimental method is now available for comparative studies of cigarettes and as a guide toward less hazardous smoking materials. Cigarette smoke inhalation in susceptible, inbred Syrian hamsters results in carcinoma of the larynx, histologically identical to human laryngeal carcinoma, which has been proven epidemiologically to be associated with smoking. Statistically highly significant cancer incidences and their direct relation to the dose of tobacco smoke inhaled qualify this procedure as a valid bioassay of tobacco smoke carcinogenesis.

80–0884. Cohen, G. M.; Mehta, R.; Meredith-Brown, M. **Large Interindividual Variations in Metabolism of Benzo(*a*)pyrene by Peripheral Lung Tissue From Lung Cancer Patients.** *International Journal of Cancer* 24: 129-133, 1979.

A very large variation (44-fold) was observed in the ability of short-term organ cultures of peripheral lung tissue from lung cancer patients to metabolize the environmental carcinogen benzo(*a*)pyrene to organic solvent-soluble metabolites. The amounts of benzo(*a*)pyrene (2μM) metabolized ranged from little (1 percent) to almost total (96.2 percent) metabolism within 24 hours of culture. Previous work has suggested a relationship between susceptibility to lung cancer and the inducibility of aryl hydrocarbon hydroxylase activity in cultured human lymphocytes. The metabolic fate of carcinogenic polycyclic aromatic hydrocarbons in the respiratory tract in vivo is undoubtedly more closely mimicked by short-term organ culture of human lung than by cultured lymphocytes. The very wide interindividual variation observed in pulmonary metabolism of benzo(*a*)pyrene in this study and the large variations in covalent binding to human bronchial DNA observed by others strongly suggest that there may be little basis for screening humans for variations in lymphocyte aryl hydrocarbon hydroxylase activity as a means of assessing their susceptibility to lung cancer. (Auth. Abs.)

80–0885. Cohen, J. D.; Bartsch, G. E. **A Comparison Between Carboxyhemoglobin and Serum Thiocyanate Determinations as Indicators of Cigarette Smoking.** *American Journal of Public Health* 70(3): 284-286, March 1980.

Cigarette smoking histories were compared to carboxyhemoglobin and serum thiocyanate concentrations obtained from 426 smokers and 191 nonsmokers. The mean levels of both carboxyhemoglobin and serum thiocyanate were significantly ($p<0.001$) higher among cigarette smokers and correlated with the number of cigarettes smoked per day. The mean carboxyhemoglobin level for smokers vs. nonsmokers was 4.5 ± 2.5 vs. 1.1 ± 1.4 percent. Mean serum thiocyanate level was 180.2 ± 55.7 vs. 73.5 ± 48.3 μm/l for smokers and nonsmokers, respectively. The specificity of both procedures was 81 percent, and serum thiocyanate had a higher sensitivity (93 vs. 83 percent), making it potentially more suitable for use as an index of cigarette smoking. (Auth. Abs. Mod.)

80–0886. Furst, A.; van Breda Kolff, K.; Dempsey, D. A. **Pulmonary Tumor Induction: Three Hydrocarbons Compared.** *Proceedings of the Western Pharmacology Society* 22: 269-271, 1979.

This study compares the carcinogenic activity of three aromatic hydrocarbons: benzo(*a*)pyrene (BaP), 3-methylcholanthrene (3-MC), and dimethylbenzanthracene (DMBA) in mice. These materials were quantitatively introduced using the intratracheal instillation method. The order of increasing potency of carcinogenic activity was BaP<DMBA<3-MC. Pulmonary adenomas were found on all animals. Squamous cell carcinomas were observed exclusively in the 3-MC-treated animals.

80–0887. Fuxe, K.; Everitt, B. J.; Hokfelt, T. **On the Action of Nicotine and Cotinine on Central 5-Hydroxytryptamine Neurons.** *Pharmacology, Biochemistry and Behavior* 10(5): 671-677, May 1979.

The actions of nicotine and its main metabolite cotinine on 5-hydroxytryptamine (5-HT) neurons in the brain of the rat have been assessed biochemically (on turnover, uptake, release, overflow, and binding of 5-HT in brain) and functionally (on extensor reflex activity, which is 5-HT dependent). Nicotine and cotinine in repeated doses of 2 mg/kg caused a reduction of brain 5-HT turnover, which was not blocked by pretreatment with mecamylamine, and nicotine significantly inhibited the effects of norfenfluramine and 5-methoxydimethyltryptamine on extensor reflex activity, effects counteracted by mecamylamine. In low concentrations cotinine weakly inhibits the uptake and retention of 5-HT and also increases its spontaneous release in vitro. The biochemical findings suggest that the reduction of 5-HT turnover caused by high doses of nicotine are mediated, at least in part, by its main metabolite cotinine. The experiments on extensor reflexes indicate that nicotine can block the functional expression of 5-HT receptor activity in the spinal cord by an action beyond the 5-HT receptor at nicotine-like cholinergic receptors whose location is also discussed. (Auth. Abs.)

80–0888. Greene, F. E.; Jernstrom, B. **Epoxide Hydratase and Glutathione *S*-Transferase Activities in Human Lungs.** *Cancer Letters* 8(3): 235-239, January 1980.

It has been reported that human lung has cytochrome P-450 dependent monooxygenase activity which is necessary for the formation of mutagenic and carcinogenic metabolites of polycyclic aromatic hydrocarbons, such as benzo(*a*)pyrene. In this study, epoxide hydratase and glutathione *S*-transferase, enzymes important in the further metabolism of certain benzo(*a*)pyrene metabolites formed by the monooxygenase system, have been detected in human lung tissues from patients undergoing surgery for lung cancer. (Auth. Abs.)

80–0889. Guimard, J. **Etude de l'Action du Condensat de Fumee de Cigarette sur le Debut de la Morphogenese du Poumon de Poulet Cultive in Vitro. [Study of the Effect of Cigarette Smoke Condensate on Initial Morphogenesis of the Chick Embryo Lung in Vitro.]** *Comptes Rendus des Seances de la Societe de Biologie et de ses Filiales* 171(3): 553-559, 1977, French.

Modification of morphogenetic relations between the mesenchyme and bronchial epithelium of the chick embryo lung was studied in vitro using cigarette smoke condensate (CSC). The cultures containing the CSC-treated mesenchyme showed a delayed morphological evolution of the associated mesobronchi in comparison with the controls. In contrast, evolution of the mesobronchi pretreated with CSC and associated with untreated mesenchyme appeared comparable to that of control mesobronchi. Mitotic activity of the mesenchyme associated with an untreated (normal) mesobronchus was significantly decreased by CSC. Histological examination revealed more accumulated cell fragments in the epithelium associated with the CSC-treated mesenchyme than in the controls. In contrast, general histological characteristics were identical in the associations of CSC-treated mesobronchi with a normal mesenchyme and in the control cultures. Morphogenesis of the mesobronchus of a chick embryo lung is modified in association with a CSC-pretreated mesenchyme inductor. Morphogenesis is unchanged if only bronchial epithelium is pretreated with CSC.

80–0890. Hamosh, M.; Simon, M. R.; Hamosh, P. **Effect of Nicotine on the Development of Fetal and Suckling Rats.** *Biology of the Neonate* 35(5/6): 290-297, 1979.

Nicotine, administered at a dose of 100μg/kg/day from day 14 of gestation, did not affect maternal food intake, weight gain, length of gestation, litter size, or fetal development; however, a daily dose of 1 mg/kg led to smaller litter size and higher incidence of stillbirth. Continued maternal administration of nicotine (100μg/kg/day) until 12 days post partum did not affect newborn growth (body weight and length and size of heart and lung) during the first week after birth; during the second week, however, the nicotine-treated group lagged behind the controls. The stomachs of pups of nicotine-treated rats contained less food than those of controls; this difference increased with age, becoming more than 40 percent at 12 days. It is suggested that lower milk production of nicotine-treated rats interferes with the normal development of the offspring during periods of rapid growth. (Auth. Abs.)

80–0891. Hoffmann, D.; Rivenson, A.; Hecht, S. S.; Hilfrich, J.; Kobayashi, N.; Wynder, E. L. **Model Studies in Tobacco Carcinogenesis With the Syrian Golden Hamster.** *Progress in Experimental Tumor Research* 24: 370-390, 1979.

The Syrian golden hamster (SGH) is today's most widely used animal for inhalation studies with tobacco smoke. Special smoking devices have been developed for the passive inhalation by hamsters of cigarette smoke. Since only a small portion of the carcinogenic smoke particulates reaches the trachea and bronchi, tumors are rarely seen in the lung. Most histopathologic changes occur in the larynx, the organ with the highest tar deposition. The changes in the larynx range from hyperplasia to papillomatous growths to occasional carcinomas, and in susceptible inbred lines of SGH to high incidences of laryngeal carcinoma. The smoke of experimental cigarettes which produces tar with reduced carcinogenic activity to mouse skin shows also reduced tumorigenic activity in the hamster larynx. Inhalation studies are also helpful for measuring the tumor-promoting activity of cigarette smoke in the larynx of hamsters where tumors are initiated with a carcinogenic hydrocarbon. During curing and smoking of tobacco, *N'*-nitrosonornicotine is formed from nicotine and nornicotine, and *N*-nitrosodiethanolamine is formed from diethanolamine, a solubilizing agent used for the spraying of tobacco with an aqueous solution of maleic hydrazide. Both nitrosamines induce papillomas in the trachea and carcinomas in the nasal cavity of the Syrian hamster. Other tobacco-specific *N*-nitrosamines are currently being assayed for carcinogenic activity and organ specificity in the SGH. (Auth. Abs.)

80–0892. Holt, P. G.; Finlay-Jones, L. M.; Keast, D.; Papadimitrou, J. M. **Immunological Function in Mice Chronically Exposed to Nitrogen Oxides (NO_x).** *Environmental Research* 19(1): 154-162, June 1979.

BALB/c mice were exposed to 10 ppm nitric oxide (NO) or nitrogen dioxide (NO_2) for 2 hours daily on weekdays, for varying periods up to 30 weeks. At intervals of approximately 7 weeks, groups of animals were subjected to a variety of pathological and immunological examinations: serum antibody responses to both T-dependent and T-independent antigens, spleen cell phytohemagglutinin and graft vs. host responses, capacity to reject a transplanted tumor, routine hematology, and lung pathology. Tests of immune function indicated suppression resulting from chronic exposure to these gases, and in most cases data indicated enhancement of immunological reactivity by shorter exposures. Nitrogen dioxide appeared somewhat more potent than NO in this regard, but the differences observed were not great. Chronic NO exposure produced a more severe lung pathology, with clear evidence of paraseptal emphysema. (Auth. Abs.)

80–0893. Huber, G. L.; Drath, D.; Davies, P.; Hayashi, M.; Shea, J. **The Alveolar Macrophage as a Mediator of Tobacco-Induced Lung Injury.** *Chest* 77(2): 272, February 1980.

The pulmonary alveolar macrophage (PAM) may represent an important pathogenic link between tobacco consumption and the development of lung disease. To study this, PAMs were harvested from rats exposed to fresh whole tobacco smoke for varying periods of up to 180 consecutive days. Tobacco dosimetry was adjusted to an exposure equivalent to 1.5 packs per day in man. At 30 days, PAM were metabolically activated, exhibited enhanced ingestion of inert particles and a depression in intracellular killing of staphylococci. An increase in macrophage cell diameter and a progressive increase in intracellular-lipid was demonstrated. Superoxide dismutase activity in PAM, an enzyme that protects the host against the damaging effects of oxidizing radicals, was depressed by 30 percent after 30 days of smoke exposure.

80–0894. Ingenito, A. J.; Durlacher, L. **Effects of Carbon Monoxide on the b-Wave of the Cat Electroretinogram: Comparisons With Nitrogen Hypoxia, Epinephrine, Vasodilator Drugs and Changes in Respiratory Tidal Volume.** *Journal of Pharmacology and Experimental Therapeutics* 211(3): 638-646, December 1979.

To determine whether carbon monoxide (CO) may effect preganglion cell retinal function at "environmental and smoking" levels, chloralose- anesthetized cats were made to inhale 0.05 or 0.1 percent CO in air over a 2-hour period while the dark-adapted electroretinogram (ERG) was monitored. The results were compared to those of air controls, nitrogen (N_2) hypoxia (10 percent O_2), changes in respiratory tidal volume and vasopressor responses induced by epinephrine, and vasodepressor responses induced with vasodilating drugs. The latter three interventions were intended to simulate some cardiovascular and respiratory changes encountered with CO and N_2. CO caused a decrease in the ERG b-wave amplitude proportional to the percent arterial carboxyhemoglobin (HbCO), with a significant mean decrement of 10 percent observed in the "environmental or smoking" range of HbCO (7.5 percent). Comparative degrees of nitrogen hypoxia tended, if anything, to result in an augmentation of b-wave amplitude. Present findings suggest that the N_2-induced enhancement is due mainly to hypocapnia secondary to N_2-induced hyperpnea. Since injected epinephrine had no significant effect on the b-wave, its release during CO or N_2 hypoxia probably had no significant role in their ERG effects. Several findings are consistent with the idea that the CO-induced b-wave decrement may have resulted from a direct toxic effect on the retina rather than to HbCO-mediated retinal hypoxia secondary to a decrease in arterial oxyhemoglobin. CO had only minimal effects on other parameters of the dark-adapted ERG of the cat. (Auth. Abs.)

80–0895. Kjellstrom, T.; Borg, K.; Lind, B. **Cadmium in Feces as an Estimator of Daily Cadmium Intake in Sweden.** *Environmental Research* 15(2): 242-251, April 1978.

Three consecutive daily fecal specimens from 80 volunteers in the age range of 5 to 69 years were collected. Cadmium was analyzed using conventional flame atomic absorption spectrophotometry. Average daily fecal weight among adults was about 120 g wet weight and average daily fecal cadmium amount was about 16μg for nonsmokers and 19μg for smokers. The difference between smokers and nonsmokers was statistically significant ($p<0.01$). Smokers had a tendency for higher fecal weights, 133 g as compared to 122 g for nonsmokers. If it is assumed that the average cadmium concentration in food and drinks was the same for smokers and nonsmokers, the daily fecal cadmium amount emerging from ingested cadmium would be 17.3μg for smokers. The remaining 1.8μg/day of the difference between smokers and nonsmokers would be a result of smoking itself. Within 4 days after a single ingested cadmium dose, about 90 percent of the dose appears in the feces. It was concluded that fecal cadmium could be used to estimate average cadmium intake via food. The cadmium intake varied with age in a similar way as calorie intake. (Auth. Abs. Mod.)

80–0896. Kumakura, K.; Karoum, F.; Guidotti, A.; Costa, E. **Modulation of Nicotinic Receptors by Opiate Receptor Agonists in Cultured Adrenal Chromaffin Cells.** *Nature* 283(5746): 489-492, January 31, 1980.

Morphological, physiological, and pharmacological evidence indicates that opioid peptides, which in the brain are located intraneurally, may function as neurotransmitters.

Similar evidence is not yet available for the opioid peptides that are stored in chromaffin cells of adrenal medulla and in axon terminals located in adrenal medulla and sympathetic ganglia. The present report contributes evidence suggesting that the opioid peptides which are stored in the axon terminals of the splanchnic nerves located in adrenal medulla may function as neuromodulators of the acetylcholine receptors located on chromaffin cells that are involved in catecholamine release. This functional role of the opioid peptides is supported by showing that primary cultures of chromaffin cells of bovine medulla contain opiate receptors. When these receptors are occupied by specific agonists, the number of nicotinic receptors and the amount of catecholamine released by maximal doses of nicotine are reduced. Like in other neuronal systems also in adrenal medulla, the action of opioid peptides is inhibitory. The specificity of this action is in part supported by the inability of opiate receptor agonists to reduce the Ca^{2+}-dependent release of catecholamines elicited by K^+ ions. (Auth. Abs.)

80-0897. Kurogochi, Y.; Nakashima, T.; Morita, N. **Inu ni Okeru Nicotine Taisha ni Kausuru Chiken. [An Observation of Nicotine Metabolism in the Dog.]** *Journal of Nara Medical Association* 29(4/5): 669-676, October 31, 1978, Japanese.

The metabolism of nicotine in the dog was studied using gas chromatography. Cotinine appeared in the serum 30 sec after a single dose of 100μg/kg nicotine was injected intravenously. Nornicotine was demonstrated 5 min after administration of nicotine to the same dog. Intravenous injection of 2μg/kg nicotine every 60 sec for 1 hour resulted in the appearance of many peaks in gas chromatography of the serum, including nicotine, cotinine, and nornicotine, but some parts of these peaks were presumed to be free fatty acids. It was also demonstrated that nicotine metabolites appearing in the serum at the beginning and disappearance curve of blood nicotine level after the termination of nicotine administration were almost the same as those present after a single administration of nicotine. Six compounds appeared in the urinary excretion in the 24 hours after a single dose of 200μg/kg nicotine. Nicotine, cotinine, and nornicotine were identified. These data indicate that nicotine metabolism in vivo starts immediately after administration of nicotine to the dog. (Auth. Abs.)

80-0898. Laties, V. G.; Merigan, W. H. **Behavioral Effects of Carbon Monoxide on Animals and Man.** *Annual Review of Pharmacology and Toxicology* 19: 357-392, 1979.

Behavioral effects of carbon monoxide (CO) in animals and man are discussed in this review. The most consistent CO effect observed appeared to be an attenuation of the rate of behavior emitted by the animal (e.g., wheel running, treadmill running, food and water intake). However, despite decreased response rates, the behavior that occurs during CO exposure is often similar to control performance. The review of human performance is organized into sections on vision and audition, motor behavior (tracking, coordination, and driving), time discrimination, and vigilance. Study results varied greatly.

80-0899. Lehrer, S. B.; Wilson, M. R.; Karr, R. M.; Salvaggio, J. E. **IgE Antibody Response of Smokers, Nonsmokers, and "Smoke-Sensitive" Persons to Tobacco Leaf and Smoke Antigens.** *American Review of Respiratory Disease* 121(1): 168-170, January 1980.

Serum samples from cigarette smokers, nonsmokers, and persons reporting "smoke sensitivity" were tested for IgE antibodies to tobacco leaf and smoke extracts by the radioallergosorbent test. Results indicated that none of the serum samples tested contained detectable IgE antibodies to smoke extracts. Occasionally, serum specimens from smokers or nonsmokers demonstrated reactivity to leaf antigen. The most significant reaction to leaf antigens was detected in serum from one of the seven smoke-sensitive subjects tested. These results demonstrate that smoking, nonsmoking, and clinical "smoke sensitivity" are not correlated with the presence of IgE antibodies to tobacco leaf or smoke antigen. (Auth. Abs.)

80-0900. Marshall, M. V.; McLemore, T. L.; Martin, R. R.; Jenkins, W. T.; Snodgrass, D. R.; Corson, M. A.; Arnott, M. S.; Wray, N. P.; Griffin, A. C. **Patterns of Benzo(*a*)pyrene Metabolism in Normal Human Pulmonary Alveolar Macrophages.** *Cancer Letters* 8(2): 103-109, December 1979.

Pulmonary alveolar macrophages (PAMs) obtained by bronchopulmonary lavage from six normal nonsmoking volunteers were incubated with (^3H)-benzo(*a*)pyrene to ascertain the normal metabolism and conjugation of polycyclic aromatic hydrocarbons. Through the use of a crude glucuronidase preparation, both glucuronic acid and sulfate conjugates were examined. Phenols and quinones were identified by high-pressure liquid chromatography as the principal free metabolites formed during 1 hour incubation with benzanthracene-induced PAMs. In addition, phenols and quinones were major substrates utilized by these cells for conjugation during the incubation period. The ranges of benzo(*a*)pyrene metabolites produced by PAMs from nonsmokers were compiled and the variation in production as well as detoxification of proximate carcinogenic benzo(*a*)pyrene metabolites are presented. (Auth. Abs.)

80-0901. Meade, P. D.; Yamashiro, S.; Harada, T.; Basrur, P. K. **Influence of Vitamin A on the Laryngeal Response of Hamsters Exposed to Cigarette Smoke.** *Progress in Experimental Tumor Research* 24: 320-329, 1979.

In the present study, young adult hamsters (aged 6-9 weeks) fed a vitamin A-free diet for 100 days prior to and during exposure to cigarette smoke for 6 weeks were examined to determine whether the low levels of vitamin A in the hamster would exaggerate the smoke-induced changes and favor the early development of preneoplastic lesions in the larynx. The highest incidence of squamous metaplasia of the laryngeal and glandular epithelium occurred in smoke-exposed hamsters fed a vitamin A-free diet. The frequency of such histopathological changes was highest near the area of transition from stratified squamous to ciliated pseudostratified columnar epithelium. No remarkable changes were found in the lower portion of the pharynx or in the stratified squamous epithelium of the larynx. The smoke-exposed hamsters showed lower liver and plasma vitamin A levels than the animals on the vitamin A-free diet not exposed to smoke.

80–0902. Mori, H.; Matsubara, N.; Ushimaru, Y.; Hirono, I. **Carcinogenicity Examination of Betel Nuts and Piper Betel Leaves.** *Experientia* 35(3): 384-385, March 1979.

A dry powder of betel nuts, piper betel leaves, and lime was administered to rats. Epidermal thickening was frequently observed in the upper digestive tracts of rats in groups fed the betel nut diet mixed with lime and the betel leaves diet, and a forestomach papilloma was seen in one rat given betel leaves diet. These epidermal changes were scarcely seen in rats given either betel nut or normal diet alone. (Auth. Abs.)

80–0903. Oei, T.P.S.; Singer, G.; Jefferys, D.; Lang, W.; Latiff, A. **Schedule Induced Self Injection of Nicotine, Heroin and Methadone by Naive Animals.** In: Colpaert, F. C.; Rosecrans, J. A. (Editors). *Stimulus Properties of Drugs: Ten Years of Progress.* Amsterdam, Elsevier/North Holland Biomedical Press, 1978, pp. 503-516.

Nicotine, heroin, and methadone were given to rats by schedule-induced self-injection. The rats were divided into three groups by body weight and food schedule. The rate of bar pressing for nicotine by the three groups was significantly different from that of the three groups pressing for saline, $p<0.05$. The rate of bar pressing between the three different body weight/schedule conditions was significant, $p<0.01$. The interaction between the drug effect and the body weight conditions was not significant. An analysis showed that bar pressing rates for nicotine were significantly higher under the food delivery schedule when compared to 80 percent body weight reduced rats without the schedule, $p<0.05$, and to the 100 percent body weight rats, $p<0.01$. The bar pressing was equivalent to a mean nicotine intake of 1.13 ± 0.36 mg/kg/hour for the food schedule group, 0.31 ± 0.13 mg/kg/hour for the 80 percent body weight reduced group, and 0.11 ± 0.07 mg/kg/hour for the normal body weight group. The difference between the nicotine intake for the 80 percent body weight rats with and without the food delivery schedule was significant ($p<0.01$). However, the nicotine intake of the 80 percent and 100 percent body weight rats without the food delivery schedule was not significantly different.

80–0904. Pruett, J. J.; Scheuenstuhl, H.; Michaeli, D.; Nevo, Z. **The Incorporation and Localization of Aldehydes (Highly Reactive Cigarette Smoke Components) Into Cellular Fractions of Cultured Human Lung Cells.** *Archives of Environmental Health* 35(1): 15-20, January-February 1980.

WI 38 human fetal lung fibroblasts were incubated with tracer amounts of ^{14}C-labeled formaldehyde and acetaldehyde which are highly reactive components of cigarette smoke. A pulse of 10 min with formaldehyde followed by a 60-min and 24-hour chase showed migration of ^{14}C-label into the nucleus. Fractionation of the nucleus revealed that the RNA fraction had the highest absolute and specific activity while the DNA and protein fractions contained considerably lower activities. All of the counts from formaldehyde were found in the adenine and guanine components of RNA. The DNA counts were distributed among adenine, guanine, and thymine. Incubation with ^{14}C-acetaldehyde showed a majority of the counts localized in the membrane plus nuclear lipid fraction. A small proportion of counts was found in the protein portion of the cells. (Auth. Abs.)

80–0905. Rafael'skaya, G. I. **Morfofunktsional'nyye Izmeneniya v Polovoy Zheleze Krys pri Vozbuzhdenii i Blokade M- i N-Kholinoreaktivnykh Sistem. [Morphofunctional Changes in the Sexual Gland of Rats Upon Stimulation and Blockade of the M- and N-Cholinoreactive Systems.]** *Farmakologiya i Toksikologiya* 1: 91-94, 1980, Russian.

Preparations of arecoline and nicotine cholinergic agents, benactyzine and etherofen cholinolytic agents have been administered intraperitoneally for 15 days to eight groups of six rats. Nicotine was given in daily doses of 0.5 and 1 mg/kg, the other three agents in daily doses of 1 and 5 mg/kg. Histological analyses of the animals' sexual glands indicated significant morphofunctional changes in the interstitial tissue and endocrinic and exocrinic systems of the testis. Nicotine was found to stimulate hormone production in the glandular cells. All preparations disturbed normal spermatogenesis in the rats. The observed effects were apparently caused by a complex interaction between the central and peripheral parts of the neuroendocrine system.

80–0906. Sahley, T. L.; Berntson, G. G. **Antinociceptive Effects of Central and Systemic Administrations of Nicotine in the Rat.** *Psychopharmacology* 65(3): 279-283, November 1979.

Nicotine (0.16-0.50 mg/kg, injected subcutaneously) was found to exert a potent antinociceptive action on thermal stimuli as measured by the tail-flick test. This antinociceptive action of nicotine could be blocked by centrally active nicotinic or muscarinic blockers implicating both classes of cholinergic receptors in this effect. Quaternary blockers, however, failed to prevent nicotine-induced antinociception. This finding, together with the ability of small doses of nicotine (25μg) to induce potent antinociceptive effects when administered centrally, suggests a central site of action for the antinociceptive action of nicotine. The present results also support the suggestion that nicotine may selectively reduce sensitivity to certain classes of pain stimuli, perhaps through a central releasing action on acetylcholine. (Auth. Abs.)

80–0907. Schwetz, B. A.; Smith, F. A.; Leong, B.K.J.; Staples, R. E. **Teratogenic Potential of Inhaled Carbon Monoxide in Mice and Rabbits.** *Teratology* 19(3): 385-391, June 1979.

Pregnant CF-1 mice and New Zealand rabbits were exposed to carbon monoxide at a concentration of 250 ppm for 7 or 24 hours daily during the period of major organogenesis, days 6 through 15 of gestation in mice, and 6 through 18 of gestation in rabbits. Carboxyhemoglobin levels in the range of 10-15 percent were observed in both species (control animals had 0.7 percent or less). Carbon monoxide was not found to be teratogenic in either species. In mice, a significant increase in the incidence of some minor skeletal variants was observed. One litter in each of the carbon monoxide-exposed groups of mice was completely resorbed; none of the litters of control mice or of control or exposed rabbits were completely resorbed. The fetuses of mice exposed to carbon monoxide for 7 hours daily were heavier than control fetuses, and those exposed for 24 hours daily were lighter than control fetuses. The reason for this result is not known. (Auth. Abs.)

80–0908. Smith, B. R.; Plummer, J. L.; Ball, L. M.; Bend, J. R. **Characterization of Pulmonary Arene Oxide Biotransformation Using the Perfused Rabbit Lung.** *Cancer Research* 40(1): 101-106, January 1980.

Rapid arene oxide biotransformation has been suggested as a protective mechanism against the effects of polycyclic aromatic hydrocarbons. This is a report on benzo(*a*)pyrene 4,5-oxide (BP 4,5-oxide) biotransformation by perfused rabbit lungs. BP 4,5-oxide (5 μmol) was conjugated with glutathione and hydrated at rates of 23.8 ± 2.3 (S.D.) and 7.60 ± 1.14 nmol/min/g of tissue, respectively. The total rate for BP 4,5-oxide biotransformation by perfused lungs was about 3 times the estimated rate of arene oxide formation from benzo(*a*)pyrene found in previous studies. A high pressure liquid chromatographic system was utilized to resolve the BP 4,5-oxide metabolites. Perfused rabbit lungs produced a cysteine conjugate from BP 4,5-oxide, but lacked the ability to conjugate *trans*-4,5-dihydro-4,5-dihydroxybenzo(*a*)pyrene with sulfuric or glucuronic acid at detectable rates. The *trans*-4,5-dihydro-4,5-dihydroxybenz(*a*)pyrene could pass freely from the tissue into the perfusion medium but remained at a relatively high concentration in the lungs, possibly due to the lipophilic nature of this metabolite. The glutathione conjugate, which diffused slowly from the tissue into the perfusion medium, showed no tendency to accumulate in the tissue. Benzo(*a*)pyrene 4,5-oxide-derived radioactivity was covalently bound to pulmonary DNA, RNA, and protein. The relative activities of toxication/detoxication pathways, the apparent inability of pulmonary tissue to conjugate *trans*-4,5-dihydroxy-4,5-dihydrobenzo(*a*)pyrene with sulfuric or β-glucuronic acid, the tendency of some benzo(*a*)pyrene metabolites to accumulate in the tissue, and the relationship of these parameters with the susceptibility of lung to the effects of polycyclic aromatic hydrocarbons are discussed. (Auth. Abs. Mod.)

80–0909. Sprince, H.; Parker, C. M.; Smith, G. G. **Comparison of Protection by L-Ascorbic Acid, L-Cysteine, and Adrenergic-Blocking Agents Against Acetaldehyde, Acrolein, and Formaldehyde Toxicity: Implications in Smoking.** *Agents and Actions* 9(4): 407-414, 1979.

Acrolein and formaldehyde, as well as acetaldehyde, are currently regarded as important toxicants in cigarette smoke. In this study with rats, previously reported protectants against acetaldehyde were tested for their effectivity as protectants against acrolein and formaldehyde. As with acetaldehyde, pretreatment with high oral doses of L-ascorbic acid, L-cysteine, and a combination of these protectants in reduced amounts with thiamin gave a high degree of protection against lethal doses of acrolein. Survivors after 72 hours were in the 90 percent range compared to survivors of pretreated saline controls in the 5 percent range. With equimolar doses of adrenergic-blocking agents, protection against acrolein after 72 hours was greater with an alpha-blocker, e.g., phenoxybenzamine (60 percent survivors), than with a beta-blocker, e.g., propranolol (15 percent survivors). Protection with L-ascorbic acid was dose-related and also additive with the adrenergic-blocking agents. With above protectants, protection against acrolein was as good, if not better, than against acetaldehyde. Protection against formaldehyde after 72 hours was only partial, being highest for L-ascorbic acid (55 percent survivors) compared to saline controls (5 percent survivors). Less effective against formaldehyde were L-cysteine (20 percent survivors) and phenoxybenzamine (15 percent survivors). Propranolol gave no protection against formaldehyde. It is speculated that in vivo protection by L-ascorbic acid and L-cysteine could occur directly by detoxification of the aldehyde toxicants or indirectly by normalization of the synthesis or release of tissue catecholamines abnormally altered by the aldehyde toxicants. Further animal experimentation is necessary before any extrapolation of these findings for human use can be considered. (Auth. Abs.)

80–0910. Stevens, M. H. **Synergistic Effect of Alcohol on Epidermoid Carcinogenesis in the Larynx.** *Otolaryngology and Head and Neck Surgery* 87(6): 751-756, November-December 1979.

Forty hamsters received weekly injections of benzo(*a*)pyrene into the larynx. Twenty of these also had 10 percent alcohol as their total liquid intake. Carcinoma of the larynx was seen in seven alcoholic vs. two nonalcoholic hamsters, supporting the evidence in humans that alcohol is a factor in epidermoid carcinogenesis of the larynx. (Auth. Abs.)

80–0911. Taniguchi, T.; Fujiwara, M.; Lee, J. J.; Hidaka, H. **Effect of Acetylcholine on the Norepinephrine-Induced Positive Chronotropy and Increase in Cyclic Nucleotides of Isolated Rabbit Sinoatrial Node.** *Circulation Research* 45(4): 493-504, October 1979.

Transmural stimulation of, or application of nicotine to, the isolated rabbit sinoatrial node resulted in initial negative and late positive chronotropy. Simultaneous application of acetylcholine and norepinephrine produced a similar biphasic chronotropic effect. These procedures produced an initial increase in cyclic guanosine 3′:5′-monophosphate (cyclic GMP) and a delayed elevation in cyclic adenosine 3′:5′-monophosphate (cyclic AMP). Nicotine exposure also increased nucleotide levels and heart rate. The content of cyclic GMP increased from 0.64 ± 0.09 to 1.33 ± 0.21 pmol/mg protein after a 40-sec exposure to nicotine (10^{-4} M), and the content of cyclic AMP increased from 16.8 ± 1.5 to 25.1 ± 2.7 pmol/mg protein after a 3-min exposure. The pacemaker rate decreased for the first 40 sec, then increased gradually over 5 min. However, the initial and late effects on rate and nucleotide levels were inhibited by pretreatment with atropine and propranolol, respectively. Pretreatment with atropine shortened the time of maximum increase in cyclic AMP level and heart rate from 3 to 1 min after the simultaneous application of acetylcholine and norepinephrine and enhanced the positive chronotropic effect. Physostigmine prolonged the duration of the increase in cyclic GMP and negative chronotropic effect after the simultaneous application. (Auth. Abs. Mod.)

80–0912. Van Cantfort, J.; Manil, L.; Gielen, J. E.; Glatt, H. R.; Oesch, F. **A New Assay for Glutathione *S*-Transferase Using (^{3}H)-Benzo(*a*)Pyrene 4,5-Oxide as Substrate. Inducibility by Various Chemicals in Different Rat Tissues Compared to That of Aryl Hydrocarbon Hydroxylase and Epoxide Hydratase.** *Biochemical Pharmacology* 28(4): 455-460, 1979.

A new assay for glutathione *S*-transferase using (^{3}H)-benzo(*a*)pyrene 4,5-oxide as a substrate is presented. The assay developed in this study was used to determine the effect of cigarette smoke inhalation on glutathione *S*-transferase activity in liver, kidney, and lung in rats and to compare this effect with that of three well-known drug metabolizing enzyme inducers (phenobarbital, 3-methylcholanthrene, and 16α-cyanopregnenolone) and with the effect on two other enzymes involved in the polycyclic hydrocarbon metabolism: aryl hydrocarbon hydroxylase and epoxide hydratase. Aryl hydrocarbon hydroxylase activity was enhanced by the three classical inducers in the liver, while only 3-methylcholanthrene and cigarette smoke significantly ($p < 0.001$) induced it in the lung and kidney. In the liver, epoxide hydratase activity was induced by 16α-cyanopregnenolone and especially by phenobarbital. In the other tissues, epoxide hydratase was never induced. This is very different from the induction of aryl hydrocarbon hydroxylase activity which was induced best by 3-methylcholanthrene.

80–0913. Vestal, R. E.; Wood, A.J.J.; Shand, D. G. **Reduced β-Adrenoceptor Sensitivity in the Elderly.** *Clinical Pharmacology and Therapeutics* 26(2): 181-186, August 1979.

The effect of age on sensitivity to both isoproterenol and propranolol has been investigated in 27 male volunteers (13 of whom were habitual smokers) aged 21-73 years. The dose of isoproterenol (given as a rapid intravenous injection) required to increase the resting heart rate by 25 bpm (I_{25}) increased with age. The I_{25} was repeated during an intravenous infusion of propranolol and the dose ratio (I_{25} after propranolol divided by the control I_{25}) determined. This was related to the concentration of free propranolol in plasma. It was found that the effectiveness of any given free concentration diminished progressively with age. These data are consistent with a diminished rsponsiveness of the β-adrenoceptor to both agonist and antagonist drugs with advancing years. Neither isoproterenol resistance nor propranolol resistance was affected by smoking habits. (Auth. Abs. Mod.)

80–0914. Yanaura, S.; Nishimura, T.; Hosokawa, T.; Abe, Y.; Iwase, H. **Gaiso Hansha no Gakurigaku Teki Kenkyu—Toku ni Kagaku Teki Shigeki ni Yoru Seki yo Hansha ni Tsuite. [Pharmacological Studies on the Cough-Like Reflex Induced by Chemical Stimulation.]** *Folia Pharmacologica Japonica* 74(3): 345-352, 1978. Japanese.

To investigate the mechanism of the cough-like reflex (CLR), 1,1-dimethyl-4-phenylpiperazinium iodide (DMPP) was employed. In dogs anesthetized with α-chloralose, CLR was induced by administration of DMPP (intravenous and intracarotid arterial), lobeline, and nicotine. Repeated administration of DMPP did not cause a tachphylaxis; however lobeline and nicotine did induce a tachphylaxis. DMPP (i.a.) lobeline, nicotine, and histamine caused an increase in respiratory resistance, while DMPP (i.v.) did not. The ED50 of morphine, codeine, oxymethebanol, picoperidamine, and piclobetol on CLR with DMPP was higher than that of those drugs employed for the peripherally-induced cough. CLR with DMPP was not affected by isoproterenol, ephedrine, atropine, and propranolol. CLR was markedly depressed by hexamethonium and slightly depressed by benzonatate. CLR was abolished after bilateral vagotomy or bilateral denervation of the carotid sinus nerves. These results indicate that the mechanism of CLR with DMPP is different from that of peripherally-induced cough and the carotid body chemoreceptor plays an important role in CLR with DMPP. This CLR may be useful for demonstrating the site of action and the mechanism of antitussive drugs. (Auth. Abs.)

See also, **80–1002**

MORTALITY AND MORBIDITY

80–0915. Enstrom, J. E. **Cancer Mortality Among Low-Risk Populations.** *CA-A Cancer Journal for Clinicians* 29(6): 352-361, November-December 1979.

This paper presents a brief epidemiological review of cancer mortality in the major low-risk populations in the United States. Data from Adventist, Mormon, and nonsmoker populations are examined. Average annual age-adjusted mortality rates and life expectancies for low-risk U.S. cohorts are compared with U.S. whites and Swedes, and tabulated. The following three cohorts of healthy nonsmokers had a combined average total cancer rate reduction of 39 percent relative to U.S. whites: the American Cancer Society, Mormons, and Adventists. There is a great deal of evidence pointing to the benefits of being a nonsmoker. If all Americans did not smoke, the resulting mortality reduction has been estimated to be 26 percent for lung cancer and other cancer deaths.

80–0916. Garfinkel, L. **Cardiovascular Mortality and Cigarette Smoking.** *World Smoking and Health* 4(3): 42-45, Autumn 1979.

Smoking and cardiovascular mortality are briefly discussed in terms of mortality ratios, autopsy evidence, and tar and nicotine content in cigarettes. Mortality ratios of persons aged 49-79 who smoked cigarettes only were given as follows: in men, the mortality ratio was 1.43 in those who smoked <10 cigarettes/day; 1.93 in the 10-19 cigarettes/day smokers; 2.15 in the 20-39 cigarettes/day smokers; and 2.59 in the ≥40 cigarettes/day smokers. In women, the ratios were 1.08, 1.76, and 2.29, respectively, for <10, 10-19, and ≥20 cigarettes/day smokers. Cerebrovascular disease and aortic aneurysm mortality ratios also increased in smokers.

80–0917. Hammond, E. C.; Garfinkel, L.; Lew, E. A. **Longevity, Selective Mortality, and Competitive Risks in Relation to Chemical Carcinogenesis.** *Environmental Research* 16(1/3): 153-173, 1978.

Longevity, mortality, and competitive risks were studied in relation to cigarette smoking in four groups of male smokers. All inhaled to a moderate degree. The first group started smoking before age 20. The majority of these subjects smoked 20-39 cigarettes/day. The second group began smoking at 20-24 years and smoked either ≥10 cigarettes/day or ≤9 cigarettes/day. The third group began smoking at 25-29 years and smoked either ≥20 cigarettes/day, 10-19 cigarettes/day, or less (but with greater inhalation). The fourth group started smoking at 30-34 years and smoked ≥20 cigarettes/day. Life expectancy at age 40 was calculated to be 37.85 for nonsmokers and 30.87 for smokers. The 6.98 years difference in life expectancy was due to lower death rates from cancer and other diseases (especially coronary heart disease) in nonsmokers. The mean ages at death from cancer will be 74.93 for nonsmokers and 69.39 for smokers.

80–0918. Mimica, M.; Malinar, M.; Durakovic, Z. **Uzivanje Kave u Odnosu na Bolesti u Nasoj Populaciji. [Coffee Drinking and Diseases in Our Population.]** *Acta Medica Iugoslavica* 32(1): 19-32, 1978, Serbo-Croatian.

The results of a 1972 clinical epidemiological study of general morbidity, in particular cardiovascular morbidity, are presented and analyzed in relation to coffee consumption in Croatia. In a random sample of 3,265 subjects, aged 38-57 years, 18.9 percent of men and 20.6 percent of women admitted drinking three or more cups of coffee per day. Coffee consumption data are tabulated in relation to occupation, anthropological characteristics, alcohol consumption, and drug and smoking habits. Of those who drank three or more cups of coffee per day, 50.3 percent were smokers, whereas only 23.3 percent of the non-coffee drinkers smoked. Contrary to expectation, bronchitis symptoms were as frequent in coffee drinkers as in nondrinkers. No statistically significant difference was found between coffee drinkers and nondrinkers with respect to the incidence of neurosis, gastritis, peptic ulcer, colitis, or cardiac diseases.

See also, **80–0928, 80–0931**

NEOPLASTIC DISEASES

80–0919. Blot, W. J.; Fraumeni, J. F., Jr.; Morris, L. E. **Patterns of Laryngeal Cancer in the United States. (Letter).** *Lancet* 2(8091): 674-675, September 23, 1978.

Trends for laryngeal cancer in Britain and Australia appear to be related to patterns of alcohol consumption (*Lancet* 1(8076): 1244-1247, June 10, 1978). The association of laryngeal cancer rates with demographic variables as well as alcohol and tobacco consumption was examined in the United States. Rates throughout the country increased sharply with urbanization, being nearly twice as high in highly urban as compared with rural areas. The urban gradient seems at least partly due to heavier alcohol and tobacco consumption among city dwellers. Other factors examined were socioeconomic status, ethnicity, and occupational exposure. Nationally the distribution of laryngeal cancer was correlated most strongly with lung cancer. To establish the correlation, lung cancer mortality was used as a substitute for unavailable tax data on tobacco sales. The data suggest that not only alcohol consumption but also tobacco smoking and occupational exposure affect the patterns of laryngeal cancer in the United States.

80–0920. Burch, P.R.J. **Smoking, Lung Cancer and Scientific Debate.** *Tabak Journal International* (2): 98, 100, April 1979.

Two papers on smoking and lung cancer that were given before the Royal Statistical Society are discussed. The theory that smoking causes lung cancer is examined and inconsistencies in the theory are pointed out. It is concluded that the evidence does not show how smoking causes lung cancer or, indeed, whether it does. The conclusion is based on the belief that there is a strong environmental bias in the causal theory which views disease as mainly resulting from environmental factors. The opposing view propounded here states that the cause of disease is essentially biological and genetic in nature.

80–0921. Calabrese, E. J. **Is the Role of the Environment in Carcinogenesis Overestimated?** *Medical Hypotheses* 5(1): 5-14, January 1979.

The dominant role of the physical and chemical environment in the development of cancer is challenged in this review. Analyses of the etiology of skin, bladder, respiratory, and gastric cancers are presented which demonstrate the considerable extent that health status may have in modifying the initiation and promotion of environmentally associated cancers. It has, for example, been reported in one study that there are differences in susceptibility to aromatic hydrocarbon-induced bronchogenic carcinoma. Among groups with bronchogenic carcinoma, those with intermediate and high aryl hydrocarbon hydroxylase inducibility had 16 and 36 times greater risk of such cancers than the low group—assuming a risk of 1.0 in the low group. All the patients with bronchogenic carcinoma had smoked for 20-40 years with an average cigarette consumption of about two packages/day. Although three did not smoke cigarettes, two smoked 10-15 cigars/day while the third was a heavy pipe smoker. It is concluded that although environmental factors may initiate and/or promote 85-90 percent of all cancers, this is misleading since it neglects the critical role of the individual's health status as a factor modifying carcinogenesis. (Auth. Abs. Mod.)

80–0922. Dulac, G.-L. **Le Cancer de la Langue. [Cancer of the Tongue.]** *Concours Medical* 99(25): 4149-4154, June 18, 1977, French.

The etiology of tongue cancer, particularly malpighian epithelioma, its early detection and differential diagnosis, and clinical and therapeutic procedures are detailed, based on 332 cases treated from 1961 to 1968 at the Curie Foundation in Paris. At that time, 25 percent of the cases of epithelioma of the mobile part of the tongue were in women who smoke. Smoking is cited as one of the six risk factors of this particular epithelioma, which includes chronic alcoholism as the most important contributing factor. The study indicates the importance of patient education with respect to early symptoms and diagnosis, and the inauspicious roles of alcohol and tobacco.

80–0923. Feldman, J. G.; Boxer, P. **Relationship of Drinking to Head and Neck Cancer.** *Preventive Medicine* 8(4): 507-519, 1979.

A case—control study of 185 male patients with head and neck cancer sites and 182 control males with cancer sites unrelated to either smoking or drinking investigated the possible mechanism of alcoholic carcinogenicity. Head and neck cancer sites were grouped into two categories based on their presumed likelihood of direct contact with alcohol in its liquid form. Comparisons were made of drinking and smoking patterns among each group of head and neck cancer patients and the controls. Of the patients with head and neck cancer, 19.5 percent were nonsmokers, 16.8 percent smoked <10 cigarettes daily, 24.9 percent 10-19 cigarettes daily and 38.9 percent ≥ 20 cigarettes daily; the percentages for controls were 51.1, 18.7, 16.5, and 13.7, respectively. Patients in both groups of head and neck sites smoked and drank significantly more than did the controls. Even after adjustment for alcohol use, heavy smokers (≥ 20 cigarettes/day) were at about 8 times greater risk of developing a head and neck site in either group. Overall, heavier drinkers were at only twice the risk after adjustment for tobacco use; but among those with high-likelihood contact sites, the risk was 4.5 times greater. The odds ratio of developing a head and neck cancer site was 18 times greater for the patient who was both a heavy drinker and a heavy smoker than it was for the abstainer. There was a suggestion that individuals diagnosed under age 65 who were heavier drinkers and heavier smokers were at greater risk to sites with a high likelihood of direct contact with alcohol. Average age at diagnosis among heavy smokers and heavy drinkers with sites in direct contact with alcohol was 53, or 9 years earlier than for nonheavy drinkers and nonheavy smokers. No significant differences in age at diagnosis by level of smoking and drinking were evident for patients with head and neck cancer sites with a low likelihood of direct alcohol contact. Regression analysis indicated that, for patients with high-likelihood sites, greater alcohol consumption as well as younger age at first drinking correlated with earlier age at diagnosis. In general, these data are consistent with the hypothesis that one form of alcoholic carcinogenicity may be by direct contact of alcohol and tobacco with the involved site. (Auth. Abs. Mod.)

80–0924. Flamant, R. **Epidemiological Research on the Relationship Between Tobacco, Alcohol and Cancer.** *Progress in Biochemical Pharmacology* 14: 36-46, 1978.

Epidemiologic studies on the relationship between tobacco and cancer, alcohol and cancer, and between both alcohol and tobacco and cancer are reviewed. In the case of tobacco, the studies indicate that the cancers most frequently induced by smoking are, in order of decreasing risk, those of the lung, larynx, pharynx, oral cavity, esophagus, and bladder. Studies of the relationship between cancer risk and the method of smoking disclosed that cancers of the lung, larynx, and bladder are related to cigarette smoking only and that the risk of developing lung cancer is lower in men who have smoked filter cigarettes for at least 10 years than in those who smoked plain cigarettes. Lung cancer mortality is lower in persons who start smoking at a later age, those who smoke for a shorter period of time, and those who give up smoking. A strong correlation exists between alcohol and cancer, even though no carcinogen factor has ever been found in alcohol. The combined effects of tobacco and alcohol are of particular interest, since the habits are closely related. Studies carried out on 65,000 cancer cases in France indicated that lung and bladder cancers are only related to the use of tobacco, whereas upper respiratory and alimentary cancers are almost equally related to alcohol and tobacco.

80–0925. Geddes, D. M. **The Natural History of Lung Cancer: A Review Based on Rates of Tumour Growth.** *British Journal of Diseases of the Chest* 73(1): 1-17, January 1979.

The literature on tumor growth rates was reviewed in an attempt to calculate the natural history of lung cancer. An exponential model of tumor growth, first suggested in 1956, can be validated by measurement of tumor doubling times, which can be calculated for lung tumors from serial chest radiographs. In addition to tumor doubling time, tumor size and total tumor mass at death must be known to calculate the natural history of lung cancer. If all the doubling times of lung cancer in the literature are pooled, then the time from malignant change to diagnosis is about 10 years; and following diagnosis, the average time to death is about 18 months. For ex-smokers, the risk of lung cancer remains high for some time after stopping smoking and is still twice that of life-time nonsmokers after 15 years. If the malignant change stops when smoking stops, more than 10 years are needed before all new tumors are large enough to cause death.

80–0926. Gredler, B.; Kunze, M.; Vutuc, C. **"Rauchen und Lungenkrebs"—Wissen, Einstellungen und Meinungen der Bevolkerung. [Smoking and Lung Cancer: Population Survey About Knowledge and Attitude.]** *Praxis und Klinik der Pneumologie* 34(1): 45-53, January 1980, German.

A nationwide survey of knowledge and attitudes regarding smoking and lung cancer in Austria showed a good distribution of relevant health information. Only 15 percent of all respondents don't realize the health hazards of smoking (20 percent of the current smokers compared to 5 percent of the ex-smokers). Of the respondents, 15 percent consider moderate cigarette consumption and 78 percent heavy cigarette consumption as increasing the risk of lung cancer "very much." Cigar and pipe smoking are known as being less dangerous (38 and 24 percent, respectively). Air pollution is considered a cause of lung cancer by 56 percent of the respondents. Nonsmokers are much more aware of the lung cancer risk associated with smoking than tobacco consumers. (Auth. Abs. Mod.)

80–0927. Haenszel, W.; Locke, F. B.; Segi, M. **A Case-Control Study of Large Bowel Cancer in Japan.** *Journal of the National Cancer Institute* 64(1): 17-22, January 1980.

This investigation of diet and other environmental factors reports on 588 patients with colorectal cancer and 1,176 hospitalized controls in three prefectures of Japan. Weak (not statistically significant) positive effects were found for social class and urbanization. History of smoking showed a small reduction (not significant) in bowel cancer risk among tobacco users. The significant associations of colorectal cancer with consumption of beef, string beans, or starches previously described for Hawaiian Japanese were not reproduced here. An association with hakusai (cabbage) agreed with other reports on a negative association with cruciferous vegetables. The failure to uncover important food effects in Japan is attributed to the difficulty of detecting case—control differences in areas with homogeneous diet practices. (Auth. Abs. Mod.)

80–0928. Reif, A. E. **Public Information on Smoking: An Urgent Responsibility for Cancer Research Workers.** National Cancer Institute Monograph 52, *Aspects of Cancer Research 1971-1978: Editorials from the Journal of the National Cancer Institute.* NIH Publication No. 79-1863, September 1979, pp. 123-128.

In an addendum to a previously published editorial (*Journal of the National Cancer Institute* 57(6): 1207-1210, December 1976), the author states that the incidence of cancer caused by smoking has increased, especially in women. For men, excess deaths due to cigar and pipe smoking add significantly to those caused by cigarettes. Data on cigarette, cigar, and pipe smokers; males, females, and males plus females; and excess deaths caused by smoking for 1978 are tabulated. See also 77-0358.

80–0929. United Nations International Labour Office. **Asbestos: Health Risks and Their Prevention.** United Nations International Labour Office, Meeting of Experts on the Safe

Use of Asbestos, Geneva, December 11-18, 1973, 1974, 102 pp.

A report of the Meeting of Experts on the Safe Use of Asbestos, held in Geneva in December 1973, is presented. The agenda of the meeting included (1) pathological effects of exposure to asbestos (including asbestosis and cancer), (2) prevention of risks due to exposure to asbestos, and (3) possibilities of international regulations. In analyzing the relationship of asbestos exposure to smoking as shown in various studies, it was concluded that smoking increases the risk of lung cancer, but not of mesothelioma.

80–0930. Vutuc, C.; Kunze, M. **Teer-Exposition bei tabakkonsumierenden weiblichen Bronchuskarzinompatienten und einer Vergleichspopulation. [Smoking and Lung Cancer in Women.]** *Praxis und Klinik der Pneumologie* 33(11): 1121-1124, November 1979, German.

In a retrospective case—control study, 60 percent (108) of 180 women with lung cancer were cigarette smokers as compared with only 21 percent (75) smokers in a control group of 360 female patients ($p<0.001$). The former group had been smoking for an average of 40 years, with a preference for high-tar cigarettes in 78 percent of the cases. The women in the control group had started smoking about 33 years ago and 57 percent preferred high-tar cigarettes. The differences were significant ($p<0.01$ and $p<0.001$, respectively). The exposure pattern of male and female lung cancer patients was markedly the same. (Auth. Abs. Mod.)

80–0931. Wagoner, J. K.; Infante, P. F.; Bayliss, D. L. **Beryllium: An Etiologic Agent in the Induction of Lung Cancer, Nonneoplastic Respiratory Disease, and Heart Disease Among Industrially Exposed Workers.** *Environmental Research* 21(1): 15-34, February 1980.

On the basis of the clear demonstration of the carcinogenicity of beryllium in several animal species along with the suggestion of an increased risk of lung cancer mortality in humans exposed to beryllium, an epidemiologic study of workers exposed to beryllium at one production facility was undertaken. Within the limitations imposed by the selection of data for calculation of cause-specific expected mortality, the study demonstrated a statistically significant increased risk of respiratory disease (neoplastic and nonneoplastic) and of heart disease mortality. The excessive risk of lung cancer mortality could not be related to an effect of age, chance, self-selection, study group selection, exposure to other agents in the study facility, or place of residence. On the basis of the frequency of cigarette smoking among those cohort members employed in 1967-1968 and the distribution of histologic types of lung cancer among deceased cohort members, it seems unlikely that cigarette smoking per se could have accounted for the increased risk of lung cancer among beryllium-exposed workers in the study cohort. Whereas a lower percentage of beryllium-exposed workers were current cigarette smokers (50.4 vs. 54.7 percent), a higher percentage of current cigarette smokers (21.4 vs. 15.3 percent) smoked more than one pack of cigarettes daily. In contrast, however, a higher percentage of beryllium-exposed workers never smoked cigarettes (27.2 vs. 24.7 percent). The findings of a statistically significant excess of lung cancer mortality among cohort members in general ($p<0.05$) and among workers observed 25 or more years since onset of beryllium exposure in particular ($p<0.01$), when taken in context with the results of earlier animal bioassay and recent epidemiologic studies, are supportive of the hypothesis that beryllium is carcinogenic to man. (Auth. Abs. Mod.)

80–0932. Whitaker, C. J.; Lee, W. R.; Downes, J. E. **Squamous Cell Skin Cancer in the North-West of England, 1967-69, and Its Relation to Occupation.** *British Journal of Industrial Medicine* 36(1): 43-51, February 1979.

During the three years 1967-69, 781 cases of squamous cell carcinoma of the skin were reported to the Manchester Regional Cancer Registry. The proportions of males to females were significantly different ($p<0.001$) among the skin cancer sites. The age-specific incidence rates were significantly different ($p<0.001$) between the sexes for the 5-year age groups of 55 years and above. Full occupational histories were obtained on 598 (77 percent) patients; a further 148 (19 percent) patients gave one main occupation only, while the remaining 35 (4 percent) patients were untraced. The occupations included farmers; textile, chemical, paper, and metal workers; and fishermen. Of the 610 patients whose smoking habits were known, 56 were pipe smokers (55 males, 1 female), 224 were nonsmokers (35 males, 189 females), and 330 were cigarette smokers (272 males, 58 females). For the males there was no significant difference between the ratio of pipe smokers to nonsmokers in each site ($0.3<p<0.5$). For both sexes, a test was performed on the ratio of cigarette smokers to nonsmokers in each site; no significant differences were found for the males ($0.3<p<0.5$) or for the females ($0.8<p<0.9$). There were also no significant associations between the skin site and either eye color or residence in the tropics for both male and female patients. (Auth. Abs. Mod.)

80–0933. Whitaker, C. J.; Moss, E.; Lee, W. R.; Cunliffe, S. **Oral and Pharyngeal Cancer in the North-West and West Yorkshire Regions of England, and Occupation.** *British Journal of Industrial Medicine* 36(4): 292-298, November 1979.

Patients with oral or pharyngeal cancer in the two main textile regions of England were matched for age and sex with patients having cancers not known to be associated with textile work. Data were recorded on age, sex, cancer site, and smoking, chewing, and drinking habits, together with dental and occupational history. There were 102 and 61 matched pairs of males and 52 and 60 matched pairs of females in the Northwest and West Yorkshire regions, respectively. There were significantly ($p<0.05$) more textile workers in the cases compared with their matched controls for only the females in the Northwest. No particular type of textile work occurred more frequently for the cases than the controls in all four matched comparisons. Only for the males in the Northwest were there significant differences ($p<0.05$) in the proportions of textile workers in the three cancer sites of the tongue, mouth, and pharynx. The results of the smoking and drinking habits of the patients in the four studies are inconsistent: while the males with oral and pharyngeal cancer in the Northwest smoked and drank significantly more ($p<0.02$) than their controls, the males in West Yorkshire did not show any significant differences. (Auth. Abs. Mod.)

80–0934. Wolf, G. T.; Chretien, P. B.; Elias, E. G.; Makuch, R. W.; Baskies, A. M.; Spiegel, H. E.; Weiss, J. F. **Serum Glycoproteins in Head and Neck Squamous Carcinoma. Correlations With Tumor Extent, Clinical Tumor Stage, and T-Cell Levels During Chemotherapy.** *American Journal of Surgery* 138: 489-500, October 1979.

Glycoprotein levels were measured in 90 untreated patients with head and neck squamous carcinoma, 51 cured patients, 11 patients with recurrent tumor, and 20 patients who were receiving preoperative immunosuppressive chemotherapy to determine the relation of glycoproteins to parameters of tumor extent and cellular immunity. Since a history of chronic smoking is almost universally reported in patients with head and neck squamous carcinoma, chronic cigarette smokers were used as controls in this study. Serum levels of the 6 proteins studied were compared in 139 healthy cigarette smokers, and in 61 nonsmokers to underline the importance of using smokers as controls. Levels of alpha$_1$-antitrypsin were significantly increased ($p<0.001$), and levels of prealbumin significantly decreased ($p<0.01$) in chronic smokers, when compared with nonsmokers. Haptoglobin levels were significantly increased in patients with stage I through IV tumors, but levels were similar for each stage. Serum levels of α_1-antitrypsin and α_1-acid glycoprotein increased progressively with increasing tumor stage. Serum α_2HS-glycoprotein, prealbumin, and albumin levels were decreased in all patients. In cured patients, haptoglobin and α_1-acid glycoprotein levels were significantly lower and α_2HS-glycoprotein and prealbumin levels significantly higher than in untreated patients. Protein levels in patients with recurrent tumors were similar to levels in untreated patients. The data show that α_1-antitrypsin and α_1-acid glycoprotein correlate better with tumor stage than with the cellular immunity parameters previously studied, and that α_2HS-glycoprotein levels may be as useful as T-cell levels for monitoring immune reactivity in patients with squamous cancer of the head and neck.

80–0935. Wynder, E. L. **Lung Cancer in Shipyard Workers. A Plea for Prevention. (Letter).** *New England Journal of Medicine* 299(12): 651-652, September 21, 1978.

Decades ago it was established that risk of lung cancer was causatively associated not only with the independent use of tobacco products but also with the possible synergistic effect of cigarette smoking and exposure to certain industrial factors. A recent paper (*New England Journal of Medicine* 299(12): 620-624, September 21, 1978) presents additional evidence for such an effect of cigarette smoking and asbestos exposure, or some other industrial carcinogen to which former shipyard workers in Georgia were exposed. It is clearly time to call for physicians to become more involved in occupational medicine and to be more concerned about their patients' lifestyles. The ultimate goal of medical practice, after all, should be to help society pass through life free of disease. Such a goal should be possible in terms of lung cancer and other diseases related to tobacco and occupation. What is required is a great national effort in the practice of preventive medicine. See also 78-1322.

See also, **80–0883**

NON—NEOPLASTIC RESPIRATORY DISEASES

80–0936. Babbott, F. L., Jr.; Gump, D. W.; Sylwester, D. L.; MacPherson, B. V.; Holly, R. C. **Respiratory Symptoms and Lung Function in a Sample of Vermont Dairymen and Industrial Workers.** *American Journal of Public Health* 70(3): 241-245, March 1980.

This study reviews the respiratory status of a sample of Vermont male dairy farmers and a comparison group from industry, matched for age, sex, and smoking. Survey instruments included a standardized questionnaire and simple pulmonary function tests. In general, past and present smokers had more respiratory symptoms than never-smokers; and farmers, in all smoking categories, reported symptoms with greater frequency than did their counterparts from industry. Forced vital capacity (FVC) tended to be lower among men with a history of smoking, but within each smoking category, dairymen and factory workers had very similar FVCs. Farmers who had never smoked or who were current cigarette users had lower FEV$_1$/FVC (forced expiratory volume at 1 sec/forced vital capacity) ratios than their controls from industry. Although sample sizes were limited, dairymen from small farms tended to be older, and have more respiratory symptoms, less satisfactory pulmonary function, and more serologic evidence of exposure to farmer's lung antigens than their counterparts from large farms. (Auth. Abs. Mod.)

80–0937. Becklake, M. R.; Liddell, F.D.K.; Manfreda, J.; McDonald, J. C. **Radiological Changes After Withdrawal From Asbestos Exposure.** *British Journal of Industrial Medicine* 36(1): 23-28, February 1979.

A group of 135 ex-employees of the Quebec chrysotile production industry comprising men who were born between 1901 and 1920, who had left the industry between 1950 and 1961 and had been radiographed before leaving, attended in 1972 for a medical examination and further chest radiograph. This report concerns 86 men (60 smokers, 17 ex-smokers, and 9 nonsmokers) whose withdrawal film was within 12 months of leaving and who had not been exposed subsequently to asbestos or other fibrogenic dust. Seven separate assessments were made of the paired radiographs, side by side in known temporal order. The assessments were classified as showing "increase" or "no change." In 66 film-pairs from men with 2 years' employment or longer, at least 4 assessments were of parenchymal increase in 6, of pleural increase in 13, and of both parenchymal and pleural increase in 2, total 21 (or 31 percent). There was similar agreement of no change in 24 pairs (36 percent), and evidence was equivocal in the remaining 21 pairs. Parenchymal increase was not agreed in any of the 20 film-pairs from men with shorter employment, but pleural increase was seen in four of these men, a proportion similar to that in those exposed longer. It is concluded that the parenchymal changes observed after leaving the industry, most of which were "attacks" rather than "progression," were attributable to the earlier occupational exposure to chrysotile; there were no important differences in age or smoking habit between those with and without parenchymal change. (Auth. Abs. Mod.)

80–0938. Buist, S.; Nagy, J. **A Longitudinal Study of Smokers and Nonsmokers. 5-6 Year Follow-up Using Spirometry and the Single Breath N_2 Test.** *Chest* 77(2): 259, February 1980.

Three hundred forty-two smokers and nonsmokers, first tested in 1971/72, were followed up for 5 to 6 years using a respiratory symptom questionnaire, spirometric tests, and the single breath nitrogen (N_2) test. Since the major objective of this study was to evaluate the role of the single-breath N_2 test in identifying the smoker most likely to progress to chronic airflow obstruction, only subjects with normal forced expiratory volume in 1 sec (FEV_1) were included. Lung aging, as measured by spirometric variables, was very slow in the nonsmokers. The fastest rate of decline in function was seen in male smokers who initially had an abnormal single-breath N_2 test. Nine male smokers (7 percent of all male smokers and 10 percent of male smokers with an abnormal single-breath N_2 test) developed an abnormal FEV_1 during the course of the study; all had had an abnormal single-breath N_2 test at the start of the study. It is concluded that men are more at risk of developing airflow obstruction than are women and that the single- breath N_2 test is usually abnormal for several years before the FEV_1 becomes abnormal.

80–0939. Dunnill, M. S. **Aetiology of Emphysema.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(5): 1015-1029, 1979.

There is now overwhelming evidence that lung destruction resulting in emphysema is largely due to enzymatic action on pulmonary connective tissues, mainly elastin. These enzymes are probably derived from neutrophil polymorphonuclear leukocytes and alveolar macrophages. Arguments are advanced to show that release of these enzymes is related to cigarette smoking. This explanation for the pathogenesis of emphysema is compatible with the distribution of the disease noted in epidemiological studies. In both males and females, a striking difference in both incidence and severity of emphysema has been observed in smokers as compared with nonsmokers. In smokers emphysema occurs much earlier and the severity of disease increases markedly with age, probably due to longer exposure to tobacco. If this conclusion with regard to the role of cigarette smoking is correct it must follow that apart from patients with α_1-antitrypsin deficiency, emphysema is a disease which could be almost entirely eliminated if the smoking habit was abandoned. (Auth. Abs. Mod.)

80–0940. Hepper, N.G.G.; Drage, C. W.; Davies, S. F.; Rupp, W. M.; LaMothe, J.; Schoenfelder, P. G.; Munson, P. **Chronic Obstructive Pulmonary Disease: A Community-Oriented Program Including Professional Education and Screening by a Voluntary Health Agency.** *American Review of Respiratory Disease* 121(1): 97-104, January 1980.

A community program for chronic obstructive pulmonary disease (COPD) that included detection by screening, professional education, community involvement, and evaluation of results is described. Key figures in the success of this program were senior medical students. In the first community, in which no professional education was offered, 45.3 percent of persons screened who had abnormal test results came away from their physicians understanding that they did not have a significant health problem. In subsequent communities, in which professional education was a part of the program, this percentage decreased steadily to approximately 11 percent. Consistently, 25 percent of the persons screened who had abnormal results did not consult a physician. Of the participants with abnormal screening results and no previous knowledge of COPD, 21 percent stopped smoking in the ensuing 2 to 3 years, whereas only 11.7 percent of the control group of persons with normal screening results did so ($p<0.01$). Persons with abnormal test results and previous knowledge of COPD stopped smoking at approximately the same rate as did the control group. (Auth. Abs. Mod.)

80-0941. Hills, E. A.; Davies, S.; Geary, M. **Frequency Dependence of Dynamic Compliance in Patients With Rheumatoid Arthritis.** *Thorax* 34(6): 755-761, December 1979.

An investigation of lung function was carried out in 99 randomly selected patients (37 men, 62 women) with classic or definite rheumatoid arthritis and in 60 control subjects matched for age, sex, and smoking habits. Among the male patients, there were 35 smokers and 2 nonsmokers, among the female patients, 28 smokers and 34 nonsmokers. There was a slight excess of heavy (>20 a day) cigarette smokers in the control population. Mean forced expiratory volume in 1 sec (FEV_1) and mean vital capacity (VC) were both significantly lower in the rheumatoid patients due to significant differences for women, but mean FEV/VC ratio was 73.0 percent in the rheumatoid group and 72.1 percent in the controls. Steady-state transfer factor was significantly greater in the normal men than in the comparable rheumatoid group, but there was no difference for women. After excluding patients with FEV_1 less than 80 percent predicted normal and patients with low compliance, 72 rheumatoid patients and 45 controls provided series of tracings that could be assessed for a fall in dynamic compliance (Cdyn) of 20 percent between 20 cycles and 60 cycles a minute. Dynamic compliance was not significantly different in any group at any rate of respiration. Fourteen of 72 rheumatoid patients and three of 45 controls showed frequency dependence. After the age of 50 the prevalence in the rheumatoid group was 11/38 and in the control group 2/27. This difference was significant ($p<0.05$). Frequency dependence was not consistently related to other abnormalities of lung function or to the duration, severity, or treatment of the rheumatoid arthritis. In various categories of smoking habits, dust exposure, or allergic tendency, the prevalence was always greater in the rheumatoid group. This provides evidence of patchy involvement of small airways, or alveoli and connective tissue, by the rheumatoid process. (Auth. Abs. Mod.)

80-0942. Hodous, T. K. **The Use of Helium-Oxygen Spirometry in Occupational Lung Disease.** *Journal of Environmental Pathology and Toxicology* 2(5): 23-30, May-June 1979.

Several new tests have been devised in the last decade to study functional impairment in small airways. Helium-oxygen spirometry, one such test, is safe, convenient, and can easily be added to conventional air spirometry apparatus. Two studies using helium-oxygen and air spirometry are presented. In the first, nonexposed controls were compared to flax workers with either low or high dust exposure. Both air and helium-oxygen flow volume curves showed decreased flow rates after a work-shift in the high exposure group but no change in controls. In the low dust exposure group, only helium-oxygen spirometry detected decreased post-shift flow rates suggesting small airways disease. Coal miners were compared to controls in the second study. Subgroups of younger subjects, smokers, nonsmokers, bronchitics, and nonbronchitics were also analyzed. In no case did helium-oxygen spirometry detect differences which were not present on routine air spirometry. (Auth. Abs. Mod.)

80-0943. McLarty, J. W.; Greenberg, S. D.; Hurst, G. A.; Spivey, C. G.; Seitzman, L. H.; Hieger, L. R.; Farley, M. L.; Mabry, L. C. **The Clinical Significance of Ferruginous Bodies in Sputa.** *Journal of Occupational Medicine* 22(2): 92-96, February 1980.

The clinical significance of ferruginous bodies in sputa was examined in a study of 674 former asbestos workers. Data from occupational histories and smoking behavior questionnaires, chest radiographs, spirometric measurements, and counts of ferruginous bodies were obtained as part of a 5-year surveillance program. Statistical analysis demonstrated that ferruginous bodies found in the sputa were significantly related to radiographic findings of interstitial pulmonary disease and pleural fibrosis and to spirometric findings of restrictive lung disease. Age and cigarette smoking were also found to be related to the presence of ferruginous bodies ($p<0.02$). (Auth. Abs.)

80-0944. Mittman, C. **Antitrypsin Deficiency: A Model to Illustrate the Pathogenesis of Emphysema.** *Nippon Kyobu Shikkan Gakkai Zasshi* 16(6): 369-382, June 1978.

The present status of knowledge regarding the relationship between α_1-antitrypsin deficiency and lung disease is reviewed here using data from various studies. In one study, antitrypsin-deficient smokers showed a significant loss of elastic recoil, and larger residual volumes and closing capacities than nonsmoking patients. In another study, changes in lung function were calculated from multiple regression equations. Flow rates changed differently in deficient and control smokers; no differences were seen in nonsmokers. For example, in a 40-year-old, 40-pack/year smoker, the maximal midexpiratory flow rate tended to improve with time in PiM subjects, while it fell with time in PiMZ subjects. Forced expiratory volume in 1 sec fell with time in all smokers, but the rate of decline was three times higher in the PiMZ subjects. The role of heredity (family history) in development of obstructive lung disease requires further investigation.

80-0945. Olivi, H.; Ferretti, R.; Lisboa, C. **Alteracion Progresiva de la Unidad Bronquiolo Alveolar en Fumadores. [Progressive Abnormalities of the Bronchiole-Alveolar Unit in Asymptomatic Cigarette Smokers.]** *Revista Medica de Chile* 106(8): 591-594, August 1978, Spanish.

The modified single-breath nitrogen test was performed in 40 asymptomatic smokers 16 to 40 years old. They were divided in three groups according to their smoking habit and were compared with 41 nonsmokers of similar age. An abnormal nitrogen gradient was found in smokers from 2.38 pack-years on, the changes being greater with increasing age and/or heavier smoking habit. An individual susceptibility to cigarette smoke can explain why the same degree of altered gradient may exist with very different smoking habits. Those more susceptible should be considered as high risk smokers. The pack-year index correlates best with abnormalities of the gradient. (Auth. Abs.)

80–0946. Perrault, J. L.; Malo, J.-L.; Bake, B.; Renzi, G.; Grassino, A. **Alpha$_1$-Antitrypsin Deficiency: Genetic, Clinical and Functional Correlations in a Three Generation Family.** *Respiration* 37(5): 291-300, 1979.

Alpha$_1$-antitrypsin (α_1-AT) deficiency is a hereditary condition transmitted as an autosomal codominant trait. Fully deficient homozygotes develop chronic obstructive pulmonary disease at an early age. A study of 12 α_1-AT-deficient members of a family spanning three generations by extensive pulmonary function tests, clinical history, and genetic determinations was made. Of the subjects, three were homozygotes who showed important clinical and lung function abnormalities by the age of 30. Two out of nine heterozygotes, a female and a male, had decreased dynamic compliance, elevated closing volume, and low partial pressure of oxygen in arterial blood. Clinical and physiological abnormalities (elevation in functional residual capacity and residual volume, low forced expiratory volume in 1 sec to forced vital capacity ratios, and elevated closing volume) were found in three subjects. However, these alterations could be attributed to their smoking habits (six had smoked for 3-25 years) or concomitant medical condition. The remaining four subjects were normal. None of the heterozygotes showed alterations in the static elastic recoil of the lung or static compliance. (Auth. Abs. Mod.)

80–0947. Pleuvry, B. J.; Maddison, S. E. **A Sex Difference in the Effects of Oral Codeine and Promethazine on the Ventilatory Response to Carbon Dioxide in Human Volunteers.** *British Journal of Clinical Pharmacology* 9(2): 159-164, February 1980.

Interactions between codeine and promethazine were studied in five male and five female volunteers (six of whom were smokers) measuring ventilation, end tidal carbon dioxide (CO_2) pressure, ventilatory response to CO_2, pulse, and blood pressure. The effects of codeine on the above measurements were compared in habitual smokers and nonsmokers Codeine displaced the ventilatory response to CO_2 to the right and increased end tidal CO_2 pressure in female subjects but not in males. Promethazine plus codeine displaced the ventilatory response to CO_2 to the right in male subjects, but tended to reduce the response to codeine in females. The slope of the ventilatory response to CO_2 was significantly steeper in smokers, and codeine caused significant reductions in slope in these subjects ($p<0.05$ in both cases). (Auth. Abs. Mod.)

80–0948. Pratt, P. C.; Vollmer, R. T.; Miller, J. A. **Prevalence and Severity of Morphologic Emphysema and Bronchitis in Non-Textile and Cotton-Textile Workers.** *Chest* 77: 323-325, February 2, 1980.

This study has examined both the prevalence and extent of centrilobular emphysema (CLE) and airway mucus secretory activity in a randomly collected series of inflation-fixed lungs obtained at autopsy from 44 cotton industry workers and 521 noncotton workers. Extent was measured by point counting. Using hospital medical records the cases have been divided into nonsmokers (105), cigarette smokers (427), and pipe-cigar smokers (33). Statistical analysis involved t-tests and the nonparametric Wilcoxon rank order test. Statistically significant differences in both prevalence and severity (extent) of CLE were demonstrable by both tests between nonsmokers and both cigarette and pipe-cigar smokers and even between cigarette and pipe-cigar smokers. No significant differences in either prevalence or severity of CLE were found between cotton and noncotton workers, either in toto or within any of the smoker groups. Results were similar with respect to mucus secretory cells in major bronchi and airways except for a possible excess of these cells in nonsmoking cotton workers, significant by t-test only. Pulmonary pigmentation was also examined by point counting in the nonemphysematous lungs. Again, significant differences were found between smokers and nonsmokers but not between cotton and noncotton workers.

80–0949. Pride, N. B.; Tattersall, S. F.; Benson, M. K.; Hunter, D.; Mansell, A.; Fletcher, C. M.; Peto, R. **Peripheral Lung Function and Spirometry in Male Smokers and Exsmokers.** *Chest* 77: 289-290, February 2, 1980.

Male smokers and nonsmokers were restudied after a 5- or 6-year period. Although initially there were large differences in mean values of closing volume (CV)/vital capacity (VC) between smokers and nonsmokers (smokers 21.0 percent, nonsmokers 10.0 percent), these differences had not widened (smokers 22.4 percent, nonsmokers 11.9 percent) even though cigarette consumption in the smokers continued at an average of 26 cigarettes/day and their forced expiratory volume in 1 sec (FEV_1) fell by an average of 30 ml/year. In another study, although some abnormalities in lung function were present at the onset of the study, the only significant difference at 4 years between the ex-smokers and the control subjects (whose smoking habits did not change) was in the slope of the single-breath nitrogen curve. The nitrogen slope declined in the smokers who quit within the first months of stopping and no further improvement took place over the subsequent years. No improvement was found in spirometric tests or maximum expiratory flow, although there was a suggestion that decline in FEV_1 slowed after smoking cessation. No improvement in CV/VC percent (measured by the single-breath nitrogen method in this study) was detected.

80-0950. Sackner, M. A.; Yergin, B. M.; Brito, M.; Januszkiewicz, A. **Effect of Adrenergic Agonists on Tracheal Mucous Velocity.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(3): 505-511, 1979.

Aerosolized fenoterol (Berotec) and subcutaneously administered terbutaline produced a modest increase of tracheal mucous velocity in conscious sheep. Aerosolized fenoterol had a similar effect in seven normal nonsmokers, but did not increase tracheal mucous velocity in eight patients with chronic bronchitis (two smokers and six ex-smokers) who had markedly slowed or absent mucous transport. The extent of impairment of the mucous transport system may limit the effectiveness of adrenergic agonists in promoting an increase in mucous transport. (Auth. Abs. Mod.)

80-0951. Schaanning, J.; Wessel-Aas, T. **Respiratorisk Funksjon hos Feiere. [The Respiratory Function of Chimney Sweeps.]** *Tidsskrift for Den Norske Laegeforening* (5): 240-241, February 20, 1977, Norwegian.

With oil heating of housing becoming increasingly common, it is important to clarify how much the lung function of chimney sweeps is affected and whether possible harmful effects become more pronounced with long-term exposure. In this study, 18 chimney sweeps were compared with controls matched for age and smoking habit. The average tobacco consumption of the two groups was relatively high, 12.5 cigarettes/day for the chimney sweeps and 13.8 cigarettes/day for the controls. The chimney sweeps had been smoking for an average of 14.6 years and the controls for an average of 15.3 years. The average ages were 34.4 and 33.2 for the chimney sweeps and controls, respectively. Respiratory and lung function tests were performed. The results of these tests were similar for both groups.

80-0952. Speizer, F. E.; Ferris, B., Jr.; Bishop, Y.M.M.; Spengler, J. **Respiratory Disease Rates and Pulmonary Function in Children Associated With NO_2 Exposure.** *American Review of Respiratory Disease* 121(1): 3-10, January 1980.

As part of a long-range, prospective study of the health effects of air pollution, approximately 8,000 children from 6 to 10 years of age from six communities had questionnaires completed by their parents and had simple spirometry performed in school. Comparisons were made between children living in homes with gas stoves and those living in homes with electric stoves. Children from households with gas stoves had a greater history of respiratory illness before age 2 (average difference, 32.5/1,000 children) and small but significantly lower levels of forced expiratory volume in 1 sec, and forced vital capacity corrected for height (average difference, 16 and 18 ml, respectively). These findings were not explained by differences in social class or by parental smoking habits. Measurements taken in the homes for 24-hour periods showed that nitrogen dioxide levels were four to seven times higher in homes with gas stoves than in homes with electric stoves. However, these 24-hour measurements were generally well below the current federal 24-hour outdoor standard of 100 $\mu g/m^3$. Short-term peak exposures, which were in excess of 1,100 $\mu g/m^3$, regularly occurred in kitchens. Further work will be required to determine the importance of these short-term peaks in explaining the effects noted. (Auth. Abs.)

80-0953. Tockman, M. S. **Follow-Up Study of Pulmonary Function in Nonsmokers.** Doctoral Dissertation, The Johns Hopkins University, Baltimore, University Microfilms 79-24643, 1979, 650 pp.

Five hundred forty-two mobile white male nonsmokers aged 35-64, residing in an area of low air pollution, were tested by spirometry and respiratory symptom questionnaires in 1965. In a followup study of this population begun in 1973, retesting of 399 (73.6 percent) was accomplished. An additional 195 nonmobile white male nonsmokers from the same Hagerstown, Maryland, community were also tested in 1973. This stable, rural population of nonsmoking males provided an ideal opportunity to examine the unconfounded natural history of pulmonary obstruction both cross-sectionally as a function of age and longitudinally over time. Differences in mortality and rates of ventilatory function deterioration were observed and related to spirometry and questionnaire histories of respiratory symptoms, respiratory illness, medical illness, and dust/fume exposure obtained at the initial survey. The majority of nonsmokers lost forced expiratory volume in 1 sec (FEV_1) at the rate of 25 cc/year independent of age, height, or respiratory symptoms. In the pattern of a decay curve, however, upon reaching a sufficiently low FEV_1 (2-survey mean $FEV_1 < 2.50$ l), a significantly more rapid rate of ventilatory deterioration ensues (34 cc/year). Individuals with 1-sec forced expiratory volumes below this critical value are more likely to demonstrate accelerated ventilatory deterioration in the presence of bronchitic symptoms. Additionally, individuals with values of FEV_1 (expressed as a percent of predicted) in the lowest 10 percent of the population (less than 69.4 percent) had a threefold greater risk of death in the interval. A history of asthma was also associated with significant risks for accelerated ventilatory decline and mortality. (Auth. Abs. Mod.)

80-0954. Warshaw, R.; Fischbein, A.; Thornton, J.; Miller, A.; Selikoff, I. J. **Decrease in Vital Capacity in PCB-Exposed Workers in a Capacitor Manufacturing Facility.** *Annals of the New York Academy of Sciences* 320: 277-283, 1979.

Pulmonary function was evaluated in 243 workers exposed to polychlorinated biphenyls (PCBs) in the manufacture of capacitors. Mean employment was > 15 years. Thirty-four of the workers (14 percent) were found to have a reduced forced vital capacity (FVC<80 percent of Morris' predicted); 24 of these workers were smokers or ex-smokers and 7 were nonsmokers. Of the 34 with reduced FVC, 27 (80 percent) demonstrated a restrictive pattern of impairment (forced expiratory volume in 1 sec/FVC$>$0.7); 20 of these workers were either current or ex-smokers. Only one of these 27 workers had an abnormal chest roentgenogram. Restrictive spirometric impairment with no radiographic change is unusual in occupational exposure. (Auth. Abs. Mod.)

80–0955. Worden, J. K.; Waller, J. A.; Ashikaga, T.; Sweeney, R. R. **Respiratory Disease in Vermont: A Population Survey for Planning a Public Education Program.** *Preventive Medicine* 9(1): 120-134, January 1980.

A survey of knowledge, attitudes, and behavior relating to lung disease was conducted in Vermont on a sample of 1,553 residents. Results are presented according to the following risk groups: those at high risk of lung disease because of advanced age (35 and older) and/or smoking; and those who already experience lung disease symptoms, both smokers and nonsmokers. Data were interpreted using Berlo's SMCR (source, message, channel, receiver) model so that results could be used directly in planning a mass media educational program of primary and secondary prevention. Several knowledge gaps were identified suitable for educational efforts aimed at the specific audiences. It was found that smokers regarded the effects of cigarette smoke as less harmful than nonsmokers ($p<0.001$), and many smokers expressed ignorance about the effects of sidestream smoke. Smokers of both sexes and age groups felt that the harm to unborn children of the mother who smokes was relatively low. Other conclusions were (a) smokers were aware of many antismoking messages, but need motivation to accept such messages; (b) persons with lung disease symptoms were reluctant to seek medical help; (c) health education messages should contain a strong call to specific action, since persons were found to have good intentions regarding respiratory health but seldom carry them out; and (d) certain mass media are likely to be more effective than others in reaching specific high-risk or symptomatic target subpopulations. (Auth. Abs. Mod.)

80–0956. Yildirim, S.; Akgun, N. **Nargile Icenlerde Akciger Fonksiyonlari. [Effects of Water-Pipe Smoking on Lung Function Tests.]** *Ege Universitesi Tip Fakultesi Dergisi Cilt* 14(4): 929-941, 1975, Turkish.

Vital capacity, forced expiratory volume in 1 sec, expiratory reserve volume, functional residual capacity, residual volume, total lung capacity, and maximum breathing capacity were measured in 17 water-pipe smokers and the results were compared statistically with those of 21 control cases. There was found to be no significant difference in the results, and it is concluded that water-pipe smoking is not as harmful as cigarette smoking because when the smoke passes through the water it may deposit some noxious matter there. (Auth. Abs.)

80–0957. Zimmermann, H.; Braendli, O. **Der Verlauf der chronischen obstruktiven Lungenkrankheit unter Therapie. [Therapeutic Course of Chronic Obstructive Lung Disease.]** *Schweizerische Medizinische Wochenschrift* 110(6): 232-236, February 9, 1980, German.

The effects of changes in smoking habit and conventional therapy (corticosteroids and inhalation therapy) on the course of chronic obstructive lung disease were observed over a 3-year period in a group of 71 hospitalized patients, 55 of whom were smokers. Vital lung functions except the forced expiratory volume in 1 sec (FEV$_1$) did not change significantly over the period of observation. The average annual FEV$_1$ decreased by 68 ml/year from the initial value of 1.28 l. Smokers of more than 3 cigarettes/day showed a better, although statistically insignificant, average vital capacity (VC) than smokers of only 1 to 3 cigarettes/day or nonsmokers (initially 3.41 l vs. 3.03 or 2.99 l, respectively). The latter group of smokers (over one-half of all smokers) had an initial capillary carbon dioxide partial pressure (pCO$_2$) at the threshold of respiratory decompensation (41.83 mmHg). After 3 years of therapy, VC decreased to 3.10 l in smokers of more than 3 cigarettes/day, and to 2.62 and 2.70 l in smokers of 1 to 3 cigarettes/day and nonsmokers. The average pCO$_2$ in smokers of 1-3 cigarettes/day increased to 43.25 l after 3 years. Thus, the study confirmed progressive deterioration of the functional parameters in patients under conventional therapy and showed little effect of nicotine withdrawal on the long-term course of chronic obstructive lung disease. Patients who continued to smoke more than 3 cigarettes/day during treatment exhibited better initial and comparable subsequent functional parameters than the other patients. Patients who continued to smoke 1-3 cigarettes/day had the worst initial and subsequent values.

CARDIOVASCULAR DISEASES

80–0958. Adey, H.; Ballantyne, D. A.; Bostock, M. I.; Fleischl, P.; Kurta, A. V.; Langley, R. B.; McPherson, I. G.; Turner, A. S. **The Prevalence of Disturbance of Cardiac Rhythm in Healthy New Zealand Adults: A Pilot Study.** *New Zealand Medical Journal* 88(625): 433-435, December 13, 1978.

Continuous portable 24-hour tape-recorded monitoring of the electrocardiogram in 189 healthy adults (130 males and 59 females) was used to provide data about dysrhythmia during normal daily activity. The age of the subjects ranged from 20 to 70 years, and the mean age was 44; 148 were less than 50 years of age. Dysrhythmia, mainly asymptomatic, occurred in 71 subjects (37 percent). In 31 subjects (16 percent) it was ventricular, in 20 (10.5 percent) it was supraventricular, and in a further 20 (10.5 percent) it was combined. In 12 subjects (6.3 percent) ventricular ectopy was considered potentially serious. Sixty-two of the subjects had never smoked and 27.4 percent of these showed ventricular ectopic activity. Forty-eight had given up smoking for at least 3 months or longer and 29 percent of these showed some ventricular ectopic activity. There were 79 smokers and 25.3 percent of these showed some ventricular ectopic activity. There was no apparent difference in ventricular ectopic activity in any of these groups. (Auth. Abs. Mod.)

80–0959. Aronov, D. M. **Sovremennye Predstavleniya o Vliyanii Kureniya na Serdechno-Sosudistuyu Sistemu. [Present-Day Concepts of the Effect of Smoking on the Cardiovascular System.]** *Terapevticheskii Arkhiv* 50(4): 117-126, 1978, Russian.

Research data from predominantly (about 85 percent) American and other Western sources are reviewed. The extent to which smoking has spread and its medical (e.g., cardiovascular) and social consequences are discussed. The 1940-75 Soviet statistics show a steadily increasing consumption of tobacco products. Antismoking campaigns and their results in different countries are briefly reviewed. In the Soviet Union, intensification of the antismoking campaign in the past few years is noted, starting with the First All-Union Conference on Smoking Prevention in 1976.

80–0960. Chahidi, H. **A Propos de 76 Cas de Thrombo-Angeite Obliterante. [Seventy-six Cases of Thromboangiitis Obliterans.]** *Phlebologie* 30(1): 103-111, January-March 1977, French.

A group of 87 patients, aged 10-40, with thromboangiitis (Buerger's disease) and a group of 9 patients, aged 40-50, with middle-age arteritis were among 156 patients treated for different arteriopathies over a 15-year period. In the first group, some of the smoking patients continued smoking even after a cervical and dorsal sympathectomy. The patients of the second group were all male and heavy smokers. All the smokers were repeatedly advised to cease smoking during hospitalization and after surgical intervention. Examination of the 64 cases of thromboangiitis in the patients hospitalized with more or less developed trophic lesions, i.e., gangrene of the toes, led to conclusions that tobacco smoking is an essential etiological factor of this disease, more important than race or climate. Pathogenesis of the disease is believed to be an allergic sensitivity of the blood vessels to nicotine. In the three patients of the first group who continued smoking, hypersensibility to nicotine appeared to be the cause of the disease spreading to the lower extremities in less than 3 months, in spite of reduced daily cigarette consumption.

80–0961. Chalmers, D. M.; Levi, A. J.; Chanarin, I.; North, W.R.S.; Meade, T. W. **Mean Cell Volume in a Working Population: The Effects of Age, Smoking, Alcohol and Oral Contraception.** *British Journal of Haematology* 43(4): 631-636, December 1979.

The effects on mean red cell volume (MCV) of age, smoking habit, alcohol consumption, menopausal status, and use of oral contraceptives have been studied as appropriate in 1,596 white men (aged 18-64) and 892 white women (aged 18-59) in working populations in North-West London. There were 801 male and 296 female smokers and 795 male and 501 female nonsmokers. In men, increasing age, smoking, and alcohol consumption each make an independent contribution to MCV. In women, the effect of smoking is similar to that in men; the effect of alcohol is less obvious, possibly because of the low stated alcohol intake in women. In each sex, MCV values for drinkers are higher than for nondrinkers, and values for smokers are higher than for nonsmokers. Smokers/drinkers have the highest values, and nonsmokers/nondrinkers the lowest; however, the differences are not large. The effect of alcohol on MCV in the population studied is not as marked as in hospital patients who admit to excessive alcohol consumption; this may partly be due to differences in methods of eliciting intake. There is a small increase in MCV following the menopause. Women on oral contraceptives show a rise in MCV with increasing age; this is not seen in women not on oral contraceptives. There is a strong inverse association between MCV and red blood cell count, which may be part of a mechanism to insure constant oxygen carrying capacity. (Auth. Abs. Mod.)

80–0962. Ducimetiere, P.; Eschwege, E.; Richard, J.; Claude, J.; Elgrishi, I. **Relationship of Glucose Tolerance to Prevalence of ECG Abnormalities and to Annual Mortality From Cardiovascular Disease: Results of the Paris Prospective Study.** *Journal of Chronic Diseases* 32(11/12): 759-766, 1979.

The relationship of glucose tolerance measured by plasma glucose level 2 hours after a 75 g glucose oral load to prevalence of electrocardiogram (ECG) abnormalities and mortality from ischemic heart disease is investigated in the Paris Prospective Study. This paper deals with findings in 6,589 men aged 42-53 working in the Paris Civil Service. The subjects were nondiabetic and not treated for hypertension. The ECG recording took place at entry in the study and was coded according to the Minnesota Code. Data on glucose level and other variables were collected at the first followup examination, 1 year later. The prevalence of definite Q wave, of ECG abnormalities according to the criteria of the Whitehall Study and the U.S. Pooling Project did not show any consistent trend among deciles of glucose. These abnormalities were not significantly associated with glucose level in the multiple logistic analysis taking into account age, systolic blood pressure, cholesterol, body mass index, and cigarette consumption. Mean annual mortality rates (all causes, cardiovascular and ischemic heart disease) were computed in the population followed for an average of 5 years, after exclusion of 105 men with definite Q wave at entry. While a possible U-shaped relationship between glucose and mortality might be suggested from quintile analysis, the multivariate analysis did not show any significant independent association between glucose level and mortality from cardiovascular or ischemic heart disease causes. (Auth. Abs.)

80–0963. Farinaro, E.; Panico, S.; Oriente, P.; Mancini, M. **The Olivetti Survey: Asymptomatic Hyperglycemia and ECG Abnormalities at Baseline.** *Journal of Chronic Diseases* 32(11/12): 773-777, 1979.

As the first step in a planned long-term followup of 694 middle-aged men employed by the Olivetti Company in Naples, baseline characteristics were determined by examination. Measurements were made of those variables demonstrated in the past to have a relationship with cardiovascular diseases (blood pressure, serum cholesterol, weight, cigarette smoking). The men were divided according to smoking status and the prevalence of electrocardiogram (ECG) abnormalities was examined. Ex-smokers had higher rates of ECG abnormalities than other smoking categories. A multiple logistics analysis was performed to clarify this finding. While the association of other variables with ECG abnormalities changed little, the t value for the smoking variable shifted from negative to positive, reaching statistical significance for those abnormalities included under Whitehall criteria. Because this population group differed somewhat from others in most industrialized countries, i.e., a lower mean serum cholesterol but a higher percentage smoking (70 percent), it was of particular interest to determine whether hyperglycemia would have an association with ECG abnormalities similar to any association found in other concurrent studies. While, in this prevalence study, there appears to be a positive (although inconsistent) association, long-term mortality followup will be needed to determine the prognostic significance of such asymptomatic hyperglycemia in the special circumstances of lifestyle characteristic of Naples. (Auth. Abs. Mod.)

80–0964. Frishman, W. H. **Involuntary Smoking: Cardiovascular Effects of Smoke on Nonsmokers.** *Cardiovascular Medicine* 4(3): 289-291, 296, March 1979.

The cardiovascular effects of smoking on the smoker have been extensively studied, but the effects of tobacco smoke on nonsmokers have received much less attention. Tobacco smoke can be a significant source of atmospheric pollution in enclosed areas. Occasionally, under conditions of heavy smoking and poor ventilation, the maximum limit for an 8-hour work exposure to carbon monoxide (CO) (50 ppm) may be exceeded. Levels of CO commonly found in cigarette smoke-filled environments have been shown to decrease the exercise tolerance of patients with angina pectoris and intermittent claudication. However, there are no studies to suggest any chronic cardiovascular consequences of involuntary smoking in humans. (Auth. Abs. Mod.)

80–0965. Green, K. G. **Long Term Multicenter Trial of Practolol in Patients Recovered From Acute Myocardial Infarction: Organizational Procedure and Results Related to Smoking Habits.** *INSERM Symposia* 76: 49-56, December 9-11, 1977.

The effects of smoking habit on the results of a controlled trial of practolol involving 3,052 patients recovering from acute myocardial infarction are studied here. An analysis of the trial results in relation to smoking habit included four findings. (1) In the untreated group, smoking did not increase the total of new cardiac events, but greatly increased the proportion of events which were fatal. The results for discontinued smokers (those who smoked before the trial but not during it) were similar. (2) Smokers and discontinued smokers had significantly ($p<0.001$) lower blood pressures (BP) than nonsmokers. However, the mean BP for discontinued smokers had risen to the level of that for nonsmokers, after 18 months. (3) Practolol appeared to have abolished the increased death rate in smokers and discontinued smokers. (4) Nonfatal reinfarction rates were almost halved by practolol in nonsmokers and discontinued smokers, but the rate for smokers was unchanged.

80–0966. Holme, I.; Helgeland, A.; Hjermann, I.; Leren, P.; Lund-Larsen, P. G. **Koronare Risikofaktorer og Sosiookonomisk Status. Oslo-Undersokelsen. [Coronary Risk Factors and Socioeconomic Status. The Oslo Study.]** *Tidsskrift for den Norske Laegeforening* (27): 1380-1382, 1394, September 30, 1977, Norwegian.

A 1972-1973 study made of 18,000 men who were 20 to 49 years old is the basis for this analysis of the link between socioeconomic status and risk factors for heart and vascular diseases. It was found that high status individuals had far lower risk profiles than low status individuals. The most dominant risk factor in the group of low socioeconomic status was cigarette smoking. The subjects were divided into five groups on the basis of income and education, with group 1 having the highest socioeconomic status. In group 1 the percentage of daily smokers was 17.7, ex-smokers 43.6, and nonsmokers 38.7. In group 5 the corresponding figures were 65.7, 18.4, and 15.9. The risk for heart infarction at an early age decreased by more than half from group 5 to group 1, mainly because of smoking. Education was an important factor in establishing smoking habits. Individuals with high education had low risk factor levels mainly because of low smoking frequency and below average lipid levels.

80–0967. Karmazyn, M.; Manku, M. S.; Horrobin, D. F. **The Mechanism of Coronary Artery Spasm: Roles of Oxygen, Prostaglandins, Sex Hormones and Smoking.** *Medical Hypotheses* 5(4): 447-452, April 1979.

A reduced oxygen supply to the heart causes coronary vasodilatation in the first instance. But if the hypoxia is severe or prolonged, the dilatation passes off and coronary vasospasm develops leading to a vicious circle with a further reduction of myocardial oxygenation. The spasm is associated with increased outflow of prostaglandin (PG)-like material and can be prevented or reversed by inhibitors of PG synthesis such as indomethacin or antagonists of PG action such as chloroquine. The spasm does not appear to be caused by thromboxane (TX) A2 since selective inhibitors of TXA2 synthesis enhance the hypoxic spasm and by themselves can cause spasm even in oxygenated hearts. The mechanism may be related to loss of negative feedback control of the PG pathway by TXA2. Oxygen may enhance TXA2 production and reduce formation of vasoconstrictor PGs, while smoking, because of the formation of carboxyhemoglobin, may have the opposite effect. Estradiol and testosterone do not influence the hypoxic spasm, but progesterone at physiological concentrations blocks it completely. Progesterone may be the protective female hormone, and the increased susceptibility to myocardial infarction in women on oral contraceptives may be related to reduced formation of endogenous progesterone. (Auth. Abs.)

80-0968. Kavanagh, T.; Shephard, R. J.; Chisholm, A. W.; Qureshi, S.; Kennedy, J. **Prognostic Indexes for Patients With Ischemic Heart Disease Enrolled in an Exercise-Centered Rehabilitation Program.** *American Journal of Cardiology* 44(7): 1230-1240, December 1979.

Indexes of prognosis were calculated for 610 postinfarction patients who participated in a vigorous exercise-centered rehabilitation program for an average of 36.5±6.5 months (mean ± standard deviation) commencing 8.2±11.7 months after infarction. Examination of risk ratios P/N (predictive value of positive test result divided by predictive value of negative test result) revealed that the most useful individual index of a poor prognosis was failure to comply with the prescribed exercise regimen. Other adverse findings included angina at the final exercise test, radiographic evidence of cardiac enlargement, horizontal or downsloping S-T depression (0.2 mv or greater), a serum cholesterol level greater than 270 mg/100 ml (7 mmol/liter), persistence of cigarette smoking, and use of digitalis and diuretic agents. The majority of the items cited had a fair specificity, but (with the exception of noncompliance with the exercise program, S-T depression, and persistent smoking) had a rather low sensitivity. Recurrence of infarction was apparently strongly associated with the attitude of the patient toward life and treatment. Of the 44 episodes, 23 occurred in patients who had stopped exercising, although questioning of these patients indicated that a continuation or a progression of their cardiac disease was rarely responsible for nonparticipation. Continued cigarette smoking was common in those with a recurrence. Two of he deaths occurred in men smoking 40-50 cigarettes/day, and one reinfarction occurred in a faithful adherent of the raining program who had returned to the habit of smoking 20-30 cigarettes/day. Although the effect of some traditional isk indicators was unchanged in postinfarction patients undergoing exercise training, the adverse influence of exercise -T segment depression, a high serum cholesterol level, and continued cigarette smoking was greater than anticipated, whereas age and hypertension had less than the expected effect. Patients with S-T depression, a high serum cholesterol level, or a continuing cigarette habit showed less benefit than their exercising counterparts, but their prognosis remains more favorable than that of subjects who do not exercise.

80-0969. Kornitzer, M.; De Backer, G.; Dramaix, M.; Thilly, C. **The Belgian Heart Disease Prevention Project. Modification of the Coronary Risk Profile in an Industrial Population.** *Circulation* 61(1): 18-25, January 1980.

The Belgian Heart Disease Prevention Project is a controlled, multifactorial prevention trial involving 19,390 males aged 40 to 59 years employed by 30 Belgian industries. These industries were paired and randomized into a control or intervention unit. For all the subjects in the intervention group and for the 10 percent of the control group that underwent the same thorough initial examinations, risk profiles were established on the basis of the initial results, according to a "risk score." In the case of smoking, fewer than 5 cigarettes/day added no points to the risk score, 5-20 cigarettes/day added 1 point and smoking more than 20 cigarettes/day added 2 points to the score. In each intervention factory, the subjects from the two highest deciles of a coronary risk-score distribution curve (85 percent of whom smoked five or more cigarettes a day) were given individual advice twice a year. Subjects who smoked five or more cigarettes a day were encouraged to quit. A health education campaign was also organized in each intervention factory. After 2 years, high-risk subjects and random samples of the control and intervention group were compared regarding the coronary risk profile by means of a multiple logistic function (MLF). In the intervention high-risk group, the MLF showed a decrease of 20 percent, and in the control group there was an increment of 12.5 percent ($p<0.001$). Comparing the random samples, an increment of 25 percent was found in the control group vs. a drop of 2.26 in the intervention group ($p<0.001$). Among the high-risk subjects, 12.2 percent of the control subjects who were cigarette smokers at entry said they had stopped smoking, whereas the corresponding percentage was 18.7 percent in the intervention group, a statistically significant difference. There was an insignificant increase of 8.6 percent in the number of cigarettes smoked daily by continuing smokers in the control group against a significant drop of 18.8 percent in the intervention group, again a statistically significant difference. (Auth. Abs. Mod.)

80-0970. Krantz, D. S.; Sanmarco, M. I.; Selvester, R. H.; Matthews, K. A. **Psychological Correlates of Progression of Atherosclerosis in Men.** *Psychosomatic Medicine* 41(6): 467-475, October 1979.

Patients who underwent repetitive coronary angiograms at an average interval of 17 months completed the Jenkins Activity Survey ($n=66$) and were given the Rosenman diagnostic interview to measure Type A behavior ($n=45$).

Significant progression of coronary artery disease was judged to be an increase in occlusion of 25 percent or more, or progression to total occlusion in any vessel. At subsequent study, 30 percent of the patients showed evidence of progression. The incidence of smokers was 22 percent in this group. Progression was much more likely in patients with initially more severe disease and was marginally related to cigarette smoking when initial level of disease was controlled. Interview assessment of Type A did not discriminate reliably between the groups in the smaller sample of patients given this measure. There was significant positive association between magnitude of Activity Survey Type A scores and progression of disease, although mean scores on the Type A scale were not reliably different between the Progression and No Progression groups. The pattern of results suggested that extreme Type B subjects (classified by Activity Survey) were unlikely to show progression over this time period. (Auth. Abs. Mod.)

80–0971. Lovlin, R. E.; Sewchand, L. S.; Beck, J. S.; Rademaker, A. W.; Rowlands, S. **Erythrocyte Volume and Count: A Linear Relation Descriptive of Population Differences.** *Scandinavian Journal of Haematology* 22(3): 197-204, March 1979.

In a sample of 215 adult males the mean red cell volume decreased linearly with increasing red cell concentration. The mean corpuscular volume and the red cell count were not correlated with age of subject. Of the 183 subjects whose smoking status was recorded, 69 were smokers and 114 nonsmokers. There was a significant difference between smokers and nonsmokers with respect to mean red cell volume ($p<0.01$), hematocrit ($p<0.01$), mean corpuscular volume ($p<0.01$), and blood viscosity ($p=0.01$), with smokers having median levels higher than nonsmokers. Smokers also had higher values of hemoglobin and mean corpuscular hemoglobin, but the differences were not significant. Optical determination of cell volume and area confirmed the linear relation of volume and count. (Auth. Abs. Mod.)

80–0972. Magnus, K.; Matroos, A.; Strackee, J. **Walking, Cycling, or Gardening, With or Without Seasonal Interruption, in Relation to Acute Coronary Events.** *American Journal of Epidemiology* 110(6): 724-733, December 1979.

The case-referent study reported here investigates the relationship between acute coronary events (ACE) and sustained light physical exercise, mainly leisure-time walking, cycling, or gardening (WCG). These activities are classified as habitual WCG (>8 months/year), seasonal WCG (4-8 months), or occasional WCG (<4 months), each category being further subdivided according to the number of hours a week: <4 hours, 4-7 hours, or >7 hours. A significant negative association is found between ACE and habitual WCG. No such relationship is evident between ACE and seasonal WCG, however, seasonal WCG being equally frequent (27 percent) in both cases and referents. These observations may indicate that any effect on the incidence of acute coronary events that is caused by walking, cycling, or gardening is neutralized if these activities are interrupted for several months of the year. The negative association between habitual WCG and ACE does not depend on the number of hours a week devoted to WCG and is not enhanced by more vigorous additional exercise. The fatality of ACE (death rate within 4 weeks) is significantly higher in the least active group (WCG<4 months/year). The effect of WCG is significantly weakened by smoking ($p<0.05$), but even among smokers the effect of habitual exercise is still present. (Auth. Abs. Mod.)

80–0973. McAloon, E. J.; Streiff, R. R.; Kitchens, C. S. **Erythrocytosis Associated With Carboxyhemoglobinemia in Smokers.** *Southern Medical Journal* 73(2): 137-139, February 1980.

During a 2-year period, 14 patients with erythrocytosis were evaluated. Carboxyhemoglobinemia secondary to cigarette or cigar smoking or both caused elevated hematocrit values in 10 of the patients, and in all 14 either red cell volume was increased or plasma volume was decreased. Six patients smoked from 1 to 3 packs of cigarettes per day and 3 smoked 6 to 12 cigars per day. One patient smoked both three packs of cigarettes and six cigars per day. Carboxyhemoglobin levels ranged from 8.5 percent to 18.0 percent, with a mean of 13.0 percent. There was no correlation between the number of cigarettes or cigars reportedly smoked and either the degree of carboxyhemoglobinemia or the hematocrit value. In three patients who stopped smoking permanently, hematocrit values decreased from a mean of 56 percent to 46 percent. Even though the high hematocrit value reversed when these patients stopped smoking, limited success was encountered in persuading the others to do so. (Auth. Abs. Mod.)

80–0974. Neuman, J.; de Neuman, M. P.; Valero, E.; Lindental, D. **Epidemiology of Coronary Heart Disease Risk Factors in a Free-Living Population.** *Preventive Medicine* 8(4): 445-462, 1979.

A project for the detection and treatment of multiple risk factors for coronary heart disease (CHD) has been initiated in Argentina. After complete medical and routine laboratory studies, 462 apparently normal subjects were found. The prevalence of risk factors was hyperlipoproteinemia (HLP) 43.9 percent; hypertension ($\geq$160-95) 17.3 percent; smoking habits 35 percent (heavy smokers ($\geq$20 cigarettes/day) 7.9 percent, significantly ($p<0.001$) more men smoked than women); psychosocial stress 49.5 percent; overweight (weight/height>1.10) 40.3 percent; sedentary habits 44.8 percent; family history of CHD 10.9 percent; diabetes 6.9 percent; and minor electrocardiographic abnormalities 6.6 percent. Risk factors associations have been demonstrated between type IV HLP, overweight, hypertension, tobacco smoking, and diabetes. Risk factor prevalence tended to increase with advancing age with the exception of smoking habits. In this apparently normal population, 36.9 percent of the men and 19.4 percent of the women had two or more major risk factors requiring treatment because of their high CHD risk. (Auth. Abs. Mod.)

80-0975. Pyorala, K.; Savolainen, E.; Lehtovirta, E.; Punsar, S.; Siltanen, P. **Glucose Tolerance and Coronary Heart Disease: Helsinki Policemen Study.** *Journal of Chronic Diseases* 32(11/12): 729-745, 1979.

The relationship of glucose tolerance to the prevalence of symptoms and electrocardiographic (ECG) signs of coronary heart disease (CHD), and to the 10-year mortality from all causes, cardiovascular diseases and CHD was investigated in 867 Helsinki policemen aged 40-59 on whom an oral glucose tolerance test was carried out in 1966-1967. An oral glucose load of 75 or 90 g according to body surface area was used and blood glucose was determined fasting, 1 and 2 hours after the glucose load. The 2-hour post-load blood glucose was found to be significantly ($p<0.001$) lower in current smokers than in nonsmokers. In multivariate analyses including age, systolic blood presure, plasma cholesterol, body mass index, and smoking, the blood glucose variables failed to show a significant independent relationship to the prevalence of ECG abnormalities and did not show a significant independent contribution in the prediction of the 10-year mortality from all causes, cardiovascular diseases or CHD. (Auth. Abs. Mod.)

80-0976. Romanski, B.; Broda, S.; Swiatkowski, M.; Zbikowska, M. **The Immunologic Response to Tobacco Antigens in Smokers. III. Type III Hypersensitivity Skin Reactions and Specific Serum Precipitins to Four Different Tobacco Extracts in Patients Suffering From Coronary Artery Disease.** *Allergologia et Immunopathologia* 7(3): 187-196, May-June 1979.

In a population of 70 patients suffering from coronary heart disease (49 smokers and 21 nonsmokers) intradermal tests and specific precipitation tests with four different tobacco extracts were performed. Skin tests with tobacco extracts were positive in 41 smokers and 7 nonsmokers. In both groups the immediate and delayed type reactions were weak and of rather doubtful specificity, while the late type reaction was usually more frequent and stronger in smokers (in 14 patients it was positive with all preparations). Specific precipitins against tobacco antigens (Ag) were found in the sera of 13 smokers but never in nonsmokers. The positive precipitation test correlated well with the positive late skin reactions to tobacco extracts. Such correlation with Ag I was observed in 9 out of 13 patients, with Ag II in four cases, with Ag IV and Ag V in 2 and 2 patients, respectively. The coincidence of strongly positive late skin reactions and positive precipitation tests with tobacco extracts seems to indicate that tobacco allergy really exists and resembles, in a number of patients with coronary artery disease, type III hypersensitivity reactions to tobacco antigens. It is postulated that immune complexes of precipitins and tobacco antigens may be formed in some patients and initiate pathological processes in coronary arteries leading to their occlusion. (Auth. Abs.)

80-0977. Rose, G. A. **Smoking.** In: Oliver, M. F. (Editor). *Coronary Heart Disease in Young Women.* Churchill Livingstone, 1978, pp. 36-42.

Several studies of coronary heart disease in cigarette smokers are briefly reviewed and data on women are assessed. Angina, coronary artery disease, and myocardial infarct are found in heavy smokers. It appears that smoking habits help identify a high-risk group and that this predictive effect is fairly independent of other major risk factors. However, the risk may be compounded when smoking occurs with other risk factors in the same woman.

80-0978. Rosenman, K. D. **Cardiovascular Disease and Environmental Exposure.** *British Journal of Industrial Medicine* 36(2): 85-97, May 1979.

This paper reviews the possible association between cardiovascular disease and occupational and environmental agents. The effects of carbon monoxide, fibrogenic dusts, carbon disulphide, heavy metals, noise, radiation, heat, cold, solvents, and fluorocarbons are discussed. New directions for investigation are suggested. In occupationally exposed workers, carboxyhemoglobin (COHb) levels in nonsmokers may be as high as, and sometimes higher than, those in nonexposed smokers. Smokers with COHb levels exceeding 5 percent run 20 times the risk of having atherosclerotic heart disease as do smokers of the same age and sex with COHb levels below 3 percent. (Auth. Abs. Mod.)

80-0979. Serebryakova, I. K. **Znachenie Nekotorykh "Faktorov Riska" v Razvitii Infarkta Miokarda. [Significance of Certain Risk Factors in the Development of Myocardial Infarction.]** *Zhurnal Eksperimental'noi i Klinicheskoi Meditsiny* 18(6): 65-68, 1978, Russian.

The effects of smoking, arterial hypertension, hereditary predisposition, and stress on the development of ischemic heart disease and myocardial infarct were studied in 187 patients. There were 165 men and 22 women, 120 of whom were 20 to 60 years old; the rest were 60 to 90 years old. It was found that 119 patients out of 187 had been smoking from 5 to over 50 years; average cigarette consumption varied from 20 to 80/day. At the time of the study, 55 smokers were younger than 60 years; 64 were over 60. Smoking, hereditary predisposition, and stress were found to be important risk factors for myocardial infarct and ischemic heart disease.

80-0980. Stamler, J. **Research Related to Risk Factors.** *Circulation* 60(7): 1575-1587, December 1979.

The epidemic of premature coronary heart disease (CHD) in the United States and other Western industrialized countries is a result of modern lifestyles and the risk factors related to them. It is scientifically correct to designate habitual eating patterns high in cholesterol, saturated fats, and calories as the primary cause of this epidemic. Such diets are responsible for the high prevalence rates in these populations of hypercholesterolemia, an established major CHD risk factor. Additional established major risk factors that enhance coronary proneness in persons and populations with the lipid nutritional-metabolic prerequisites for severe atherosclerosis are hypertension and cigarette smoking. A sedentary lifestyle at work and leisure and incongruent behavior (e.g., the type A behavior pattern) are probable additional adjuvant risk factors in such populations. Data on the impact of major risk factors in one population enable prediction of risk in other populations. In populations with lifestyles rendering them generally coronary-prone, accurate assessment of CHD risk can be made at least from youth on, probably even in the first decade of life, enabling a preventive public-health strategy that combines efforts to improve lifestyles (diet, smoking, and exercise) for the whole population from childhood (primary-habit formation) through middle age, with special concentrated attention to identify very high risk individuals and families. (Auth. Abs.)

80–0981. Staugard, F. **Forsok med Halsoundervisning Baserad pa Riktad Halsokontroll. [Experimental Health Education Based on Health Control of Selected Groups.]** *Nordisk Medicin* 93(11/12): 287-288, 1978, Swedish.

In a city in Sweden all government employees aged 30-39 were invited for clinical examinations and health education. The 1,091 participants represented 94 percent of the individuals invited. Forty-three percent of the women and 48.5 percent of the men were smokers. Of the female smokers, 15.4 percent smoked 1-9 cigarettes per day and 24.1 percent smoked 10-39 cigarettes per day. Of the male smokers, 18.2 percent smoked 1-9 cigarettes per day and 16.7 percent smoked 10-39 cigarettes per day. The health status of 71.5 percent of the women and 44.0 percent of the men included one or more risk factors for heart and vascular diseases. Health checkups are regarded as an efficient means to increase the individual's motivation to apply the health education given at the primary health care center, especially with regard to the prevention of heart and vascular diseases.

80–0982. Steinmetz, J. R. **The Assessment of Personal Characteristics and Health Practices Associated With Coronary Heart Disease in Adults.** Doctoral Dissertation, Temple University, University Microfilms 77-13,530, 1977, 101 pp.

The major purpose of this study was to construct a self-administered instrument that would identify the presence or absence of risk factors associated with coronary heart disease. The specific risk factors included in this study were associated with (1) family health history, (2) the subject's medical history, (3) smoking practices, (4) physical activity level, (5) dietary habits, and (6) the selected personality characteristics. A secondary purpose of this study was to analyze the differences among risk factors associated with men and women within and between selected age groups. In order to field test this instrument, it was administered to male and female subjects between the ages of 21 and 65. In the case of smoking practices, it was found that most of the subjects, both male and female, were either nonsmokers or heavy smokers. There were 132 individuals (80.49 percent) who did not smoke or who had stopped smoking for 6 or more years. Twenty-two (13.42 percent) of the respondents smoked two or more packs of cigarettes per day, and only seven individuals reported smoking at a moderate level. The data supported the conclusion that the questionnaire is a reliable instrument for assessing the personal characteristics and health practices associated with coronary heart disease. Statistical treatment of the data indicated a reliability coefficient of .963 for the combined risk factors.

80–0983. Wald, N. J. **Cigarette Smoking and Coronary Heart Disease in Young Women.** In: Oliver, M. F. (Editor). *Coronary Heart Disease in Young Women.* Churchill Livingston, 1978, pp. 43-58.

This paper describes patterns of cigarette consumption among women in the United Kingdom and summarizes the results of studies investigating the relationship between smoking and coronary heart disease (CHD) in women. Although extensive prospective epidemiological data are only available from one study, information on the prevalence of CHD in women suggests that smoking is associated with the disease, that the risk is similar to that found in men, and that there is a dose—response relationship between amount smoked and the incidence of the disease. There is evidence that smoking is associated with early menopause, and this may account for the observation that early menopause is associated with an increased risk of CHD. The interaction between smoking and other risk factors is inconclusive, but one investigation suggests that they act multiplicatively, so that the risk in women with three or more risk factors may be very high indeed.

See also, **80–0916, 80–0998**

PREGNANCY AND INFANT HEALTH

80–0984. Bystrom, G.; Lundgren, M.; Meyer-Lie, A.; Palmborg, M. **En Studie Over Orsaker till Missbildningsbord och Perinatal Dodlighet i Varmland. [A Study of Factors Causing Birth Defects and Perinatal Mortality in Varmland.]** *Lakartidningen* 74(38): 3167-3169, 1977, Swedish.

This study of factors causing birth defects and perinatal mortality does not support the idea that phenoxy acid used in forestry causes malformations. Interviews were conducted with 128 mothers of malformed or perinatally dead children born 1970-1975. This group included control individuals who had either given birth to children with trisomy or whose children had died due to birth complications in which exogenous factors did not affect the pregnancies. The smoking habits of the control group mothers were the same as those of pregnant women in general, but there was a higher frequency of smokers among women who had had ablatio placentae or who had given birth to macerated fetuses. Cigarette smoking seems to be linked to an increased risk for certain types of malformations, especially split lip and split jaw, as well as caudal malformations. These malformations are probably caused by several genetic as well as exogenous factors. Smoking may enhance the effect of a local factor or may cause the increased frequency of caudal malformations in certain geographical areas like Varmland in Sweden.

80–0985. Hollinshead, W. H., III. **The Consequences of Smoking in Pregnancy.** *Rhode Island Medical Journal* 62(6): 207-212, June 1979.

Recent findings on the fetal effects of maternal smoking are presented, including decreased birthweight, spontaneous abortions and anomalies, perinatal mortality, and perhaps slowed childhood development. The risk to succeeding generations is suggested as another powerful argument against smoking, with special relevance to young women, who are the only category of Americans with an increasing proportion of smokers. (Auth. Abs.)

See also, **80–0890**

80–0986. Phelan, J. P. **Diminished Fetal Reactivity With Smoking.** *American Journal of Obstetrics and Gynecology* 136(2): 230-233, January 15, 1980.

The fetal activity-acceleration determination (FAD), or nonstress test, has been demonstrated to be an effective method for the antepartum assessment of fetal status in high-risk pregnancies. A reactive or negative test has been equated with fetal well-being, while fetal compromise has been related to a nonreactive or positive FAD. The present investigation was designed to determine the effects of chronic cigarette smoking on fetal reactivity as measured by the FAD. Increased fetal reactivity might be expected from recent investigations of the acute effects of cigarette smoking on fetal breathing movements and the fetal heart rate. However, in comparing 128 smoking and 350 nonsmoking pregnant women with high-risk pregnancies, nonreactive or positive FAD's were demonstrated to be significantly higher in the smoking group ($p<0.005$). (Auth. Abs.)

80–0987. Wood, C.; Gilbert, M.; O'Connor, A.; Walters, W.A.W. **Subjective Recording of Fetal Movement.** *British Journal of Obstetrics and Gynaecology* 86(11): 836-842, November 1979.

A group of 137 patients (34 smokers), 113 of whom had obstetric complications, recorded fetal movements by a subjective method. Low fetal movement counts were associated with maternal cigarette smoking ($p<0.01$) and prolongation of pregnancy, and high counts with maternal ingestion of sedatives or tranquillizers, abnormal fetal heart rate in labor, and a true knot in the umbilical cord. The absence of fetal movement during four consecutive 20-minute counting sessions was not associated with poor fetal outcome. (Auth. Abs. Mod.)

OTHER DISEASES AND CONDITIONS

80–0988. Bastiaan, R. J.; Waite, I. M. **Effects of Tobacco Smoking on Plaque Development and Gingivitis.** *Journal of Periodontology* 49(9): 480-482, September 1978.

A study was undertaken to test the hypothesis that patients who smoke tobacco have a more rapid rate of plaque deposition and development of gingivitis than those patients who do not smoke. Twenty subjects participated in this study; their age range was 17 to 30 years. Ten were smokers and 10 were nonsmokers. After an initial thorough removal of all deposits, oral hygiene measures were stopped for 10 days. On days 3, 7, and 10, plaque levels were evaluated using the

Plaque Index, and the gingival status was evaluated using the Gingival Index. Samples of plaque were stained by the Gram technique and examined microscopically. Plaque levels appeared to be higher in smokers than nonsmokers, but the differences were not statistically significant. No consistent differences were evident in the gingival status of the two groups. Microbiological analysis showed a statistically significant increase in the percentage of gram-positive bacteria to gram-negative bacteria in the smokers as compared to the nonsmokers on day 3. However, these differences were not maintained in the plaque samples taken after the third day. (Auth. Abs.)

80–0989. Daftary, D. K.; Pitkar, V. K.; Gupta, P. C.; Pindborg, J. J.; Metha, F. S. **A Study of the Natural History of Oral Preleukoplakia.** *Acta Odontologica Scandinavica* 36(6): 327-331, 1978.

Preleukoplakia, thought to be a precursor stage of leukoplakia, is defined as a very mild reaction of the oral mucosa, appearing as a gray or grayish-white area with indistinct borders. The paper describes 659 preleukoplakias detected among 20,358 individuals in two districts in India. In Bhavnagar district, 99.5 percent of the preleukoplakias occurred among men; 23.5 percent of the hookah (clay pipe) smokers and 9.3 percent of the bidi smokers had preleukoplakias. In Ernakulam district, 79.6 percent of the preleukoplakias were found among men, and the mixed habit group of bidi smoking and tobacco chewing had the highest prevalence (6.1 percent). The incidence of preleukoplakias was lowest in those with no smoking or chewing habits both in Bhavnagar and Ernakulam (0.03 and 0.2 percent, respectively). The preleukoplakias showed a marked tendency to regress; among the bidi smokers in Bhavnagar, 75 percent regressed. In two patients preleukoplakia turned into carcinoma. (Auth. Abs. Mod.)

80–0990. Kuznetsov, V. S.; Morozov, A. B. **Znachenie Otdel'nykh Faktorov v Formirovanii Rasprostranennosti Neyrosensornoy Tugoukhosti. [Significance of Individual Factors in the Neurosensory Hearing Impairment Pattern.]** *Vestnik Otorinolaringologii* (2): 3-7, March-April 1979, Russian.

The effects of internal factors and environmental conditions on the incidence of neurosensory hearing impairment were determined from the results of clinical and statistical observations of 8,700 children and adults residing in the city of Cheboksary. Neurosensory hearing impairment was diagnosed in 2.5 percent of the patients. Analysis of the statistical data attested to the effect of working conditions (noise, vibrations, elevated temperature, dust, etc.) on the incidence of impairment among blue collar or white collar workers. This incidence was 2 percent in nonsmokers, 3.5 percent in smokers of 10 cigarettes/day, and 5.7 percent in smokers of more than 10 cigarettes/day.

80–0991. Mitchell, C.; Stuart, R. B. **Weight Control and Smoking Reduction: Friends or Enemies?** *Canadian Journal of Public Health* 70: 399-404, November-December 1979.

Smokers who succeed in giving up smoking frequently complain about weight gain as a negative concomitant of their success. In this study, the reciprocal possibility was measured: an increase in smoking by those who succeed in losing weight. Data were collected from 363 female members of Weight Watchers' classes, all of whom had achieved their goal weights from 1 to 8 weeks before data collection. Slightly over half (51.3 percent) of the sample reported smoking at some time. Of these, 79 (21.8 percent of the entire sample) stopped smoking before joining a Weight Watchers' class, leaving 99 (27 percent of the original sample) who smoked at the start of their weight-loss efforts. Of these smokers, 67 percent continued to smoke at approximately the same rate until they reached their desired weights, 16.5 percent increased their smoking, 8.2 percent decreased their smoking, and 8.2 percent stopped smoking entirely. As the above results indicate, weight losers did not increase the rate of their smoking, nor did the techniques of behavioral self-management targeted to weight control generalize to smoking. Incidental data indicate that obesity and smoking appear to be independent threats to health and well-being and need not replace one another when treatment for either stresses the building of positive skills rather than simply stressing the suppression of the problem behaviors. (Auth. Abs. Mod.)

80–0992. Sweet, J. B.; Butler, D. P. **Effect of Smoking on the Incidence of Localized Osteitis Following Mandibular Third Molar Surgery.** *Quintessence International* 9(2): 9-10, February 1978.

In order to determine the effect of smoking on the incidence of localized osteitis in persons having undergone mandibular third molar surgery, 252 patients (15 to 35 years of age) were questioned preoperatively about their smoking habits. Only 71 of the 252 (28 percent) were smokers. This small group, however, had 9 of 14 (over 64 percent) of the total postoperative localized osteitis cases. The incidence of postoperative localized osteitis in smokers was over four times greater than that in nonsmokers. Among smokers, postoperative localized osteitis occurred in 9 out of 142 surgical sites (6.4 percent), but among nonsmokers in only 5 of 362 surgical sites (1.4 percent). The difference was statistically significant ($p<0.01$).

80–0993. Walker, V.; Taylor, W. H. **Cigarette Smoking, Chronic Peptic Ulceration, and Pepsin 1 Secretion.** *Gut* 20(11): 971-976, November 1979.

The relationship between the secretion of pepsin 1 (the most electronegative of the pepsins) and the smoking habits of 219 patients (142 smokers and 77 nonsmokers) has been investigated. Significantly more cigarette smokers with peptic ulceration (72.5 percent) secreted pepsin 1 in greater than trace amounts after pentagastrin or histamine than did nonsmokers with ulceration (51.2 percent) ($p<0.02$). Differences of a similar order were found for men with duodenal ulcer, women with duodenal ulcer, and all patients with gastric ulcer, but the difference was statistically significant only for men with duodenal ulcer ($p<0.05$). Significantly more patients with peptic ulcer smoking 6 to 15 cigarettes/day secreted moderate or high concentrations of pepsin 1 than did heavier smokers or nonsmokers. There was no significant association between cigarette smoking and pepsin 1 secretion among 74 patients without ulceration. Maximal acid output was not significantly related to smoking in any group studied. The findings add to the increasing body of evidence linking pepsins and pepsin 1 with the pathogenesis of peptic ulceration. (Auth. Abs.)

80–0994. Wojtyniak, B.; Gorynski, P. **Czestosc Wystepowania i Dynamika Nadwagi w Grupie Doroslych Mieszkancow Krakowa. Czesc II. Nadwaga wg Wyksztalcenia, Srodowiska Pochodzenia i Nalogu Palenia. [Prevalence and Dynamics of the Overweight in the Sample of the Adult Inhabitants of Cracow. Part II. Overweight by Education, History of Residence and Smoking Habit.]** *Przeglad Lekarski* 35(8): 683-688, 1978, Polish.

The effect of social conditions on obesity in 1,293 females and 965 males living in Cracow was studied. These persons were examined twice in 1968 and 1973. To define their obesity, the Broc index with the Ries adjustment was applied. Obesity and education are inversely related among males and females. In females, higher education correlates with lower prevalence of obesity whereas in males the opposite has been observed. The frequency of obesity was lower in nonsmokers as compared to smokers, and the probability of becoming overweight was highest in persons who gave up smoking. (Auth. Abs.)

See also, **80–0880**

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–0995. Ahmed, Z.; Abuhay, M. **The Prevalence of Cigarette-Smoking Among Secondary School Children in Gondar City, Ethiopia.** *Ethiopian Medical Journal* 17(2): 41-46, April 1979.

The smoking habits of 538 schoolchildren (255 boys and 283 girls between 12 and 23 years of age) in the city of Gondar, Ethiopia, were studied in a sample survey by means of a questionnaire. The prevalence of cigarette smoking was 27.5 percent among boys. In contrast, only 2.8 percent of the girls were smokers. About 85 percent of both sexes were regular rather than experimental smokers. Among the regular smokers, 55 (83.3 percent) reported smoking 1-10 cigarettes per day, while the remaining 16.7 percent smoked 11-20 cigarettes per day. All female regular smokers fell into the former category. Thus, the majority of the regular smokers were "light" smokers, while "heavy" smokers were not encountered. The majority of students started smoking between the ages of 16 to 18 years. Whether or not a student smoked was significantly associated with having friends who smoked ($p<0.001$).

80–0996. Bonham, G. S.; Leaverton, P. E. **Use Habits Among Adults of Cigarettes, Coffee, Aspirin, and Sleeping Pills, United States, 1976.** Vital and Health Statistics, Series 10, Data From the National Health Survey, No. 131, DHEW Publication No. (PHS) 80-1559, October 1979, 52 pp.

A representative sample of the noninstitutional population of the United States was surveyed and information concerning the use of cigarettes, coffee, aspirin, and sleeping pills was obtained. Current smokers comprised 45.9 percent of the population with 9-11 years of school completed, in contrast to 26.9 percent of the population who had completed college. Over half of the people who were separated (51.1 percent) or divorced (56.7 percent) smoked cigarettes compared with just over a third of the people who had never married (34.6 percent) or who were currently married (36.6 percent). A greater percent of black than white people smoked, but the average amount smoked was less among black smokers than white smokers. Persons of other races were the least likely to smoke, although smokers of other races smoked about the same amount as did black smokers. Smoking also varied by occupation. A greater percent of blue-collar workers (48.1 percent) than white-collar workers (35.5 percent) smoked, with service and farm workers falling in between. There was a difference in the percents of men and women who had tried to stop smoking and who were successful. Men were more likely than women to try to stop and also more likely to be successful: 80 percent of men who had ever smoked and 75 percent of women had tried to stop smoking, and 41 percent of men and 30 percent of women had actually stopped. Among persons who had ever smoked cigarettes, the percentage of males who had not tried to stop generally declined with age while the percentage of women who had not tried varied little with age.

80–0997. Challberg, K. **Smoking Habits of Rhode Island Physicians.** *Rhode Island Medical Journal* 62(6): 245, June 1979.

A study of the smoking habits of Rhode Island physicians showed that while there was a low percentage of smokers compared to the general population, there were also relatively as many lawyers who were nonsmokers. Only 19.0 percent of physicians and 25.0 percent of lawyers said that they were smokers compared to 44.3 percent of the general male population 25 years of age and over. For the physicians vs. the lawyers, 35.4 vs. 35.8 percent, respectively, had given up smoking, whereas in the general male population ex-smokers made up 30.5 percent of the total. A substantial 45.6 percent of physicians and 39.0 percent of lawyers had never smoked. The study suggests that one explanation for the low prevalence of smoking is the socioeconomic status of these professional groups. Among the physician population, professional specialty appeared to be a factor in whether or not a physician smoked.

80–0998. Dennison, D.; Golaszewski, T.; Klick, R.; Wolfgang, J. **The Heart Health Profile and "The Bogus Pipeline."** *Health Education* 11(1): 33-34, January-February 1980.

A heart health education evaluation instrument was developed to assess secondary level programs. In developing the instrument, self-reported behaviors related to physical activity, diet quality, smoking, and relaxation were monitored for 4 consecutive days. Significant correlations were found between smoking and cardiovascular efficiency in males. There were no significant correlations between biomedical and self-reported data in women. A significant multiple correlation among skinfold measurements, family history, smoking and ingestion of saturated fat was related to serum cholesterol and cardiac efficiency in males, but not in females. The instrument, called Heart Health Profile, yields valid and reliable data on physical activity levels, nutritive information, and smoking frequencies.

80–0999. Fisher, E. B., Jr. **Encouraging Nonsmoking With Positive Incentives.** *World Smoking and Health* 4(3): 25-29, Autumn 1979.

Natural rewards (e.g., fewer health problems), timed cessation, contingency contracts, adolescent smoking prevention, nonsmoking incentives, and employment bonuses are discussed as positive incentives for quitting smoking.

80–1000. Freour, P.; Coudray, P.; Bernadou, M.; Gachie, J.-P.; Labadie, J.-C.; Dones, J.-P.; Saudou, G.-F. **Sondage sur 2.000 Scolaires de la Gironde Pour une Etude de l'Impact de la Campagne Anti-Tabac d'Octobre 1976. [Survey of 2,000 Students of the Gironde Department to Determine the Impact of the October 1976 Anti-Smoking Campaign.]** *Bordeaux Medical* 10(30): 2163-2168, December 1977, French.

Secondary schoolchildren (aged 12-15) were surveyed in 1977 through a questionnaire about their smoking habits and about the impact of a national antismoking campaign conducted from October to November 1976. It was found that 13 to 60 percent of the boys aged 12 to 15 and 7 to 50 percent of the girls in the same age bracket were smoking at least one cigarette daily. Of the children whose parents smoked, 41 percent of the boys and 39 percent of the girls smoked, whereas the children of nonsmokers included 26 vs. 29 percent (male vs. female) smokers. The greatest impact of the antismoking campaign was due to the TV campaign which consisted of 9 20-sec messages at prime time. Rights of nonsmokers, a pregnant woman, smoking and pollution, the cold ashtray, and a child imitating smokers were mentioned by the children as the most impressive TV spots. It is concluded that the impact of the campaign was very favorable due mainly to its presentation on TV.

80–1001. Gilbert, D. G. **Extraversion, Type of Smoker and the Effects of Nicotine on Physiological and Self-Report Measures of Emotion.** Doctoral Dissertation, Florida State University, Tallahassee, University Microfilms 78-15456, 1978, 73 pp.

Although nicotine causes significant increases in physiological arousal, most smokers report that smoking relaxes them. The effect of nicotine on self-reports of emotion and on physiological arousal was studied in 24 male and 24 female students (between 18 and 25 years of age) who reported smoking an average of six or more cigarettes a day. Subjects were classified as introvert or extravert on the basis of the Eysenck Personality Inventory. The students smoked high- or low-nicotine cigarettes before and during the course of emotionally arousing videotapes. Skin conductance, heart rate, and electromyograms were recorded during the videotape viewings. The subjects were then asked to indicate what effect they thought the different cigarettes had on their arousal level and emotional responses. The results indicate that nicotine decreased skin conductance response magnitude, an index of cortical arousal, but did not reduce self-reports of emotion. Introverts and extraverts did not differ in terms of the effect nicotine had on their experienced emotions or on their indices of cortical arousal. In addition, introverts reported smoking more frequently for tranquilization and in high arousal situations than did extraverts.

80–1002. Gilbert, D. G. **Paradoxical Tranquilizing and Emotion-Reducing Effects of Nicotine.** *Psychological Bulletin* 86(4): 643-661, July 1979.

Investigations of the effects of nicotine on emotion and on indices of central and autonomic nervous system arousal are reviewed. Mechanisms that may account for the paradoxical finding that nicotine increases autonomic nervous system end-organ arousal yet frequently reduces self-report and behavioral indices of emotion are evaluated. A number of mechanisms have been proposed, but none are backed by a convincing network of supportive data. The literature suggests that the mechanism(s) by which nicotine reduces indices of emotion is influenced by a wide variety of variables, including behavioral activity level, central nervous system arousal level, type of emotion, time since the administration of the drug, and the rate and dose of its administration. There is the need to demonstrate a paradigm that reliably causes nicotine-induced reductions of emotion. Once reliability has been established, further studies can vary the paradigm's potentially influential variables. (Auth. Abs.)

80–1003. Kawano, M. M. **Community Smoking Education Through School Children.** *World Smoking and Health* 4(3): 38-41, Autumn 1979.

According to 1977 statistics 30 percent of the population in Japan smoked cigarettes. Despite the law prohibiting smoking before the age of 20, nearly one-half of college students and a high percentage of high school students smoked cigarettes. The Japanese Government monopolizes the production and sale of tobacco in Japan. Television advertisements of tobacco are not prohibited and no health warnings are printed on cigarette packages. In June 1978, in Kitakyushu, an industrial city in Japan, there was a demonstration on behalf of the rights of nonsmokers. A booklet containing information about tobacco smoking, increased lung cancer incidence, smoking and illness, smoking as a cause of coronary infarction, smoking-related mortality, nicotine, carbon monoxide, and smoking and pregnancy was published. Copies of the booklet were distributed to schools and public officials. One year after distribution, cigarette sales in Kitakyushu dropped to 94.9 percent of the previous year's total, as compared to 99.8 percent in all Japan.

80–1004. Kucek, P. **Vplyv Fajcenia na Vykon v Senzomotorickej Ulohe. [The Effect of Smoking on Performance of a Sensorimotor Task.]** *Ceskoslovenska Psychologie* 20(1): 55-65, 1976, Czechoslovakian.

A sensorimotor test was administered to a group of 46 healthy male smokers who had smoked an average of 10 to 25 cigarettes/day over a period of 4 to 7 years and to a control group of 32 male nonsmokers. All subjects were 19 to 25 years old. The task consisted of directing a light beam on a target revolving along an irregular elliptical path so that movements of the beam and target coincide. Each time the light beam missed the target, an error was recorded. Each subject was to operate over 15 min (15 revolutions) using his right hand to direct the beam. The group of smokers was given a lighted cigarette to smoke during the 8th to 12th revolutions. An unfavorable effect of smoking on sensorimotor efficiency was reflected in the increased number of errors made by the smokers. The average length of faulty performance during the 8th revolution was a maximum of 33.9 sec for smokers vs. 31.5 sec for controls. However, a decrease in the average length of faulty performance for smokers during the final revolutions supported their subjective appreciation of cigarettes as positive stimulators.

80–1005. Mello, N. K.; Mendelson, J. H.; Sellers, M. L.; Kuehnle, J. C. **Effects of Heroin Self-Administration on Cigarette Smoking.** *Psychopharmacology* 67(1): 45-52, January 1980.

Cigarette smoking increased during heroin self-administration in comparison to drug-free and methadone detoxification conditions in eight heroin addicts given naltrexone placebo ($p<0.01$) and three heroin addicts given buprenorphine placebo. Cigarette smoking was stable across conditions for one subject who did not use heroin during naltrexone blockade of heroin effects. Five subjects smoked significantly more ($p<0.01$) during the hour following a heroin injection than during the preceding hour, and two subjects in the same group smoked significantly less following a heroin injection ($p<0.05$). Subjects smoked significantly more during the evening and night when self-administering heroin than during baseline conditions ($p<0.05$), but subjects did not sleep significantly less during heroin self-administration. The peak of the intercigarette interval distribution remained between 16-30 min during baseline and heroin conditions. However, the increased smoking during heroin use appeared to reflect a higher rate of smoking rather than a generalized increase across intercigarette intervals. (Auth. Abs.)

80–1006. Natvig, H. **Rokevanene hos Ansatte i Bedrifter med Bedriftshelsetjeneste. [The Smoking Habits of Employees of Companies With Company Health Care Service.]** *Tidsskrift for den Norske Laegeforening* (27): 1376-1380, September 30, 1977, Norwegian.

An analysis of smoking habits was made based on questionnaires from 20,000 men and 8,000 women employed in companies with health services. The results indicate that the number of daily smokers in Norway has decreased while the number of ex-smokers has increased. Health was the main reason for giving up smoking for 50 percent of the men and 47.5 percent of the women aged 15-19, and for 64 and 54.6 percent, respectively, of the total group. The percentages of men and women who had started to smoke below the age of 15 were 9 and 5.7 percent, respectively. For the men, 65.5 percent and for the women, 55.5 percent started to smoke at 15-19 years of age; 20.9 percent of the men and 24.4 percent of the women started at 20-24; and 4.6 percent of the men and 14.4 percent of the women started after age 25. The largest percentages of male current smokers and the lowest percentages of ex-smokers were found within the aluminum industry (78.6 and 17.0, respectively), the food industry (66.0 and 15.6), and the foundry and iron alloy industries (65.7 and 19.2). On the other hand, the lowest percentages of male current smokers and largest percentages of ex-smokers were found among individuals with administrative occupations or with office employment (52.1 and 18.8, respectively) and among employees of the construction industry (52.8 and 26.3) and of the railroad, mail, and telephone services (52.9 and 23.3), as well as employees of the electrotechnical and fine mechanical industries (53.3 and 17.0).

80–1007. Orlien, N. **Rokevaner Blant Studentene ved Norges Tekniske Hogskole, Skolearet 1975/1976. [Smoking Habits of the Students at the Norwegian Technical University in the Schoolyear of 1975/1976.]** *Tidsskrift for den Norske Laegeforening* (1): 2-5, 9, January 10, 1977, Norwegian.

The smoking habits of students at the Norwegian Technical University, Trondheim, were examined by means of questionnaires presented to a 20 percent sample during 1975-1976. The response rate was nearly 90 percent based on 644 students. Of the 45 women, 13 percent were smokers and 5 percent ex-smokers. An average of 21 percent of the men were smokers. The percentage of male smokers within the various departments of the university varied from 43 in shipbuilding to 12 in electrical engineering. The percentages of ex-smokers varied between 6 and 17. Students with the highest matriculation marks had the lowest percentage of smokers. Of the male smokers, 20 percent smoked 1-4 g tobacco/day, 46 percent smoked 5-14 g/day, 30 percent smoked 15-24 g/day, and 4 percent smoked 25 g/day or more. Smoking was most commonly started between the ages of 14 and 17. Of the ex-smokers, 46 percent had stopped smoking for health reasons.

80–1008. Wechsler, H.; McFadden, M. **Drinking Among College Students in New England. Extent, Social Correlates and Consequences of Alcohol Use.** *Journal of Studies on Alcohol* 40(11): 969-996, November 1979.

A survey of over 7,000 New England college students revealed that fewer than 5 percent of both men and women abstain from alcohol, that men tend to drink more than women, and that both men and women drink more frequently as they progress in college; high school drinking patterns were more important than any other variable in determining alcohol use in college. Among men, more than 80 percent of those classified as abstainers or infrequent light drinkers reported they never smoked cigarettes, compared with slightly more than half (56 percent) of those classified as frequent heavy drinkers and about two-thirds of other drinkers. In contrast, more than five times as many heavy drinkers as abstainers or infrequent light drinkers (16 vs. 3 percent) said they smoked at least one pack of cigarettes (20 cigarettes) a day. The relationship was even stronger among women. Nearly all women who abstained from alcohol said they never smoked cigarettes, compared with slightly more than one-fourth (28 percent) of the frequent heavy drinkers. Conversely, while 27-30 percent of the heavy drinkers reported they smoked a pack of cigarettes or more each day, this was reported by only 1 percent of the abstainers, 5 percent of the infrequent light drinkers, and about one-sixth of the frequent light and intermediate drinkers. (Auth. Abs. Mod.)

80–1009. Weeks, D. J. **Do Chronic Cigarette Smokers Forget People's Names?** *British Medical Journal* 2(6205): 1627, December 22-29, 1979.

In an effort to determine whether cigarette smoking affects memory, 37 smokers and 37 nonsmokers were given a test which required the subjects to remember the names of unknown persons. The two groups were similar with respect to a number of variables, such as intelligence, perceptual aptitude, age, sex, social class, and educational level. The test score was the number of faces correctly named. Nonsmokers scored significantly higher on the memory test than smokers ($p < 0.005$). The results suggest that chronic cigarette smoking adversely affects memory.

See also, **80–0926, 80–0970, 80–0982, 80–1013**

SMOKING CESSATION METHODS

80–1010. Bauermann, E.; Woeber, K. **Handreichung und Leitfaden zur Raucherentwohnung (fur behandelnde Arzte und zu betreuende Patienten). [Aid and Guide to Smoking Withdrawal (for Practicing Physicians and Patients to be Treated).]** *Rehabilitation* 31(3/4): 31-56, 1978, German.

This article presents recommendations for physicians and guidelines for patients for smoking withdrawal. Before proceeding with any treatment, a physician should advise the patient about the relationship between smoking and potential or actual disease and urge him to stop smoking. To help the patient stop, the physician should determine the type of smoker as the prerequisite to any treatment. Smokers can be divided into four categories: occasional smokers of the "relaxing" type (25 percent), habitual smokers of the stress type (45 percent), psychological or physical addicts (25 percent), and multiple addicts (5 percent) who are alcoholics or drug addicts in addition to smokers. Methods and programs of smoking withdrawal geared to each type of smoker are outlined. The first and part of the second categories of patients can and should be treated in the physician's office, either by individual counseling or group therapy. The last two categories of patients should be confined in a specialized clinic. Reference tests are introduced for the benefit of the first two categories of patients and the physicians. They include the motivation test "Why do I Smoke?" and a behavior test relating to the behavior triangle of habit, desire intensification, and satisfaction values. Samples of corresponding questionnaires are given.

80–1011. Fee, W. **Searching for the Simple Answer to Cure the Smoking Habit.** *Health and Social Service Journal* 87(4529): 292-293, February 18, 1977.

The relative efficacy of various treatment methods was studied in persons attending an antismoking clinic in Scotland between 1964 and 1974. The methods employed included group therapy, chemotherapy, hypnosis, and aversion therapy. From 1964 to 1970, 510 patients were treated, of whom 342 (67 percent) completed treatment. Of these, 249 (73 percent) claimed to have stopped smoking, but only 68 (12 percent) were still abstinent after 6 months. Significantly more men completed treatment and stopped smoking than women. In a comparison of treatment by hypnosis, aversion therapy, drug therapy (fenfluramine), and placebo, successful quitters constituted 66 percent, 57 percent, 24 percent, and 46 percent, respectively. In a trial of combined psychotherapy and chemotherapy (hypnosis and either Avena sativa, nicotine chewing gum, ascorbic acid, or a placebo), between 42 and 53 percent of the patients in each group claimed to have stopped smoking. Overall, group therapy alone provided the best results and chemotherapy alone the worst. In addition, smoker typing (i.e., whether the individual smoked for pleasure or to reduce anxiety or tension) is recommended because the success of aversive and supportive therapies varied, depending on whether the individual smoked for positive or negative effect.

80–1012. Foxx, R. M.; Brown, R. A. **Nicotine Fading and Self-Monitoring for Cigarette Abstinence or Controlled Smoking.** *Journal of Applied Behavior Analysis* 12(1): 111-125, Spring 1979.

This study compared four treatment approaches to cigarette smoking: (1) a nicotine fading procedure, in which subjects changed their cigarette brands each week to ones containing progressively less nicotine and tar; (2) a self-monitoring procedure in which subjects plotted their daily intake of nicotine and tar; (3) a combined nicotine fading/self-monitoring procedure; and (4) a slightly modified American Cancer Society Stop Smoking Program. Thirty-eight habitual smokers (who had been smoking between 1 and 45 years) were assigned to one of the treatment groups. The study had two goals: (1) to achieve a clinically significant percentage of abstinence, and (2) to reduce the nonabstainers' smoking to a "safer" level by having them smoke low tar and nicotine cigarettes. The 18-month followup results showed that the nicotine fading/self-monitoring group was the most successful: 40 percent were abstinent and all who had not quit were smoking cigarettes lower in tar and nicotine than their baseline brands. Half the nonabstainers had decreased their rate of smoking relative to baseline while the other half had increased. Furthermore, the fading/self-monitoring group achieved the largest reductions from baseline in daily nicotine and tar intake (61 and 70 percent, respectively). The results suggest that the study's goals were achieved and that the nonaversive combined procedure could be used to treat not only habitual smokers but also smokers with severe cardiovascular and respiratory problems, because it does not have some of the inherent limitations of the successful aversive smoking cessation procedures. (Auth. Abs.)

80–1013. Rosenbaum, M.; Argon, S. **Locus of Control and Success in Self-Initiated Attempts to Stop Smoking.** *Journal of Clinical Psychology* 35(4): 870-872, October 1979.

The relationship between self-control behavior and individual differences on Rotter's I-E scale was investigated. Subjects ($n = 42$) who were successful in self-initiated attempts to stop smoking showed a more internal locus of control than subjects who were either unsuccessful in their attempts to quit or who never attempted to quit smoking on their own. Further, it was found that subjects who succeeded in quitting were more likely to use abrupt smoking cessation techniques than those who failed in their attempts to quit smoking ($p < 0.01$). The results of this study point out the importance of the locus of control orientation in designing self-control programs for the modification of smoking behavior. (Auth. Abs.)

80–1014. Schwartz, J. L. **Review and Evaluation of Methods of Smoking Cessation, 1969-77. Summary of a Monograph.** *Public Health Reports* 94(6): 558-563, November-December 1979.

Smoking cessation is examined from several viewpoints: the intervention techniques of self-care, medication, and hypnosis; the service packages delivered by nonprofit institutions, medical sponsors, and commercial enterprises; behavior modification techniques; and the mass media and community programs. Each is briefly described.

TOBACCO MANUFACTURING AND PROCESSING

80–1015. Campbell, J. S. **Changing Technology in Tobacco Processing and Manufacturing.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 101-116.

The tobacco industry is currently in a state of dynamic change. It is being pounded by antismoking campaigns, continuously rising taxes, and increasing raw material costs. In its attempt to placate critics, to supply consumers with a reasonably priced and satisfying smoke, and to remain in business and make a profit, the industry is reacting by experimenting in assorted ways. Processing and manufacturing practices are being altered, and there are continual industrial developments (e.g., filters, sheet, freeze-dried and puffed tobacco, and synthetic substitutes). See also 72-1011.

80–1016. Parker, J. A.; Montgomery, R. T. **Design Criteria for Ventilated Filters.** *Beitraege zur Tabakforschung International* 10(1): 1-6, December 1979.

A number of filters were studied to determine the effect of air dilution on smoke filtration, both in well characterized acetate tow items and also in commercial filters that incorporated air dilution. A direct reduction of condensate and nicotine was observed in proportion to the degree of air dilution. Particulate filtration efficiency increased with air dilution. Gas phase delivery of aldehydes and carbon monoxide decreased in greater proportion than the amount of air dilution. The commercial brands tested closely fitted the model developed in the filter tow study, except where mouthpieces or other flow channeling devices altered the filtration characteristics of the cigarette in varying degrees. Significantly, the results of the studies showed that the amount of air dilution, and not the physical means of air mixing, was the defining parameter to predict filter performance. Ventilation gives a useful design capacity that extends control of smoke filtration behavior. By appropriate selection of tow denier per filament, total denier and cross-section shape, filter length and diameter, and the level of air dilution, filter properties can be adjusted to control gas and particulate phase deliveries, and the proportionality between the two. (Auth. Abs.)

80–1017. Reynolds, M. L. **Influence of Filter Additives on Smoke Composition.** In: *Recent Advances in Tobacco Science.* Series 4, Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, New York, 1978, pp. 47-67.

This paper discusses the types of material patented as selective filter additives and assorted negative effects on taste, efficacy, stability, cost, toxicity, manufacturability, and pressure drop; and positive effects on selective removal of nicotine, nicotine enhancement, carbon monoxide oxidation, and nitric oxide removal.

80–1018. Tsoukatos, M. P.; Karageorgos, A. C. **Development of a Radioisotopical Tracing Method to Follow the Flow of Cut Tobacco During its Manufacture.** *Tobacco Science* 21: 61-64, 1977.

A new radioisotopical tracing method in which technetium 99m (^{99m}Tc) is used to follow the flow of cut tobacco in cigarette manufacture is described. The method is simple, accurate, inexpensive, safe to personnel, and facilitates quality improvements and cost of production studies. Usability of treated tobacco is retained. The method has been successfully applied on an industrial scale in determining the degree of distribution homogeneity of a 1 percent mixture of ^{99m}Tc-tagged tobacco, homogeneity of top-flavoring sprayed on cut tobacco, and the percentage of recovered cut tobacco which is lost during recycling in cigarette manufacture. (Auth. Abs.)

TOBACCO ECONOMICS

80–1019. Anschel, K. R.; Brannon, R. H. **Impact of Declining Demand for Tobacco on the Economy of Kentucky.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 207-229.

The production of Burley tobacco makes a very substantial contribution to the economy of the Commonwealth of Kentucky, providing an estimated 550 million dollars in personal income, 50,000 man-years of employment, and 23.5 million dollars in State and local revenues. In terms of the total economy of the State, however, these represent a small fraction (about 5 percent of income and something less of public revenues). Should demand for Burley decline by 15 percent over the next decade, it is anticipated that income and revenues from Burley will decline by about the same proportion. In terms of total State income and tax receipts in 1968 this decline would be small (less than 1 percent). However, tobacco is of major importance in selected counties, providing in 1 county at least 26 percent of real property values and in 19 others 10 percent or more. Burley production may provide directly and indirectly as much as 50 percent of personal income in some counties and 15-30 percent of income in the counties most heavily dependent on Burley. A decline of 15 percent in tobacco income could have serious repercussions for these counties. Income could fall by as much as 7.5 percent and real property assessments by as much as 3.75 percent. In this paper an attempt is made to estimate the role of Burley in the economy of Kentucky and to examine the implications of a possible decline in the demand for tobacco. If local and State governments are to plan adequately for such an eventuality, then more sophisticated studies based on data not now available must be carried out. Of particular import are the resulting changes in income, migration, and public revenues and expenditures. See also 72-1011.

80–1020. Beale, C. L. **Impact of Changing Tobacco Demand and Technology on Population and Migration.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies*. College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 243-250.

In 1960, about 2.8 million people lived in counties showing high ratios of tobacco acreage per total households, thus having implicitly high susceptibility to labor displacement as reductions in tobacco production or labor requirements per acre occurred. Of this population somewhat more than a fifth lived in Burley areas, and the average retention of population in the Burley counties was considerably higher in the 1960's than in the 1950's, apparently because of improved nonagricultural opportunities. Flue-cured areas contrasted in trend with Burley areas by showing slight overall population decline. Outmigrants from the flue-cured areas proved to be disproportionately blacks in the 1960's, with whites experiencing a markedly improved population retention. See also 72-1011.

80–1021. Bordeaux, A. F., Jr.; Hourigan, W. W. **Effects of Government Tobacco Programs.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies*. College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 253-270.

Government tobacco programs have provided price stability for tobacco growers and this has reduced the risk and uncertainty associated with tobacco production. Programs have increased prices above free market levels by combining production controls and high penalties for those who did not comply with these controls. Capitalization of higher prices into allotment values provided windfall gains to original allotment owners and forced later owners to buy allotments at high prices. Tobacco programs have done little to alleviate low returns to resources used in tobacco production, and this continues to be a major problem for many farmers today. The tobacco program offers little promise as a means for improving the returns to resources used on farms in tobacco-producing States. Government treasury costs of tobacco programs are low relative to other major commodity programs since tobacco programs use the market place to pass the costs of the program on to consumers in the form of higher prices. The general public is not subsidizing the consumer of tobacco products to a large extent unless health research expenditures are charged against tobacco. The distribution of benefits from tobacco programs appears to be less regressive than benefit distribution under other government commodity programs, and recent modification in the programs may further improve the distribution of benefits. With the small average size of allotment, the magnitude of benefits received by many producers is far below the amount necessary to provide farm families a decent level of living. See also 72-1011.

80–1022. Bradford, G. L.; Thompson, J. F. **Impact of Eliminating the Tobacco Price-Support Supply-Control Program.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies*. College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 271-293.

In this paper some economic effects of a sudden price support program termination are presented. It is concluded that Burley and flue-cured market prices would decrease by an average equal to the annual allotment value (currently 20-25 cents per pound). Secondly, farmland values would be expected to drop sharply, the exact magnitude of that drop currently estimated to be approximately 20 percent of Kentucky's farmland value and 26 percent of North Carolina's farmland value. Finally, the effects of program termination upon production relocation, allotment consolidation, and farm income and resource-utilization changes are discussed. See also 72-1011.

80–1023. Carley, D. H. **Economic Importance of Tobacco in the National Economy and in the Southern Region.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies*. College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 85-99.

The expenditure sector of the tobacco industry (domestic consumer expenditures plus income from exports), shows an almost uninterrupted increase since 1940. Growth in expenditures was at a rate of about 7.5 percent compounded annually from 1940 to 1954, and about 5.0 percent compounded annually from 1955 to 1969. Taxes on tobacco products accounted for 1.14 percent of total Federal revenue collections in 1969 compared to more than 2.0 percent in the late 1950's and early 1960's. State taxes on tobacco products also represent an important source of tax revenue in many States. Unless there are drastic tax rate increases, total taxes will probably continue at about 40 percent of domestic consumer expenditures for all tobacco products. Employment in manufacturing, excluding stemming and redrying, was about 60,000 in 1967, down from 70,000 or more in earlier years. The largest drop in employment was in cigar manufacturing. The sectors involved with the unmanufactured leaf (production, warehousing, and stemming-redrying) have shown decreases in the share of consumer expenditures. As tobacco prices have increased, and amount of leaf used domestically has decreased, the value of leaf used has remained fairly level. As a consequence, tobacco production has been fairly stable, with the export market absorbing the additional leaf not used for domestic manufacturing. See also 72-1011.

80-1024. Hoff, F. L.; Stovall, J. G. **Impact of Changing Demand for Burley Tobacco on Producers and Resource Owners.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 183-205.

Examination of the Burley industry indicated that a dichotomy exists with respect to the adjustment problems facing Burley growers. One group consists of a large number of older, poorly educated, low income farmers who have few resources and who are predominantly owners of the land they manage. The other group includes the younger, better educated, upper class farmers who have a much larger resource base and are less dependent on tobacco as a major income source. Findings of a previous regional adjustment study showed that the income effects stemming from changes in Burley prices and allotments varied between different production regions. Two factors accounting for these variations in magnitude are (1) the proportion of farm income derived from the sale of Burley tobacco, and (2) the number of alternative enterprises that compete with tobacco for resources. An analysis of Burley operators, landlords, and nonoperator tenants in two areas of Kentucky showed that partnership and part owner operators in Bourbon County, and nonoperator tenants and partnership operators in Metcalfe County were the most dependent on tobacco as the major source of family income. Low income classes were more dependent on tobacco than the high income classes. Finally, a short-run analysis was conducted to determine the income effect that would occur as a result of a price or allotment reduction. Nonoperator tenant families in Metcalfe County had the largest percent decline in family income, while landlord families in both counties had the least decline. Also, the percent reduction was greater for the low income classes, especially in Bourbon County. See also 72-1011.

80-1025. Hoover, D. M. **The Structure of Tobacco Production Units.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 129-149.

In this paper the basic characteristics of tobacco production units with respect to management, labor supply, allotment ownership, and income distribution are described. Also, major changes occurring in the organization of production are surveyed. Attention is devoted to Burley and flue-cured tobacco, and to those areas in which production is concentrated. See also 72-1011.

80-1026. Israel, M. **Tobacco Consumption and Taxation in Various Countries.** *Tabak Journal International* (3): 207-216, June 1979.

Data for the years 1970-1976 on consumption, expenditures, and taxation (in U.S. dollars) are presented for various countries. The information discussed and tabulated includes the following: (1) annual per capita expenditure for tobacco; (2) ratios between total annual expenditure for tobacco and consumption and between total annual expenditure and gross domestic product at cost; (3) number of working hours needed by each individual for his yearly expenditure on tobacco; (4) per capita tobacco tax and ratio of tobacco tax to total revenues; (5) cost of a pack of 20 cigarettes; and (6) number of cigarettes smoked per inhabitant per year.

80-1027. Johnson, P. R. **The Impact on the Economy of a Decline in Demand for Tobacco.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 231-242.

The view taken in this paper is that changes in relative rates of growth impose windfall gains and losses on holders of assets, including human capital. These gains and losses, however, tend to balance out. The effect is on the personal distribution of income, not on the level of national income. There are obvious and clear losers from a leftward shift in the demand function for a particular commodity. There are also obvious and clear gainers when the demand function for a good shifts sharply to the right. These gains and losses accrue to particular individuals. It seems typical that the losers are identified but the gainers are not. Traditionally, even when the losers are clearly identifiable, no policy of compensation has been pursued. The most easily identifiable losers from a leftward shift for tobacco are holders of current production rights. It may be that they will be the recipients of compensation. That is, as pressure from the market forces a change in programs concerning tobacco, these changes will represent a compensation for a decline in value of production rights that comes with more dependence on the market. See also 72-1011.

80–1028. Maxwell, J. C., Jr. **Maxwell Report: Reaching a Plateau.** *Tobacco Reporter* 107(3): 41-43, March 1980.

Cigarette industry estimates indicate that consumption should be up modestly in 1980. In 1979, the trend toward low-tar cigarettes accelerated, and they now constitute more than 42 percent of total volume. Industry sources attribute the increase partly to a statement by an HEW scientist who implied that Carlton cigarettes were not all that bad. The move toward low-tar cigarettes is considered encouraging by the industry as it believes tar to be the main factor contributing to disease in smokers. Some feel that the industry has done such a good job of moving to lower tar (over 90 percent of the cigarettes sold in the United States have less than 20 mg) that antismoking forces no longer need to be quite as adamant.

80–1029. Miller, R. H. **Trends in Tobacco Consumption and Projections to 1980.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 57-82.

If the decline in per capita use continues as in recent years and exports hold their own in an expanding foreign market, by 1980 the U.S. production requirements could be around 1,675 million pounds annually, some 15 percent below 1970. Burley requirements might decline at a slower rate, but Burley production would probably have to be held at the minimum allowed under the poundage program during most of the 1970's to work off excess stocks. Production of fire-cured, dark air-cured, and cigar leaf may decline faster because of declining demand. The projections discussed here are for slightly lower U.S. tobacco production and disappearance by 1980. They assume continuation of the present government tobacco programs. Alternative projections or programs would have different effects for the tobacco producers; the storage, processing, and exporting industries; and the government. Government programs for tobacco affect growers who obtain most of their income from sales of farm products as well as those who earn most of their income from nonfarm sources. Working out the best solution to the problems of these various groups will require concerted effort by Federal and State governments and the people involved. The possibility of a smaller market for cigarette tobacco in the years ahead may spur other parts of the industry to support an expanded or redirected medical research effort just as Kentucky is doing, to resolve smoking and health issues. New developments could affect the cigarette market, such as research results clearing cigarettes of responsibility for health problems, new technology in tobacco growing resulting in a modified product, or manufacturing that would eliminate or substantially reduce allegedly harmful elements. See also 72-1011.

80–1030. Schmidt, F. **Volkswirtschaftliche Aspekte des Rauchens. [National Economic Aspects of Smoking.]** *Das Oeffentliche Gesundheitswesen* 41(10): 724-730, October 1979, German.

A number of antismoking measures are suggested to overcome the social irresponsibility and profit motivation of the tobacco industry and government inertia. The following measures are proposed: (1) prohibition of public advertising of tobacco products; (2) a 10 percent cut in government subsidies for tobacco cultivation in the countries of the European Economic Community and allocation of the amount saved (estimated to be about 60 million German marks) to antismoking education in the schools; (3) a stepwise increase in the tobacco tax, e.g., by 5 percent annually over the next 5 years, as an effective means of checking consumption; (4) a rehabilitation surcharge on cigarettes to defray the cost of smoking-related disease; (5) a general prohibition of smoking at work; and (6) prohibition of research sponsored directly by the tobacco industry. In West Germany, the cost of smoking-related diseases is estimated to be 3-4 billion German marks and the losses in production due to smoking-related diseases and premature death from smoking 8.3 and 9 billion German marks, respectively. These and other statistics make it clear that the total cost of smoking, estimated to be over 30 billion German marks, is much higher than the revenue from tobacco taxes.

80–1031. Shugars, O. K.; Gavett, E. E. **Changing Technology in Tobacco Production.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 167-179.

U.S. farm wages, tobacco price, fertilization practices, and foreign competition are discussed. Farm labor input must be reduced. Harvesting aids can be useful as an interim solution, but new, labor-extensive methods are needed for producing, harvesting, and marketing tobacco. Engineers must be challenged to come up with new ideas, plant breeders with modified plants, marketing people with a modern system, and buyers with a willingness to accept tobacco from such systems. See also 72-1011.

80–1032. Soule, D. M.; Cabaniss, J. S. **Kentucky Cigarette Taxation.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 117-126.

Kentucky is third lowest among the States in special taxation of cigarettes, does not have local rates, and has made few changes during the last 20 years, while the national average of State rates has quadrupled. This paper is concerned with the possible effects of a higher Kentucky cigarette tax on the economy of Kentucky, although some attention is given to the effects of higher tax rates nation wide. Demand elasticity estimates provide very little guidance in estimating changes in cigarette consumption resulting from cigarette price changes in a particular State. Based on the few available estimates, it appears that the price elasticity of demand for cigarettes is sufficiently inelastic to assure that the cigarette tax is a good revenue producer and that higher rates in Kentucky, within the ranges set by other States, will yield increased revenue. See also 72-1011.

80–1033. Stevens, D. J. **Burley Exports: Demand May Strengthen.** *Tobacco Reporter* 107(3): 33-35, March 1980.

Burley exports in the 1979/80 crop year are projected to increase slightly from the expected record level of 1978/79. It is estimated that shipments in the October 1978-September 1979 period totaled over 120 million pounds on a farm-sales-weight basis, and were valued at over 200 million dollars. The continued worldwide growth of blended cigarette output and the declining value of the U.S. dollar have been instrumental in maintaining U.S. exports.

80–1034. Stovall, J. C. **An Overview of Social and Economic Issues Confronting the Tobacco Industry in the Seventies.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 3-13.

The tobacco industry is an important sector of the U.S. economy. Total expenditures for tobacco products exceed 10 billion dollars annually. Federal, State, and local tax revenues from tobacco products totaled 4.5 billion dollars in 1970. In the tobacco growing regions, tobacco often represents the major farm income source. Some 330,000 farmers and many other landowners and farm laborers derive income from tobacco production. In Kentucky and North Carolina, tobacco makes up about one-third of all gross farm receipts. See also 72-1011.

80–1035. Tobacco International. **Flue-Cured Program Has the Votes for Survival.** *Tobacco International* 181(26): 112, 117, December 28, 1979.

An overwhelming number of growers (97.8 percent) approved the quota program for flue-cured tobacco, 1980 through 1982. The 1980 flue-cured quota calls for 977 million pounds with 85 million pounds of carry-forward from the 1979 crop. The effective quota will be the same for both years. Price supports will be available at a level about 9 percent higher than 1979 crop support prices, which averaged 129.30 dollars per 100 pounds.

80–1036. Tobacco International. **Canadian Industry Looks to Lower Tar Deadline.** *Tobacco International* 181(26): 81, December 28, 1979.

Member companies of the Canadian Tobacco Manufacturers' Council indicate that they are on target in complying with a Health and Welfare Canada mandate which would result in an industry sales weighted average of 12 mg of "tar" delivery per cigarette by January 1, 1985. The sales weighted "tar" average for Canadian cigarettes has been reduced from 16.96 mg at the end of 1973 to 14.43 mg in June 1979—a drop of 14.92 percent. The percentage of market held by cigarette brands delivering 12 mg of "tar" or less has risen from a share of 8.3 percent in 1973 to one of 23 percent in June 1979 while cigarette brands delivering 19 mg or more of "tar" had a 30.4 percent market share in 1973 and currently hold just 1.8 percent.

80–1037. Tobacco Reporter. **Dominican Tobacco: Export Prices Are Good.** *Tobacco Reporter* 107(3): 38-39, March 1980.

Tobacco production for 1979 in the Dominican Republic is estimated at 48,350 metric tons, up almost 3 percent from previous reports. The forecast for 1980 is tentatively set at 43,750 metric tons which could represent a 6 percent reduction from 1979 levels, if correct. Total tobacco leaf imports during 1979 rose to 1,500 metric tons while exports increased by 1.3 percent from 1978 to an estimated 37,380 metric tons. The Tobacco Instituto has submitted to the Government of the Dominican Republic a project to curtail imports gradually over the next 5 years. In order to accomplish this, the Dominican government would probably have to increase the taxes on cigarette production as a trade-off for the lost revenues formerly raised by customs duties on imported tobaccos.

80–1038. Voland, M. E. **Socio-Economic Characteristics of Tobacco Producers.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 151-166.

Farm operators who raise tobacco in Kentucky generally resemble farm operators in the State. Three-fourths of the farms also have nonfarm sources of income. Since the average farm size in Kentucky is only about 120 acres, a large proportion of these moderately sized farms cannot be expected to give significantly higher returns. Educational levels of farm operators are generally lower than those of their counterparts in the metropolitan sector, ranging from a 3-year difference for younger groups (under 35) to only 1 year for the oldest groups (over 65). By tradition, farm operators are generally conservative. This factor, combined with low incomes, presents characteristics of farmers who will generally resist innovations. Considering the possibility of farm operators migrating to employment and making successful adjustments to new modes of employment in new community settings, gloomy results are found. Without a great deal of support in terms of training, mobility assistance, and counseling and support in the new community, the rate of failure is extremely high. The adjustment of the farmers' children to the industrial labor market presents similarly gloomy prospects. Educationally, the rural youngster is not at the same level as his urban counterpart; at any given grade level, the attitudes of rural-reared young people may prove to be a stumbling block to adjustments as they move to urban centers. Their occupational aspirations are generally less realistic than their urban counterparts, and their attitudes toward continued education and training for employability are less favorable. See also 72-1011.

BILLS AND LEGISLATION

80–1039. Brody, A.; Brody, B. **The Legal Rights of Nonsmokers.** New York, New York, Avon Books, 1977, 228 pp.

Smoking threatens the well-being of the nonsmoker as well as the smoker. This guide for the layman demonstrates both the health hazards and the costs inflicted upon nonsmokers, and then presents existing and suggested methods of reducing these threats by lawsuits, statutes, and nonlegislative means. Special features of this guide are a State-by-State listing of statutes restricting smoking, and a selection of Federal and State regulations and local ordinances limiting smoking. Also included are lists of nonsmokers' and major antismoking organizations.

80–1040. U.S. House of Representatives, 95th Congress, 2nd Session. **Economic Impact of Tobacco Program. Hearing Before the Subcommittee on Tobacco of the Committee on Agriculture.** U.S. House of Representatives, Serial No. 95-UUU, 1978, 101 pp.

Various persons, including businessmen and U.S. Representatives and Senators, have spoken here concerning the economic impact of terminating the tobacco price support program on the U.S. economy. The tobacco-growing States, total tobacco acreage, and tobacco farm labor are discussed.

80–1041. U.S. Supreme Court. **Federal Employees for Non-Smokers' Rights, *et al.*, Petitioners, vs. United States of America, *et al.*, Respondents. Petition for a Writ of Certiorari to the United States Court of Appeals for the District of Columbia Circuit.** U.S. Supreme Court, No. 79-147, October Term, 1979, 52 pp.

The present case raises significant Federal questions where resolution will have an extremely broad impact. First, the case concerns the rights of over three million Federal employees to smoke-free workplaces. In deciding this question, the Court will determine for the first time whether Federal Government employees may bring similar actions to ensure the safety and health of their workplaces from a variety of hazards. Second, if upon remand it is held that there is a Federal common law right to a smoke-free workplace, the impact is likely to be felt in the private sector. Common law injunctive actions in State court may be brought or there may be State or Federal rulemaking to protect the rights of nonsmokers. Third, in deciding whether an action for injunctive relief is implied in the Occupational Safety and Health Act of 1970 (OSHA), the Court's decision will affect other cases in which other implied rights and remedies under OSHA are alleged. Finally, in ruling on the present case, the Court will address the wider issue of how broadly the courts should act to uphold the congressional mandate of a safe workplace and a safe environment.

GENERAL

80–1042. American Automatic Merchandiser. **Tobacco Action Network Meets Anti-Smoking Activism Challenge.** *American Automatic Merchandiser* 21(12): 28, 30, 32, December 1979.

To help combat antismoking activism, the Tobacco Institute has mounted a grass roots effort under the umbrella of the Tobacco Action Network (TAN). TAN is alarmed over the spread of antismoking legislation and the strategy changes of antismoking activists. In particular, social stigmatization is being used to make smoking unpopular and the smoker unwanted and scorned. One extremist group, Society for Mortification and Smoker Humiliation (SMASH), has suggested planting bits of explosives in cigarettes and using a spray to make smokers nauseous. TAN volunteers number 12,000 nationally in 13 States. The major role of a TAN volunteer is to provide an alert when restrictive smoking or punitive taxation legislation is proposed.

80–1043. Brailey, A. G., Jr. **The Promotion of Health Through Health Insurance.** *New England Journal of Medicine* 302(1): 51-52, January 3, 1980.

Public awareness of the association of hypertension, cigarette smoking, and elevated serum cholesterol with coronary disease has increased markedly, as has the view that one has personal responsibility for one's health. Insurance companies also play an important role in health promotion. Approximately 40 life insurance companies now offer life insurance to nonsmokers at lower than standard rates, 10 offer lower rates to nonsmokers of standard build only, and 3 offer discount rates for "regular smokers." However, there have been no similar initiatives among health insurance companies.

80–1044. British Medical Journal. **Medical Charities and Prevention.** *British Medical Journal* 2(6205): 1610, December 22-29, 1979.

Efforts at preventing cardiovascular disease (e.g., reducing cigarette consumption) receive lower priority than efforts at curing in Britain as compared with many other developed countries. In 1978, 800,000 pounds were spent for establishment and maintenance of chairs in cardiovascular disease, and a similar amount was spent on grants for specific research projects. Less than 4 percent of these grants have been for epidemiological or preventive studies. However, the American, Australian, Canadian, Dutch, Danish, Irish, French, and New Zealand Heart Foundations spent amounts ranging from 160,000 to 44 million pounds on health education (e.g., smoking and lung cancer) and prevention.

80–1045. Christen, A. G. **Tobacco Chewing and Snuff Dipping. (Letter).** *New England Journal of Medicine* 302(14): 818, April 3, 1980.

Physicians should be aware that snuff dipping and tobacco chewing are increasing among U.S. youth, particularly in the South. The tobacco industry has launched an intensive advertising campaign using well-known sports personalities to promote smokeless tobacco. In view of the harmful changes produced by smokeless tobacco in the hard and soft tissues of the mouth, physicians are urged to take a strong public stand against this advertising campaign aimed at young people.

80–1046. Doyle, N. C. **Smoking—A Habit That Should be Broken.** Public Affairs Committee, Inc., Pamphlet No. 573, New York, September 1979, 28 pp.

This pamphlet on smoking is one of a series published by the Public Affairs Committee to educate the American public on vital economic and social problems. The topics discussed include (1) why teenagers smoke, (2) special problems of female smokers, (3) protecting nonsmokers, (4) smoking at work, (5) costs of smoking, (6) tar and nicotine levels, (7) trying to quit, and (8) not starting at all.

80–1047. Dugan, W. M., Jr. **Help Your Patients Quit Smoking.** *Journal of the Indiana State Medical Association* 71(7): 702, July 1978.

Physicians are the key influence in persuading patients to quit smoking. A recent Gallup poll showed that 71 percent of patients who smoked a pack a day or more would quit smoking cigarettes if their physicians urged them to do so. Using these findings, an American Cancer Society official urges physicians to follow the Society's three-point program: (1) urge all patients who smoke to quit; (2) give your patients the assistance they need; and (3) follow up to help your patients remain abstinent. The Society has a number of aids available for physicians to help their patients stop smoking, including the Help Quit Kit.

80–1048. Elovainio, L. **Kansanterveysjarjestot ja Tupakoinnin Vahentaminen. [National Health Organizations and the Lowering of Smoking Frequency.]** *Sosiaalilaaketieteellinen Aikakauslehti* (3/4): 213-214, 1977, Finnish.

Although national health organizations have strived to decrease tobacco consumption in Finland for 30 years, the collaboration between authorities and the national health organizations has only recently become intensified. The larger organizations have formed unofficial collaboration groups that have contacted the decision makers and the mass media. In order for such collaboration to become efficient, the authorities should make long-term plans for antitobacco activities and inform the national health organizations about such plans at an early point in order to enable them to coordinate their plans accordingly. In its proposal the tobacco political action group defined the contribution that national health organizations can provide in health education for reduced tobacco consumption. The health organizations should set up the antismoking campaign programs and produce support material.

80–1049. Golf, S. **Somatisk Sykelighet hos Psykiatriske Langtidspasienter. [Somatic Diseases in Long-Term Psychiatric Patients.]** *Tidsskrift for den Norske Laegeforening* (30): 1559-1561, October 30, 1977, Norwegian.

In order to clarify the frequency of somatic diseases in long-term psychiatric patients, 78 male patients aged 45-89 who had spent more than 10 years in a psychiatric clinic were examined clinically. Additional information was obtained from hospital records and through interviews of the permanent staff and the patients themselves. Among the patients, 68 percent were nonsmokers, 13.2 percent smoked less than 10 cigarettes per day, 15.8 percent smoked 10-20 cigarettes per day, and 2.6 percent smoked more than 20 cigarettes per day. The low prevalence of somatic diseases, especially ulcer, suggested a lack of disease-provoking factors in the institutional environment. The low tobacco consumption is probably one important reason for the almost complete absence of ulcer and the fact that no patients had asthma or chronic obstructive lung disease. The low prevalence of coronary heart disease is probably a reflection of the low tobacco consumption as well as the lower than average occurrence of obesity.

80–1050. Gooding, L.; Hillhouse, A. **Nonsmoking Campaigns in England and Scotland.** *World Smoking and Health* 4(3): 14-17, Autumn 1979.

Action on Smoking and Health campaigns in England and Scotland are briefly described. Their focus is on reduction of public smoking (e.g., food shops, cinemas, railways, airlines). Models for nonsmoking action are similar for the two countries, and include such steps as problem areas, public opinion surveys, publicity, and identification of those with the power to bring about change.

80–1051. Hamburg, D. A. **Disease Prevention: The Challenge of the Future. Sixth Annual Matthew B. Rosenhaus Lecture.** *American Journal of Public Health* 69(10): 1026-1033, October 1979.

This report contains a general discussion of public health achievements and directions in disease prevention across the world. Considered are population growth, cardiovascular disease, smoking, cancer, adolescence, and health and behavioral research.

80–1052. Health Insurance Institute. **Health and Health Insurance: The Public's View.** Health Insurance Institute, Washington, D.C., 1979, 45 pp.

This booklet is the second annual Health Insurance Institute report on public attitudes toward the health care system. The report is based on a sample survey of 1,508 persons, representative of U.S. adults, in May and June of 1978. Among the findings were (1) 67 percent of Americans feel they take good care of their health, e.g., 60 percent either smoke less than a pack of cigarettes a day or do not smoke at all; (2) about 8 in 10 Americans are satisfied with the quality of their health care, but only 29 percent with its cost; and (3) 40 percent of the public expressed satisfaction with their health insurance, a decline from 46 percent in 1977.

80–1053. Hockett, R. C. **The Tobacco Health Issue: An Overview of Medical Research.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 29-56.

An overview of the tobacco and health issue is presented by describing where the Council for Tobacco Research is placing the emphasis in its program and why, since this involves implicit judgments as to the validity and significance of the various observations that have given rise to present widespread attitudes on the subject. The present attitudes toward the tobacco-health issue have such momentum, and the progress of sound and methodical research is so slow, that it is expected that the situation for tobacco producers and processors will worsen before it improves. As a better understanding of the origins of the constitutional diseases is gained, there will be a progressive attenuation of the claims that cigarettes are a major health hazard. See also 72-1011.

80–1054. Holbrook, J. H. **Tobacco Smoking.** In: Isselbacher, K. J.; Adams, R. D.; Braunwald, E.; Petersdorf, R. G.; Wilson, J. D. (Editors). *Harrison's Principles of Internal Medicine. Ninth Edition.* New York, McGraw-Hill Book Company, 1979, pp. 938-941.

This chapter contains information on a wide range of topics related to smoking. Included are discussions of cigarette smoke (e.g., constituents), pharmacology (e.g., nicotine, carbon monoxide), epidemiology, characteristics of smokers, clinical disease correlations (e.g., cardiovascular disease, cancer, respiratory disease), pregnancy, passive smoking, types of tobacco smoking, and cessation of smoking. It is stated that premature coronary heart disease (CHD) is one of the most important consequences of cigarette smoking, the risk of fatal or nonfatal CHD being 60-70 percent higher in male smokers than nonsmokers, and the risk of sudden death being 2-3 times more likely in 35- to 54-year-old male smokers than in nonsmokers.

80–1055. Isacsson, S.-O. **Rokning och Barnens Miljo—Ett Problem for Modravard, Barnavard, Skolan. [Smoking and the Environment of the Child—A Problem of Maternity Care, Pediatric Care, and the School System.]** *Lakartidningen* 74(19): 1917-1926, 1977, Swedish.

A 1976 symposium concerning smoking and the child's environment is discussed and the following topics are presented: smoking and pregnancy, smoking and the child's environment, smoking and respiratory tract diseases in children, and smoke components and their effects on the body. In the discussion following the symposium several new findings were presented. The experiences of an antismoking clinic serving the schools in Stockholm for 6 years were discouraging due to high costs and low effectiveness. The clinic's activities are now being decentralized to take place within the individual schools. Teachers, parents, and trained staff will work together. In Norway in 1975 all the children aged 12-15 (200,000) were interviewed in their schools about their smoking habits. In families where both parents smoked and allowed their children to smoke, more than 70 percent of the 9th graders (15-year-olds) were daily smokers. When neither parent smoked and the children were not allowed to smoke, less than 10 percent of such children smoked. The deciding factor was not the example set, but whether permission was given or not. A study at a maternity ward in Sweden showed that it was possible to motivate new mothers to stop smoking. The central role of pediatricians was emphasized, as they are able to spread information about smoking through hospitals, pediatric centers, day care centers, preschools, and schools. It was suggested that various means for prohibition of smoking in hospitals should be attempted.

80–1056. Kekki, P. **Kokemuksia Keski-Ikaisten Miesten Seulontatutkimuksesta. [Results of Health Examinations of Middle-Aged Men.]** *Suomen Laakarilehti* (23): 1759-1762, August 10, 1978, Finnish.

Long-term improvement of the health level of the population can be achieved through periodic health examinations of selected groups of the population. The results from one such examination of 487 males aged 45-54 are presented. The participants represented 54.4 percent of the individuals invited within a health care service area. An investigation of smoking habits by means of questionnaires showed that 26 percent of the participants were nonsmokers, 30 percent ex-smokers, and 44 percent current smokers. Of the smokers, 17 percent smoked 15-19 cigarettes per day, and 45.9 percent smoked 20 or more cigarettes per day. Obesity was found to be more common among smokers than nonsmokers; 59 smokers were 6-10 kg overweight as compared to 39 nonsmokers; 40 smokers and 24 nonsmokers were 11-15 kg overweight; and 66 smokers and 23 nonsmokers were more than 16 kg overweight. Of the severely obese (20 kg or more overweight), 40 percent had a diastolic blood pressure $\geq$ 105 mmHg, compared to only 11 percent of the entire group. A comparison made between farmers and other workers with elevated blood pressure without considering obesity revealed that 21 percent of the farm workers and 29 percent of other workers had elevated blood pressure. Hypertension was more common in nonsmokers than in smokers, both among farmers (14 vs. 7 percent) and among other workers (18 vs. 10 percent).

80–1057. Kritskii, V. N. **Kurit' Vredno. [Smoking is Unhealthy.]** *Meditsinskaya Sestra* 37(2): 44-47, February 1978, Russian.

Data on the history and health effects of smoking are presented. The adverse effects of smoking on the central nervous system, cardiovascular system, gastrointestinal tract, and respiratory organs are discussed. The mortality from myocardial infarction in the 40-50 year age group is estimated to be three times higher in smokers than in nonsmokers. Among the persons suffering from tuberculosis of the respiratory system, 95 percent are smokers. Lung cancer incidence is 15 to 20 times higher in smokers than in nonsmokers, with 96 percent of lung cancer patients being long-term smokers. The detrimental effect of smoking on women, fetuses and infants is emphasized. A shortened life span and premature aging are also among the effects of smoking. The unhealthy effect of passive smoking results from an estimated 2 l of smoke from a single cigarette, which contains up to 6,000 soot particles/cm^3. It is recommended that people quit smoking entirely. Mouth washing with 1 percent silver nitrate before smoking, taking "Tabex" or "Lobesil" tablets, and, most effectively, subcutaneous injections of 1 percent lobelin hydrochloride are recommended for those who cannot quit smoking at once.

80–1058. Miller, R. R.; Fox, E.R.W. **On Smoking and Advertising. (Letters).** *Western Journal of Medicine* 131(4): 334, October 1979.

In an editorial rebuttal, a director of media relations for the Tobacco Institute refutes charges that advertising seeks new young customers and is responsible for the teenage smoking problem. The director states that the aim of cigarette advertising is to maintain or increase brand loyalties in established smokers, not to entice new smokers. The response of the author of the original editorial reiterates the view that the advertising message is clear: (1) smoking is glamorous, (2) smoking is to enjoy gracious living, (3) smoking is sexy, and (4) smoking is charismatic. Further, the original author states that the antismoking campaign must be stepped up, and that the print media will hopefully follow the examples of TV and radio in carrying no cigarette ads.

80–1059. Morrison, A. B. **Prevention in the Canadian Health System.** *Preventive Medicine* 7(4): 498-504, 1978.

A major reason for encouraging and facilitating coordinated program planning and implementation is the shared responsibility for health and well-being among governments, public and private agencies, business, and industry. In Canada, the provisions of health care services have been acknowledged as primarily a responsibility of provincial government. The use of alcohol and tobacco products is an area of major importance to disease prevention in Canada. A cross-Canada campaign, the National Education Week on Smoking, is intended to increase awareness among smokers and the general population about the serious health consequences of tobacco use and exposure to tobacco smoke. It is sponsored by the Canadian Council on Smoking and Health with federal cooperation and assistance. Over 10 percent fewer Canadians now smoke than did so a decade ago. Tar and nicotine levels in cigarettes have decreased by one-third during the last 10 years. Mechanisms for cooperation in health matters among levels of government have improved significantly over the past 3 years, as development of a system of federal-provincial advisory committees has enabled improved coordination and integration of efforts. Important advances have also been made in government-industry liaison based on the premise that neither industry nor government has a monopoly on competence or integrity. (Auth. Abs. Mod.)

80–1060. National Cancer Institute; National Heart, Lung, and Blood Institute. **Smoking and Health. A Program to Reduce the Risk of Disease in Smokers. Status Report—December 1978.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, and National Heart, Lung, and Blood Institute, December 1978, 110 pp.

This report summarizes the status of the Smoking and Health Program, with emphasis on activities conducted during the past calendar year (January through December 1978). The report starts with a historical background that describes the environment within which the program evolved, and then presents a general summary of the health consequences of smoking. Details of the program itself are then described, including the current status of research activities, a summary of selected accomplishments, and a description of program funding. Among the program accomplishments are identification of individuals at high risk, methods of smoking intervention, and development of less toxic smoking products.

80–1061. Paarlberg, D.; Hathaway, D. E.; Marshall, J. P. **Reactions to a Policy Prescription for Tobacco in the Seventies.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 319-332.

80–1062. Rimpela, M. **Fran Forskning till Intervention—mot en Halsoinriktad Tobakspolitik. [From Research to Intervention—Toward Tobacco Politics Focusing on Health.]** *Nordisk Medicin* 92(3): 73-78, 1977, Swedish.

The authors offer their views on tobacco industry policies of the 1970's. The potential loss of government subsidies and the free market system are discussed in relation to crop migration, manufacturing and processing costs, and rural populations. See also 72-1011.

The problem of smoking serves as a typical example of how new health problems are discovered and attacked. It is useful to study why it has taken such a long time before efficient social action has been taken against smoking. This analysis of Finnish and Swedish tobacco politics shows that the interests of the tobacco industry have been favored by the medical world, the authorities, and the legal system It is assumed that the tobacco industry will continue growing until the year 2000. It has not been threatened by either medical research or health education. Attempts by the health authorities to initiate legislation about health-promoting tobacco politics have been opposed by financial and industrial authorities.

80–1063. Rudd, R. W. **A Policy Prescription for Tobacco in the Seventies.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 295-317.

Contradictions in government tobacco policies are briefly discussed. Also considered are topics relevant to tobacco production in the 1970's including expected demand, cigarette tobacco program experiences, cotton and tobacco program similarities, dark tobacco experiences, marketing, poundage regulation, price support, tobacco-limiting easements, export subsidy removal, and intercounty tobacco allotment leasing. See also 72-1011.

80–1064. Seltzer, C. C. **Constitutional and Genetic Factors in Relation to Tobacco Smoking.** In: Bordeaux, A. F., Jr.; Brannon, R. H. (Editors). *Social and Economic Issues Confronting the Tobacco Industry in the Seventies.* College of Agriculture and Center for Developmental Change, University of Kentucky, Lexington, February 1972, pp. 17-27.

The extent of the influence and the relative role (e.g., causal or contributory, great or small, direct or indirect, separate or joint) of genetic factors in the totality of smoking behavior and disease need to be known. Further, a presentation of the constitutional genetic and other nonsmoking environmental factors in the form of risks or competitive risks relative to health and disease, as has been the case for smoking, is needed. Only when sufficient knowledge of the host factors associated with smokers and nonsmokers is accumulated, can the role of smoking in health and disease be assessed. See also 72-1011.

80–1065. Tobacco International. **FTC Asking For Anti-Smoking Ads.** *Tobacco International* 181(26): 120, December 28, 1979.

The Federal Trade Commission has recommended that Congress consider spending funds for antismoking messages to be carried in the news and broadcast media. In its annual report to Congress, the Commission noted that the antismoking messages that were broadcast between 1967 and 1970 made the public aware of the health consequences of smoking. These messages were dropped after broadcast commercials for cigarettes were banned in 1970.

80–1066. World Tobacco. **Selling Cigarettes After Advertising is Outlawed.** *World Tobacco* (65): 79-80, 82-83, 85, 87, July 1979.

In most of the countries in which a total ban on advertising has already been imposed (e.g., Italy, Norway, Finland) and in countries with various restrictions (e.g., in print media, posters, and cinemas), new tactics have been used by the tobacco industry to market its products. The industry is attempting to establish brand success and smoker loyalty. Promotional techniques such as umbrellas, T-shirts, recipe books, and leather goods are among those increasingly employed in countries with tobacco advertising restrictions.

AGRICULTURE

80–1067. Karagezov, G. S. **Sroki Posadki: Ikh Vliicanie na Urozhai i Kachestvo Tabaka. [Planting Times: Their Influence on Yield and Quality of Tobacco.]** *Tabak* (4): 61-62, 1978, Russian.

Experimental planting of Trapezond 285 tobacco had been carried out on a grade testing plot in Eastern Georgia to optimize planting time with respect to yield and quality. Chemical analysis of the sun-dried tobacco showed that total nitrogen, nicotine, and protein contents increased in late-planted tobacco. The nicotine content of the dry tobacco planted between April 10 and May 10 varied from 2.13 to 2.41 percent, while that of tobacco planted between May 20 and June 10 was 2.52 to 3.10 percent. Concurrently, carbohydrate content of the dry tobacco decreased from 5.34-6.60 to 3.72-4.71 percent. Flavor and taste, as well as the other factors affecting yield and quality of the raw tobacco, decreased in tobacco planted at a late date.

80–1068. Mateeva, A.; Dirimanov, M.; Stefanov, D.; Dimitrov, At. **Fiziologo-Biologicheska Sushtnost na Izmeneniyata v Tyutyunevila List Prichineni ot Smucheshti Nepriiateli i ot Preparati. [Physiological and Biochemical Changes Caused in Tobacco Leaves by Pests and Preparations.]** *Bulgarski Tiutiun* 22(9): 37-42, 1977, Bulgarian.

The efficiency of four insecticides (Pirimor 50, Sayfos DP-70, Orthene, and Hex 1901) in tobacco plant louse control, and the effect of these preparations on enzyme activity, chlorophyll A and B levels, and carotenoid content of tobacco plants were determined. The first treatment was made about 1 month after planting. Samples for analysis were taken at 12 hours, and at 4, 8, and 15 days after each treatment during leafing and during the next stage of 10-12 leaves development. All of the insecticides exhibited 96-99.7 percent efficiency in controlling plant lice 5 days after treatment. Variations in peroxidase and catalase activities occurred in both stages of plant development after treatment with each insecticide. The chlorophyll and carotenoid content during the leafing stage increased following insecticide treatment, with the greatest increase of 15 to 20 percent occurring 15 days after treatment. Treatment at the 10-12 leaves stage resulted in a 25 percent increase in chlorophyll and carotenoid levels on the 8th day after treatment, and in stabilization of the increase on the 15th day.

80–1069. Menser, H. A.; Chaplin, J. F. **Ozone Responses of Pennsylvania Seedleaf Tobacco Cultivars.** *Tobacco International* 182(4): 69-72, February 22, 1980.

Several cultivars of Pennsylvania Seedleaf (Type 41) cigar-filler tobacco were tested in an exposure chamber for tolerance to photochemical air pollution, primarily ozone (O_3). Controlled dosages of O_3 (10, 15, 20, and 25 pphm by volume) for 2 hours at 25°C, 60 ± 5 percent relative humidity, and 22.2 klux (fluorescent CW) caused the least leaf injury to Pennbel-69 and Pennleaf-1, and the most injury to Red Rose. The O_3 tolerances of Greider, Holbein, and Swarr-Hibshman were not appreciably different, and were intermediate between the least and most sensitive cultivars. Injury ranged from as little as 2 percent at 10 pphm O_3 to 50 percent at 25 pphm O_3. The relative sensitivity of cultivars to oxidants in field studies was similar to O_3 sensitivity in controlled exposures. Estimated oxidant injury to fleck-sensitive wrapper selection Bel-C grown in the field was 40 percent in 1970 and 55 percent in 1971; injury to Red Rose was 15 percent in 1970 and 38 percent in 1971. When fertilization was increased from 2,240 to 4,480 kg/hectare of 4-8-12, weather fleck injury decreased from approximately 55 to 30 percent in Bel-C and from 25 to 10 percent in Greider and Swarr-Hibshman, but remained at about 25 and 35 percent, respectively, in Holbein and Red Rose. Nitrogen components concerned with leaf utilization did not seem to be measurably affected by weather fleck, perhaps because leaf injury was not extensive. Fertilization rates were the apparent cause of differences in the levels of total nitrogen (N), protein N, NO_3- N, NH_2- N, and nicotine. Current levels of fleck tolerance in Type 41 tobacco seem adequate based on field observations within the production area. (Auth. Abs.)

80–1070. Rosa, N. **Sucker Control Chemicals Commonly Used in Ontario, 1967-1976.** *Tobacco International* 182(3): 54-56, February 8, 1980.

A number of sucker control chemical formulations for inhibiting axillary bud growth in tobacco have been evaluated. The fatty acid esters and the C8 and C10 fatty alcohols were the most effective in control of sucker growth. Generally, contact-type chemicals resulted in increased yields through reduced sucker development, increased levels of reducing sugars, and decreased levels of total alkaloids. Other agronomic and physical parameters showed some changes as a result of treatment, but these changes were minimal. (Auth. Abs.)

80–1071. Sheen, S. J.; DeJong, D. W.; Chaplin, J. F. **Polyphenol Accumulation in Chlorophyll Mutants of Tobacco Under Two Cultural Practices.** *Beitraege zur Tabakforschung International* 10(1): 57-64, December 1979.

Quantitative variation in plastid pigments and polyphenols during leaf growth and after curing have been studied with several chlorophyll-deficient genotypes of tobacco. Under culture for flue-cured tobacco, the chlorophyll-deficient pale-yellow (NC95-Py) and yellow-green (SC58-yg) lines did not differ in polyphenol accumulation from the corresponding normal green recurrent parents NC95 and SC58. A negative correlation was evident between concentrations of polyphenol and chlorophyll. Decrease of the latter during leaf growth was accompanied with a decline of polyphenoloxidase and peroxidase activities. When the chlorophyll-deficient genotypes were grown under conditions for Burley tobacco, concentrations of chlorophyll and polyphenol maintained a steady level and were positively correlated, while the oxidases tended to increase. Burley 21 contained the lowest amount of polyphenols among the chlorophyll mutants, although the low chlorophyll content of its mature leaves was comparable to that of SC58-yg. Use of cultural practices for flue-cured tobacco caused an accumulation of polyphenols in green leaves. There was a greater loss of soluble polyphenols during air curing than during flue curing. Results indicate that the interaction of cultural practices with chlorophyll-deficient genes affects the metabolism of chlorophylls and polyphenols during leaf growth and maturation. Introduction of these genes into tobacco cultivars may provide a means of enhancing uniformity of leaf maturity and modifying certain leaf chemical constituents. (Auth. Abs.)

80–1072. Tokarz, S. **Wplyw Nawozow Magnezowych na Plon i Niektore Cechy Jakosciowe Tytoniu. Czesc I. Doswiadczenia Wazonowe. [Effect of Magnesium Fertilizer on Yield and Certain Characteristics of Tobacco Quality. Part I. Vegetational Experiments.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* (1/2): 5-27, 1977, Polish.

Vegetational experiments were carried out in 1973 and 1974 to explore the effect of magnesium fertilizers on the yield and chemical composition of light yellow cigarette tobacco from Virginia Izyda variety. Magnesium sulfate, potassium magnesium sulfate, polyhalite, magnesium carbonate-lime, magnesium silicate-lime, and calcined dolomite were applied. The last three fertilizers were used in quantities calculated to obtain 0.5, 1.0, and 1.5 Hh (hydrolytic acidity). The soils at pH 5.70 and 5.50 containing 1.20 and 1.47 mg/100 g soil, respectively, were used in the experiments. It is shown that magnesium fertilizers contributed to an increase in tobacco leaf size and accelerated tobacco flowering by 3-6 days. Leaf yield increased significantly after application of magnesium silicate-lime fertilizer at 0.5 Hh. Only a slight yield increase was noted after application of other magnesium fertilizers. Also, magnesium fertilizers were shown to decrease protein and total nitrogen contents of the leaves, particularly of the lower and upper leaves. The decrease in protein and total nitrogen was more pronounced at the lowest dose (0.5 Hh) than at the highest dose of magnesium lime fertilizers. At the same time, potassium content of leaves decreased, while magnesium content increased.

80–1073. Umamaheswara Rao, M.; Patel, U. R. **A Note on the Effect of NPK Fertilisation on the Performance of Two Flue-Cured Virginia Tobacco Varieties Under Gujarat Conditions.** *Tobacco Research* 5(2): 140-142, December 1979.

In order to determine optimum fertilizer requirements and suitable varieties for improved yield and quality of Indian cigarette tobacco, three levels of nitrogen (N) (20, 30, and 40 kg N/hectare (ha)), two levels of phosphorus (0 and 80 kg P_2O_5/ha), and two levels of potassium (0 and 80 kg K_2O/ha) were evaluated in two varieties of Virginia tobacco (HR62-7 and Hicks special). Variety HR62-7 gave higher yields, leaf production, and better growth characteristics (larger leaf size, plant size, and number of curable leaves) than Hicks special. However, HR62-7 was late flowering as compared to Hicks special. There was a progressive and significant increase in green leaf yield with increasing levels of N. Cured leaf exhibited a similar, but insignificant trend. No significant differences were seen in green and cured leaf yields with P_2O_5 applications. Application of K_2O resulted in significant yield character improvement. Economically, concentrations of 30 kg N/ha and 80 kg K_2O/ha were advantageous.

See also, **80–1074, 80–1086, 80–1260**

CHEMISTRY

80–1074. Andersen, R. A.; Tso, T. C.; Chaplin, J. F. **Total Carbonyls and Phenols in Experimental Burley and Bright Tobacco.** *Journal of Agricultural and Food Chemistry* 27(4): 891-895, 1979.

Soluble carbonyls and phenols were determined in several experimental tobacco materials that were produced by practices that altered concentrations of these health- and quality-related components. An improved spectrophotometric method was used for measuring total carbonyls as quinoidal anions of their 2,4-dinitrophenylhydrazones. Phenolic estimations were based on the extent of hydrogen bonding of phenols to insoluble polyvinylpyrrolidone. Carbonyl and phenolic levels decreased in bright tobacco that was grown at higher-than-normal plant population density or reconstituted into sheets after flue curing. Carbonyl concentrations reduced in air-cured Burley leaves that were harvested from successively higher leaf positions on the stalk, whereas phenolics increased with ascending stalk position. Phenolics were reduced in tobacco that was treated for removal of soluble protein. (Auth. Abs.)

80–1075. Boguslawska, M.; Nagorski, B. **Oznaczanie Cyjanowodoru w Dymie Tytoniowym. [Hydrogen Cyanide Determination in Tobacco Smoke.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* (1/2): 49-67, 1977, Polish.

Spectrophotometric and potentiometric methods for determining hydrogen cyanide (HCN) in tobacco smoke have been developed. The colorimetric reaction method for spectrophotometric detection results in a light blue complex whose maximum absorption is at 630 nm. The results of spectrophotometric analyses of cigarette smoke for HCN content are given. The potentiometric method consists of titration of HCN and hydrogen sulfide (H_2S) with silver nitrate solution. Comparison of the potentiometric HCN data with the spectrophotometric data showed significant discrepancy for the gas-vapor and the liquid-solid phases of tobacco smoke. The potentiometric method was then modified by the addition of lead carbonate to eliminate excess sulfide ions prior to HCN measurement. The HCN data obtained by the modified potentiometric method for both phases of cigarette smoke were in good agreement with the spectrophotometric data. Both methods are useful for determination of HCN and H_2S in cigarette smoke.

80–1076. Elmenhorst, H.; Nicolaus, G. **Dihydro-γ-Pyrone im Tabakrauch. Nachweis von 2,3-Dihydro-5-Hydroxy-6-Methyl-4H-Pyran-4-on in Rauch von Cigaretten aus Burley-, Virginia- und Orienttabaken. [Dihydro-γ-Pyrones in Tobacco Smoke. Identification of 2,3-Dihydro-5-Hydroxy-6-Methyl-4H-Pyran-4-one in the Cigarette Smoke of Burley, Virginia, and Oriental Tobaccos.]** *Beitraege zur Tabakforschung International* 10(1): 39-47, December 1979, German.

A new compound—2,3-dihydro-5-hydroxy-6-methyl-4H-pyran-4-one (I)—and its homolog—2,3-dihydro-3,5-dihydroxy-6-methyl-4H-pyran-4-one (II) were identified and determined quantitatively in the smoke of nonadditive-containing cigarettes made from Burley, Virginia, and Oriental tobacco varieties. Smoking was done on a CSM-10 smoking machine. The semivolatile smoke fraction was condensed and analyzed by gas chromatography coupled with mass spectrometry. In addition, the unstable I compound and the II compound were identified by exact mass determination, mass spectrum of the deuterated I and II compounds, and nuclear magnetic resonance spectroscopy. The concentrations of I were found to be 26, 20, and 6 μg/cigarette in the smoke from Oriental, Virginia, and Burley tobacco cigarettes, respectively. The corresponding figures for II were 11, 13, and 4 μg/cigarette. The different concentrations of I in the smoke may indicate that I is the cause of variety-specific difference in tobacco flavor. This new flavor compound has a caramel odor with fruity overtones. Because of the similarity of chemical structure I to maltol and cyclotenes, the majority of the aroma substances in tobacco smoke may have the structure of an alicyclic or *o*-heterocyclic enolone.

80–1077. Gopalam, A.; Gopalachari, N. C. **Biochemical Changes During Maturation of Flue-Cured Tobacco. I—Changes in Leaf Pigments.** *Tobacco Research* 5(2): 113-117, December 1979.

A comparative study of the changes in leaf pigments during maturation in two varieties of tobacco (Kanakaprabha and CTRI Special) in five different maturity stages has been made. Kanakaprabha leaves contained significantly higher amounts of chlorophyll a and b. Chlorophyll degradation during maturation was common in both the varieties. However, in Kanakaprabha leaves, degradation of chlorophyll a and b was slower in the initial stages of maturation. Carotene and xanthophyll levels in the two varieties were not different. There was a progressive decline in carotenes in Kanakaprabha leaves as maturation progressed. In CTRI Special there was a rapid decline in leaf carotenes in the early stages of maturity, and later on, the rate of decline slowed. Xanthophylls increased in the early phases of maturity and declined in overmature leaves in both the varieties. (Auth. Abs.)

80–1078. Keith, C. H. **The Use of Pressure Drop Measurements for Estimating Ventilation and Paper Porosity.** *Beitraege zur Tabakforschung International* 10(1): 7-14, December 1979.

Equations for calculating dilutions in vented cigarette filters and tobacco columns from pressure drop measurements are derived. These calculated dilutions are found to agree closely with those directly measured for a variety of commercial and experimental cigarettes wrapped with perforated and porous tipping and with different cigarette papers. The effect of hole placement on dilution and cigarette pressure is estimated. The effects of filter dilution on particulate matter, condensate, and carbon monoxide (CO) delivery are discussed. A method for measuring filter dilution during smoking by means of pressure drop measurements is outlined and results for a typical vented filter cigarette are shown and discussed. The use of pressure drop measurements for estimating paper porosity and amount of dilution in the tobacco column on finished cigarettes is outlined. The dilution obtained by this method was generally higher compared to the results found by a model based on a capillary flow regime, which suggests that flow through ordinary cigarette paper is a combination of capillary and orifice flow. The nondestructive nature and simplicity of pressure drop measurements make this method of measuring dilution useful in quality control of cigarette production. (Auth. Abs. Mod.)

80–1079. Kuter, E. **Zastosowanie Chromatografii Gazowej do Oznaczania Fenoli w Dymie Tytoniowym. [Use of Gas Chromatography for the Determination of Phenols in Tobacco Smoke.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* (1/2): 41-48, 1977, Polish.

A method for identifying and quantitatively determining carcinogenic phenols in cigarette smoke condensate and cigarette filters using gas chromatography is described.

Phenol; *o*-, *m*-, and *p*-cresols; *o*-chlorophenol; pyrocathecol; and hydrochinone were identified by comparing relative retention times with those of standard solutions. Before chromatographic separation, phenols were converted to nonpolar silicilic ethers. Phenol, *p*-, *o*-, and *m*-cresols were determined quantitatively in smoke from a king size filter cigarette and a 64 mm long nonfilter cigarette. Test cigarettes were smoked under standard conditions. Statistical analysis of the data of five sequential determinations shows the coefficient of variation to be in the 1.31-1.83 percent range.

80–1080. Manole, G. **Obtinerea Unor Arome Pentru Tigarete de Tip Superior. [Obtainment of Some Aromas for High Quality Cigarettes.]** *Productia Vegetala—Cereale si Plante Tehnice* 29(10): 24-29, October 1977, Romanian.

From the dust of Virginia and Burley tobacco varieties, a slightly acid fraction containing several aromatic compounds was obtained by extraction, filtration, and chromatographic column separation. This fraction may become a base for tobacco aroma compositions which may also contain other aromatic extracts or synthetic aromas. (Auth. Abs. Mod.)

80–1081. Mayo, R. **Practical Use of Tar Prediction From Ground Tobacco and Cut Rag.** *Tobacco International* 182(2): 12-13, 19, January 25, 1980.

A method and the machinery to provide rapid and accurate measurement of the tar potential of tobacco products is described. The machine, when used with other standard laboratory equipment, can easily and quickly produce data from unprocessed tobacco which, when combined with known cigarette design interactions, will give a prediction of the tar yield of completed cigarettes; and typically, a correlation coefficient of 0.98 when compared with conventional cigarette smoke has been produced. To obtain tar levels for monitoring the accuracy of the tar predicting machine, cut rag can be used to bypass the milling process.

80–1082. Moldenhauer, W.; Kuenstler, I. **Beitrag zur Bestimmung von Metobromuron-Ruckstanden im Tabak. [Contribution to the Determination of Metobromuron Residues in Tobacco.]** *Berichte des Instituts fuer Tabakforschung, Dresden* 24/25: 75-79, 1977/78, German.

A modified method for quantitative serial determination of metobromuron residues in tobacco is reported. The method includes alkaline hydrolysis with distillation, distillate acidification and concentration, diazotization of 4-bromoaniline in the solution and coupling of the diazo-product with N-(1-naphtyl)-ethylenediamine dihydrochloride. The azo dye thus obtained is purified by column chromatography and measured spectrophotometrically. The result is given as 4-bromoaniline, a metabolite of metobromuron which can then be calculated. The sensitivity of the method is 0.5 ppm with 2.8 percent relative error.

80–1083. Morgan, W. D. **New Ways of Measuring Cadmium in Man.** *Nature* 282(5740): 673-674, December 13, 1979.

For the general population, the main source of cadmium for nonsmokers is food, the average diet containing 0.01-0.04 μg/g. Cigarettes contain 1-2 μg cadmium, of which 5 percent may be absorbed, so that a smoker of 30 cigarettes/day for 10 years may accumulate up to 10 mg of the metal in addition to a dietary intake of up to 15 mg in the same period. Several methods and instruments for measuring cadmium in humans are mentioned.

80–1084. Nagaraj, G.; Chakraborty, M. K. **Lipophilic Constituents of NATU Tobacco: III. Fatty Acids.** *Tobacco Research* 5(2): 125-128, December 1979.

The nonvolatile fatty acids and their methyl esters were isolated from the hexane extract of Natu tobacco. They were analyzed by gas liquid chromatography and identified by their R_T and equivalent chain length values. The following acids $C_{10:0}$, $C_{12:0}$, $C_{14:0}$, $C_{14:1}$, $C_{16:0}$, $C_{16:1}$, $C_{18:1}$, $C_{18:2}$, $C_{18:3}$, $C_{18:0}$, and $C_{20:0}$ were identified as constituents of this tobacco. Among these $C_{14:1}$ is a newly found constituent of tobacco leaf. $C_{18:3}$, and $C_{16:0}$ are the major nonvolatile fatty acids of Natu tobacco. (Auth. Abs.)

80–1085. Prasad Rao, J.A.V.; Nageswara Rao, C. R.; Gopalachari, N. C. **A Note on the Feasibility Studies on the Prediction of Total Volatile Bases From Total Nitrogen and Nicotine in F.C.V. Tobacco.** *Tobacco Research* 3(2): 111-112, 1977.

Thirty-eight representative cured leaf samples of flue-cured variety (fcv) tobacco grown in black soil were analyzed for nitrogen (N), nicotine, and total volatile bases, and the data were statistically analyzed by the multiple regression technique using total N and nicotine as independent variables. The simple correlation coefficients for these variables indicated a very high correlation (0.80 and 0.95) with total volatile bases. In view of these correlation coefficients, the multiple regression equation can be used to calculate total volatile bases from total nitrogen and nicotine in fcv tobacco.

80–1086. Reisch, W. **Der Tabak und seine Inhaltsstoffe—Neue Aufgaben der Tabakzuchtung. [Tobacco and its Constituents—New Problems for Tobacco Culture.]** *Der Deutsche Tabakbau* 58(22): 265-268, November 15, 1978, German.

Data from the literature are cited to support the contention that not all kinds of smoking present a health risk to smokers and that smoking is not a risk to nonsmokers (passive smokers). Positive aspects of smoking are stressed. Although it is believed that actual scientific data do not justify the present-day attacks on smoking, the existence of new problems for tobacco culture is recognized. The problem for scientific research is to develop a tobacco variety which would neither excessively endanger human health nor interfere with environmental and hygienic requirements. Toward this aim, the Land Institute Forchheim for Tobacco Cultivation and Tobacco Research carried out an experimental study of chemical selection of mildew-resistant hybrid tobacco strains low in nicotine and secondary alkaloids. Nicotine, nornicotine, and anabasine were determined in the tobacco, and condensate, carbon monoxide (CO), and nicotine were analyzed in the smoke of the newly developed varieties. In most of the new mildew-resistant strains, a clear decrease in these constituents was detected in comparison with the standard varieties. For example, the nicotine content of 33 out of 45 new Burley tobacco strains was below the nicotine content of the standard tobacco (0.99 percent), averaging 0.42 percent. The smoke condensate, CO, and nicotine levels of 19 out of 24, 14 out of 24, and 28 out of 45 new strains of the same tobacco, respectively, were lower than their levels in the standard Burley tobacco (23.4, 11.6, and 1.7 mg/cigarette, respectively), averaging 19.5, 11.2, and 1.4 mg/cigarette.

80–1087. Sakuma, H.; Shimojima, N.; Sugawara, S. **Tabako Enchu no Gisan Oyobi Sakusan no Kani Buusekiho. [Method for the Rapid Determination of Formic and Acetic Acids in Cigarette Smoke.]** *Japan Tobacco and Salt Public Corporation Central Research Institute Scientific Papers* (119): 107-109, 1977, Japanese.

A method for the rapid and simultaneous determination of formic and acetic acids in cigarette smoke is described. The smoke condensate of five cigarettes is collected in a cold trap cooled with dry ice—acetone and extracted with 5 ml of 2.5 percent sodium bicarbonate. The extract is washed with ether and dichloromethane, and then dried under reduced pressure. Two ml of n-buthanol, 0.2 ml of concentrated sulfuric acid, and 2 g of anhydrous sodium sulfate are added to the dried residue and the mixture is heated at 100°C for 15 min. After cooling, the reaction mixture is shaken with 1 ml of n-pentane and 5 ml of water. One μl of the pentane layer is analyzed by glass capillary column gas chromatography. (Auth. Abs.)

80–1088. Snook, M. E.; Fortson, P. J.; Chortyk, O. T. **Application of Gel Chromatography to Characterize More Completely the Phenols of Cigarette Smoke.** *Tobacco International* 182(6): 78-84, March 21, 1980.

The phenols of cigarette smoke condensate (CSC) were isolated in a greatly enriched fraction by a combination of base extraction, silicic acid chromatography, and gel chromatography. The constituents of the phenol isolate were characterized by ultraviolet spectra and by gas chromatography (GC)-mass spectrometry analyses. Silicic acid chromatography effectively separated the monohydroxybenzenes from the dihydroxybenzenes. For the first time, gel chromatography on Bio-Beads SX-8 was used to produce a relatively pure phenol isolate and to greatly enrich the phenols. Furthermore, partial separation of phenols by the gels greatly aided identification of compounds in multiple component GC peaks. Several hydroxypyrones and alkylnaphthoquinones were detected and over 135 phenols were characterized in the gel fractions. The presence of indenols in CSC is reported for the first time. (Auth. Abs.)

See also, **80–1258, 80–1263**

80–1089. Specht, W.; Tillkes, M. **Gaschromatographische Bestimmung von Ruckstanden an Pflanzenbehandlungsmitteln nach Clean-up uber Gelchromatographie und Mini-Kieselgel-Saulenchromatographie. 1. Mitteilung: Organochlor-Pflanzenbehandlungsmittel in Tabak und Tabakerzeugnissen. [Gas Chromatographic Determination of Pesticide Residues in Plants Using Gel Chromatography and Silica Gel Mini-Column Chromatography. 1. Organochlorine Pesticides in Tobacco and Tobacco Products.]** *Beitraege zur Tabakforschung International* 10(1): 73-79, December 1979, German.

A versatile chromatographic method has been developed for determining pesticide residues in tobacco and tobacco products. An improved procedure with an acetone-water mixture is used to extract both lipophilic and hydrophilic substances from tobacco. The key step of the method is the separation of the co-extracted plant constituents from the active substances (residues) by gel permeation chromatography on polystyrene gel. The dry residue from the evaporation of the extracted organic phase is dissolved in ethyl acetate and the solution is processed in an automated gel permeation chromatograph, using the (1:1) ethyl acetate/cyclohexane mixture as eluant. In a test with known quantities of organochlorine pesticides added to untreated tobacco, the pesticide residues were totally recovered in 65 ml of eluate. The eluate is further purified by gas chromatography on a mini-column of deactivated silica gel, and two fractions of 10 ml each are collected separately. Residues of 33 organochlorine pesticides were determined in these eluates. Typical detection thresholds were 0.002 mg/kg for α-benzene hexachloride and 0.02 mg/kg for p,p′-DDT. Recoveries of 90 to 100 percent of the chlorinated pesticide residues were obtained in the tests with Burley and Virginia tobacco samples. The method is suitable also for extraction and purification of polar pesticides, e.g., organophosphorus compounds, in a single sample.

80–1090. Victorian Cancer News. **Herbal Cigarettes Not Safe.** *Victorian Cancer News* 91: 2, July-September 1979.

The Australian Government Analyst has examined five types of nontobacco smoking material manufactured and recently marketed in Australia. Tar levels of these herbal materials ranged from 5.2 to 14.1 mg/cigarette, which are within the range of those found in standard cigarette brands. Carbon monoxide content ranged from 13.6 to 18.8 mg/cigarette, similar to the amounts found in Australian filter cigarettes. On the basis of the above findings, these herbal cigarettes should not be regarded as harmless.

PHARMACOLOGY AND TOXICOLOGY

80-1091. Adir, J.; Wildfeuer, W.; Miller, R. P. **Effect of Ethanol Pretreatment on the Pharmacokinetics of Nicotine in Rats.** *Journal of Pharmacology and Experimental Therapeutics* 212(2): 274-279, February 1980.

The pharmacokinetics of nicotine was compared in male Fischer-344 rats pretreated with an ethanol or a sucrose solution for 12.5 days. The animals received either ethanol at doses of 4 g/kg/day for 7 days followed by 8 g/kg/day for 5.5 days or daily doses of an isocaloric-isovolumetric sucrose solution for 12.5 days by gastric intubation. On the 14th day, a 0.4 mg/kg dose of (^{14}C methyl)nicotine was injected intravenously into both groups, and blood samples were collected at timed intervals for 30 hours. Nicotine and its metabolites in plasma were separated by thin-layer chromatography and quantitated by liquid scintillation counting. The plasma levels of total radioactivity, nicotine, cotinine, and other polar metabolites were significantly lower in the ethanol as compared to the sucrose-treated rats. Ethanol pretreatment produced no changes in the hybrid rate constants describing the biphasic decline of plasma nicotine concentration, but its apparent volume of distribution and total plasma clearance were increased by 45 percent ($p<0.05$) suggesting that ethanol may alter the distribution of nicotine. The apparent volume of distribution of cotinine and its rate of production also were significantly increased indicating an ethanol-related induction of this major metabolite of nicotine. (Auth. Abs.)

80-1092. Aleksandrov, V. N. **Fiziologo-Gigienicheskaia Reglamentatsiia Rabochei Sredy. [Physiological and Hygienic Regulation of the Work Environment.]** *Voenno-Meditsinskii Zhurnal* (3): 47-50, March 1978, Russian.

Carboxyhemoglobin (COHb) levels in blood and mental capacity were determined in 54 tests with 6 healthy men before and after inhalation of exhaust gases which contained carbon monoxide (CO) in maximum tolerable (MT) or maximum permissible (MP) concentrations. The MT is defined as the concentration 104 ± 13.7 mg/m^3 CO in exhaust gas which may cause a temporary decrease in mental capacity and reversible functional changes. At the end of the tests with CO at MP, the COHb concentration was 4.52 ± 0.53 percent. In the tests with CO at MT level, COHb content increased from 1.76 percent before inhalation to 9.3 ± 0.49 and 12.3 ± 0.75 percent after 3- and 4-hour long inhalations, respectively. However, the COHb level decreased to 4.6 ± 0.44 percent 3 hours after inhalation was discontinued. During inhalation, the respiratory volume in 1 min, pulse rate, and respiration rate increased. Mental capacity indices remained high until the middle of a test with a 4-hour inhalation of CO at MT level; the indices then decreased to an intermediate level toward the end of the inhalation. The rate of information processing decreased temporarily by about 25 percent after the 4-hour inhalation. Using a computer model, the maximum COHb level compatible with high mental capacity was estimated to be 6 or 10 percent for CO inhalation at the MP or MT level, respectively. A simplified formula was derived for calculating MT of CO vs. exposure time in emergency situations.

80-1093. Baettig, K.; Martin, J. R.; Classen, W. **Nicotine and Amphetamine: Differential Tolerance and No Cross-Tolerance for Ingestive Effects.** *Pharmacology, Biochemistry and Behavior* 12(1): 107-111, January 1980.

Rats chronically treated twice daily with nicotine (0.4 mg/kg, s.c.) or *d,l*-amphetamine (1.5 mg/kg, s.c.) exhibited different patterns of anorexia, hypodipsia, and body weight loss. Amphetamine-treated rats developed tolerance to these ingestive effects and to weight loss, whereas nicotine-injected rats did not. There was little, if any, evidence for cross-tolerance when the drugs were switched between the two groups. These results indicate that different mechanisms underlie the ingestive effects of nicotine and amphetamine. (Auth. Abs.)

80-1094. Basrur, P. K.; Harada, T. **Alterations in the Nasal Mucosa of Syrian Golden Hamsters Exposed to Cigarette Smoke.** *Progress in Experimental Tumor Research* 24: 283-301, 1979.

Syrian golden hamsters subjected for 4 weeks to smoke inhalation from three types of Canadian experimental cigarettes were killed at intervals of 0, 1, and 2 weeks following the last smoke exposure, and the cellular response of the nasal mucosa after cessation of smoking was examined to compare the effect of these cigarettes on regenerative capacity. The incidence of acute rhinitis was greater in all smoke-exposed groups of animals killed immediately after the last exposure (at 0 time) than that in the controls, and the frequency and severity of inflammation were greater in hamsters exposed to the cigarette rated as strong on the basis of the smoke components. The inflammatory reaction was observed mainly in the respiratory region of the nasal cavity and was characterized by vacuolar degeneration and desquamation of the epithelium accompanied by leukocyte infiltration and increased mucous secretion. Surface morphology of the nasal mucosa of smoke-exposed hamsters revealed a decrease in the ciliated cell-lined zones and a widening of the squamous cell-lined zones in the anterior nasal fossa. After a 1- or 2-week recovery period, the inflammatory changes in the smoke-exposed hamsters decreased to control levels, but the regenerated epithelium was atypical in the group exposed to smoke from strong cigarettes. The persistence of some of the cellular changes and the hyperactivity of the mucosal cells of hamsters exposed to smoke from high total particulate matter cigarettes suggest that the degree of regenerative response may be dependent upon the extent of damage elicited by the types of cigarettes used for smoke generation. (Auth. Abs.)

80–1095. Cantrell, E. T.; Marshall, M.; McLemore, T.; Ghanayem, B.; Busbee, D. **Secondary Metabolism of Benzo(*a*)pyrene in Human Cells.** *Proceedings of the Western Pharmacology Society* 22: 273-276, 1979.

The data presented here indicate that cultured human lymphocytes and pulmonary alveolar macrophages have the capacity to metabolize benzo(*a*)pyrene (BP) to several hydroxy and quinone derivatives. These are further metabolized to polar conjugates, including sulfate and glucuronides. Glutathione conjugates were not considered in this study, but these probably contribute to the large amount of water-soluble radioactivity remaining after enzymatic hydrolysis. The proportions of the various primary metabolites and conjugates vary between individuals, and vary with the time of incubation and BP concentration in a single individual. The complexity of the metabolism of BP in human tissues complicates interpretation of earlier reports suggesting a relationship between smoking, aryl hydrocarbon hydroxylase, and cancer risk.

80–1096. Cohen, G. M.; MacLeod, M. C.; Moore, C. J.; Selkirk, J. K. **Metabolism and Macromolecular Binding of Carcinogenic and Noncarcinogenic Metabolites of Benzo(*a*)pyrene by Hamster Embryo Cells.** *Cancer Research* 40(2): 207-211, February 1980.

The metabolism and macromolecular binding of four metabolites of benzo(*a*)pyrene in hamster embryo fibroblasts has been studied. Two noncarcinogenic phenolic derivatives, 3-hydroxybenzo(*a*)pyrene and 9-hydroxybenzo(*a*)pyrene, are rapidly metabolized, primarily to their respective glucuronic acid conjugates and other H_2O-soluble conjugates (78.4 to 80.8 percent of total radioactivity). Water-soluble conjugates were also formed from the carcinogenic phenol, 2-hydroxybenzo (*a*) pyrene, and from 7,8- dihydro - 7,8-dihydroxybenzo(*a*)pyrene, but in lower amounts (36.8 to 43.8 percent of total radioactivity). With each of the compounds, from 10 to 20 percent of the radioactivity was converted to ethyl acetate-soluble metabolites. The amount of unmetabolized 2-hydroxybenzo(*a*)pyrene recovered intracellularly was 20-fold higher than that recovered in incubations with the other phenols. Covalent binding to nuclear macromolecules was monitored after isopyknic separation. Binding of the three phenols tested was similar and was lower than the binding of benzo(*a*)pyrene to nuclear DNA, RNA, and protein. In contrast to the results with the monohydroxybenzo(*a*)pyrenes, high levels of covalent binding were observed with 7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene; binding to DNA was 8-fold higher (315 pmol bound per mg DNA) than binding of benzo(*a*)pyrene to DNA. (Auth. Abs.)

80–1097. Dansette, P. M.; Alexandrov, K.; Azerad, R.; Frayssinet, C. **The Effect of Some Mixed Function Oxidase Inducers on Aryl Hydrocarbon Hydroxylase and Epoxide Hydrase in Nuclei and Microsomes From Rat Liver and Lung. The Effect of Cigarette Smoke.** *European Journal of Cancer* 15(6): 915-922, 1979.

The activities of aryl hydrocarbon hydroxylase (AHH) and epoxide hydrase (EH) in rat lung microsomes and nuclei were compared to those of liver from untreated (C) rats and rats pretreated with phenobarbital (PB), methylcholanthrene (MC), and cigarette smoke. The pretreatment of rats with three different types of mixed function oxidase inducers produced different effects on the induction of AHH and EH in rat liver microsomes and nuclei. With MC, a sixfold increase in AHH enzyme activity was observed in both microsomes and nuclei from liver, while the PB and cigarette smoke did not increase AHH activity in these fractions. Epoxide hydrase in liver microsomes and nuclei was increased fourfold by PB while MC and cigarette smoke inhalation by rats showed no effect. The pretreatment of rats with MC increased lung nuclear and microsomal AHH six- to ninefold; cigarette smoke induced these activities threefold. Phenobarbital did not cause any significant induction. Lung nuclear and microsomal epoxide hydrase were not affected by pretreatment of rats with MC or PB. The only significant induction of epoxide hydrase in lung tissue was observed in the microsomal fraction and was caused by inhalation of cigarette smoke. This activity increased rapidly within 2 hours of exposure to the cigarette smoke, reached a value two to three times that of controls, and subsequently began to decline 6 hours after inhalation. (Auth. Abs.)

80–1098. Depersdorff, J.; Augsten, M. **Die Bestimmung des Thiocyanats im Serum. Eine Moglichkeit zur Objektivierung des Zigarettenrauchens. [The Determination of Thiocyanate in Serum. A Possibility for the Detection of Smokers.]** *Deutsche Gesundheitswesen* 34(24): 1120-1123, 1979, German.

Thiocyanate concentration in the serum of 750 adult men and 544 adult women was determined photometrically at the same time as subjects smoking habits were surveyed. The purpose was to assess intensity of smoking. There were 63.7 percent male and 12.7 percent female smokers in the population sample. The breakdown of smokers by age and sex was typical for a West German rural population. Based on cigarette consumption and considering passive smoking effect in the case of nonsmokers or light smokers (1-5 cigarettes/day), it was shown that the average thiocyanate concentration was 137 and 148μm/l in male and female smokers of 11-20 cigarettes/day, respectively, vs. 66μm/l in nonsmokers of both sexes. The average values increase with the increasing daily cigarette consumption. Thiocyanate level increases are approximately 56 and 49μm/l in male and female smokers, respectively, vs. 31μm/l in nonsmokers of both sexes. A marked decrease of these values with advanced age is particularly pronounced in male smokers. Smoke inhalation decreases the percentage of men smokers with thiocyanate concentrations ≤90μm/l from 28.9 to 26.1. This percentage increases to 43.8 in noninhaling smokers only. Inclusion of the thiocyanate concentration test in general health checkups is advised.

80–1099. Descotes, J.; Brazier, J. L.; Ollagnier, M.; Evreux, J.-C. **Influence de la Consommation de Tabac sur la Pharmacocinetique de la Cafeine. [Influence of Tobacco Consumption on Caffeine Pharmacokinetics.]** *Therapie* 34(5): 619-624, September-October 1979, French.

A study of caffeine pharmacokinetics in four healthy male volunteers showed a wide difference between three nonsmokers and one smoker. The mean value of caffeine half-life in the nonsmokers was 7.6 hours (6.4-9.1 hours), corresponding to an overall clearance of 0.830 ml/sec (0.744-0.944). In the only smoker, the overall clearance of caffeine was larger (1.554 ml/sec), generating a decreased half-life of approximately 4.1 hours. His pharmacokinetic parameters reached a somewhat normal level 3 months after he stopped smoking. (Auth. Abs.)

80–1100. Dodds, W. J.; Christensen, J.; Dent, J.; Arndorfer, R. C.; Wood, J. D. **Pharmacologic Investigation of Primary Peristalsis in Smooth Muscle Portion of Opossum Esophagus.** *American Journal of Physiology* 237(6): E561-E566, December 1979.

In 40 animals, swallow-induced peristalsis was recorded using a high-fidelity manometric infusion system. The recording assembly had eight recording sites, 2 cm apart, that spanned the entire esophageal body. Primary peristalsis was recorded before and after administration of a variety of pharmacologic agents. A combination of hexamethonium and atropine, or a large dose of nicotine (500 μg/kg) abolished peristalsis in the smooth-muscle esophageal segment. It was concluded that (1) primary peristalsis in the smooth-muscle portion of the opossum esophagus is partially determined by muscarinic neurotransmission, which may have a postganglionic as well as ganglionic locus, (2) predominantly cholinergic ganglionic synapses with nicotinic and muscarinic receptors are present in the neural pathways eliciting primary peristalsis in esophageal smooth muscle, and (3) no evidence was shown for a ganglionic synapse in the neural motor pathway to the esophageal striated musculature. (Auth. Abs.)

80–1101. Dodson, R. A.; Johnson, W. E. **Effects of Ethanol, Arecoline, Atropine and Nicotine, Alone and in Various Combinations, on Rat Cerebellar Cyclic Guanosine 3',5'-Monophosphate.** *Neuropharmacology* 18(11): 871-876, November 1979.

Rat cerebellar cyclic guanosine 3',5'-monophosphate (cGMP) concentrations were determined by radioimmune methods after sacrifice with focused microwave fixation in animals pretreated with ethanol, arecoline, atropine, and nicotine alone and in various combinations. Although arecoline and nicotine treatments resulted in large cerebellar increases ($p<0.05$) in cGMP, they failed to antagonize ethanol's depressive effects. Atropine treatment did not augment ethanol-induced decreases in cGMP content. These data suggest that ethanol's depressant actions on cerebellar cGMP are independent of cholinergic mechanisms. (Auth. Abs.)

80–1102. Dohi, T.; Tsujimoto, A. **Possible Mechanisms of Sustained Tremor Induction by Nicotine in Pilocarpine Treated Animals.** *Archives Internationales de Pharmacodynamie et de Therapie* 235(1): 62-72, September 1978.

Alterations of steady state and metabolism of putative neurotransmitters were studied in the brain of rats with a sustained tremor evoked by nicotine and pilocarpine. The norepinephrine content in several brain regions was decreased, whereas striatal dopamine was unchanged. Homovanillic acid concentrations were slightly increased, while 5-hydroxyindoleacetic acid concentrations were markedly increased. Pilocarpine alone increased the 5-hydroxytryptamine (5-HT) turnover rate at high ambient temperature (30-32°C), but not at normal ambient temperature (22±2°C), atropine partially blocked the increase of 5-HT turnover rate by pilocarpine. Pretreatment with *p*-chlorophenylalanine and hemicholinium-3 decreased the tremor intensity elicited by nicotine and pilocarpine. It is proposed that the increase of cholinergic function caused by nicotine and the activation of muscarinic receptors and the increase in 5-HT function caused by pilocarpine might be involved in the mechanisms of tremor induction by nicotine and pilocarpine. (Auth. Abs.)

80–1103. Dollery, C. T.; Fraser, H. S.; Mucklow, J. C.; Bulpitt, C. J. **Contribution of Environmental Factors to Variability in Human Drug Metabolism.** *Drug Metabolism Reviews* 9(2): 207-220, 1979.

Drug metabolism in 96 London factory and office workers has been studied after simultaneous single doses of antipyrine (phenazone) and paracetamol (acetaminophen). Metabolic clearance of both drugs was significantly greater in subjects eating meat more than once a week than in those who ate meat less frequently. The precise contribution of diet could not be clearly defined since 90 percent of meat-eaters were European while all except one of the vegetarians were Asian. Clearance of both drugs increased with alcohol consumption. In addition, antipyrine and paracetamol clearances were significantly higher in smokers than in nonsmokers ($p<0.001$ and $p<0.05$, respectively). Regular intake of coffee and/or tea had a similar effect but, in the case of paracetamol, this did not attain statistical significance. Antipyrine clearance was lower among women who took the oral contraceptive pill than among those who did not, while paracetamol clearance was not significantly different. These findings may have implications for the use of drugs which are administered regularly in order to achieve steady-state plasma and tissue concentrations. (Auth. Abs. Mod.)

80–1104. Dossing, M.; Paulev, P.-E. **Does Smoking Influence the Lead Body Burden in Workers?** *Danish Medical Bulletin* 27(1): 55-58, February 1980.

The lead content in the urine excretion of 32 workers and 6 office employees at an ore dressing plant in a Greenland mining town was measured over a 24-hour period after an infusion of 25 mg $CaNa_2EDTA$ per kilo body weight. The content of both blood and erythrocyte lead was measured before, during, and after the standard calcium disodium edathamil ($CaNa_2EDTA$) test. The aminolevuline acids in the urine and serum were also measured. The workers had been exposed to lead dust for an average of 4,300 hours. The workers were divided into three comparable groups according to their smoking habits: (1) nonsmokers, (2) smokers (1-20 cigarettes/day), and (3) heavy smokers ($>$20 cigarettes/day). None of the employees demonstrated symptoms or signs of lead poisoning. No differences materialized in the lead variables between the three groups. However, the $CaNa_2EDTA$ lead excretion was significantly higher in the nine nonsmoking workers than in comparable office employee smokers ($p<0.005$). It is concluded that any preventive measures against industrial exposure to lead should be directed at the sources of lead dust in the production plant. A ban on cigarette smoking on the premises would be insufficient as an isolated measure. (Auth. Abs.)

80–1105. Ellis, K. J.; Vartsky, D.; Zanzi, I.; Cohn, S. H. **Cadmium: In Vivo Measurement in Smokers and Nonsmokers.** *Science* 205(4403): 323-325, July 20, 1979.

Absolute amounts of cadmium (in mg) in the left kidney and concentrations of cadmium ($\mu g/g$) in the liver were measured in vivo in 20 healthy adult male volunteers. Organ cadmium levels of smokers were significantly elevated above those of nonsmokers. No relationship was evident between body stores of cadmium (liver and kidney) and cadmium or β_2-microglobulin in urine or blood. The average total body burden of cadmium in man at age 50 is estimated to be 19.3 mg for nonsmokers and 35.5 mg for smokers (38.7 pack-year smoking history). Biological half-time for the whole body was, on average, 15.7 years (10- to 33-year range). Dietary absorption was 2.7 μg/day. Cigarette smoking resulted in the absorption of 1.9 μg/pack. (Auth. Abs.)

80–1106. Fee, J.P.H.; Black, G. W.; Dundee, J. W.; McIlroy, P.D.A.; Johnston, H.M.L.; Johnston, S. B.; Black, I.H.C.; McNeill, H. G.; Neill, D. W.; Doggart, J. R.; Merrett, J. D.; McDonald, J. R.; Bradley, D.S.G.; Haire, M.; McMillan, S. A. **A Prospective Study of Liver Enzyme and Other Changes Following Repeat Administration of Halothane and Enflurane.** *British Journal of Anaesthesia* 51(12): 1133-1141, December 1979.

A prospective study of liver enzymes and other measurements following repeat administrations of halothane or enflurane was carried out in patients undergoing minor urological operations. The patient populations were similar with respect to frequency of factors which might influence liver function, social habits (e.g., tobacco and alcohol consumption), drug therapy, and time intervals between administrations. Sixty-three received two or more administrations of halothane and 66 received two or more administrations of enflurane, both drugs given with nitrous oxide in oxygen. There was a greater frequency of increased enzymatic activity following repeat administrations of halothane than following enflurane, and the average alanine aminotransferase and gamma glutamyl transpeptidase concentrations were increased to a greater degree following halothane than enflurane. There was a significant difference in abnormal enzyme activity 2 weeks after halothane administration between smokers and nonsmokers ($p<0.05$). There was no change in the eosinophil count and no significant postoperative morbidity. Changes in alanine aminotransferase, lactate dehydrogenase, and gamma glutamyl transpeptidase occurred more frequently in obese patients receiving halothane. (Auth. Abs. Mod.)

80–1107. Gielen, J. E.; Goujon, F.; Sele, J.; Van Cantfort, J. **Organ Specificity of Induction of Activating and Inactivating Enzymes by Cigarette Smoke and Cigarette Smoke Condensate.** *Archives of Toxicology* (Supplement 2): 239-251, 1979.

Inhalation of cigarette smoke specifically induces the rat lung and kidney aryl hydrocarbon hydroxylase (AHH) in less than 4 hours. The epoxide hydratase (EH) and the glutathione S-transferase are not significantly modified by a similar treatment in any of the rat tissues. Compared to the kidney AHH, the lung hydroxylase is 3-4 times more sensitive to small concentrations of cigarette smoke and seems to have a longer biological half-life. In both tissues, the induced AHH presents the same in vitro sensitivity to various inhibitors as a polycyclic hydrocarbon induced AHH. In primary fetal rat liver cell culture, the cigarette smoke condensate fractions (CSCF) induce both the AHH and EH activity. Nevertheless, the AHH activity responds faster and to lower concentrations of CSCF than the EH activity. The liver cell culture constitutes a unique tool for a comparative study of the AHH and EH induction mechanism. Low concentration (10 μM) of benz(*a*)anthracene induces only the AHH activity while trans-stilbene oxide enhances selectively the EH activity. Appropriate concentrations of CSCF or of phenobarbital (PB) determine a parallel induction of both enzymes. The results are discussed on the basis of (a) the existence of specific mechanisms of AHH regulation in the lung and in the kidney and (b) the existence of coordinated or independent biochemical control of the AHH and EH activity. (Auth. Abs.)

80–1108. Greenblatt, D. J.; Allen, M. D.; Harmatz, J. S.; Shader, R. I. **Diazepam Disposition Determinants.** *Clinical Pharmacology and Therapeutics* 27(3): 301-312, March 1980.

Factors influencing diazepam kinetics were assessed in four equal groups ($n = 11$) of young male and female (aged 21 to 37 years) and elderly male and female (aged 61 to 84 years) subjects, all of whom were healthy. In all 44, plasma diazepam concentrations were determined by electron-capture gas-liquid chromatography in multiple samples drawn for as long as 9 days after a single 5-10 mg intravenous dose. Based upon total (bound + free) diazepam concentrations, volume of distribution (V_d) ranged from 0.7 to 4.7 l/kg, and became larger both with increasing age and with female sex. Clearances of total (bound + free) diazepam in young and elderly females were nearly identical (0.51 and 0.48 ml/min/kg), but clearance was higher in young than in elderly males (0.39 and 0.24 ml/min/kg, $p < 0.01$). The unbound fraction of diazepam in plasma (range, 0.9-2.7 percent) did not depend on sex, but was greater in the elderly than in the young. After correction of kinetic data for individual differences in free fraction, V_d was larger in females than in males, but the effect of age was small. Clearance of unbound diazepam (intrinsic clearance) tended to be higher in females than in males of both age groups, and was higher in the young than in the elderly of both sexes (male: 29.9 and 14.9 ml/min/kg, $p < 0.005$; female: 43.6 and 28.0 ml/min/kg, $p < 0.05$). Smoking was associated with higher clearance values, particularly among young subjects. (Auth. Abs. Mod.)

80–1109. Hannan, M. A.; Estes, R. S.; Hurley, L. H. **Induction and Potentiation of Lethal and Genetic Effects of Ultraviolet Light by Tobacco Smoke Condensates in Yeast.** *Environmental Research* 21(1): 97-107, February 1980.

Tobacco smoke condensates (TSC) were tested for DNA repair inhibition in both repair proficient and different classes of repair deficient strains of *Saccharomyces cerevisiae*. The condensates were also tested for induction and potentiation of mutations and mitotic gene conversion in unirradiated and uv-irradiated yeast cells. Results showed that TSC sensitized all the strains of yeast to uv-inactivation indicating that it acts in a nonspecific manner and does not specifically inhibit a particular repair pathway. Genetic studies showed that TSC, without exogenous metabolic activation, failed to produce mutations while it induced mitotic gene conversion in the diploid strain. At specific concentrations, TSC potentiated both mutagenic and gene convertogenic effects of uv-light while at higher concentrations of TSC a reduction of mutations was observed. The results are discussed as they relate to carcinogenesis and cocarcinogenesis/tumor promotion. (Auth. Abs.)

80–1110. Hayashi, S.; Miyazaki, M.; Toda, N. **Responsiveness to Vasoactive Agents of Cerebral and Mesenteric Arteries Isolated from Control and Reserpine-Treated Dogs.** *British Journal of Pharmacology* 68(3): 473-478, March 1980.

Pretreatment of dogs for 20 to 24 hours before the start of experiments with reserpine (0.5 mg/kg) depleted noradrenaline from cerebral and mesenteric arteries, the diminution being greater in the latter arteries. Contractile responses of helically-cut strips of cerebral and mesenteric arteries to noradrenaline were unaffected by pretreatment with reserpine. Tyramine-induced contractions of mesenteric arteries were markedly attenuated by reserpine-pretreatment, whereas the contraction of cerebral arteries was not influenced. The addition of 10^{-4} M nicotine relaxed the basilar and middle cerebral arteries contracted with prostaglandin $F_{2\alpha}$, the relaxation being significantly greater in the latter arteries (13.4 ± 3.3 vs. 46.7 ± 5.6 percent, $n = 10$, relative to relaxations induced by 10^{-4} M papaverine; $p < 0.01$). The nicotine-induced relaxation was not significantly altered by pretreatment of dogs with reserpine. In mesenteric arterial strips, nicotine in a concentration of 10^{-4} M caused a contraction (565 ± 180 mg, $n = 13$), which was abolished by pretreatment with reserpine. In prostaglandin-contracted cerebral and mesenteric arterial strips, relaxant effects of acetylcholine, isoprenaline, and K^+ were not significantly influenced by reserpine-pretreatment. It appears that tyramine and nicotine do not release noradrenaline from dog cerebral arteries in amounts sufficient to cause significant contractions. Attenuation of the response to angiotensin II by pretreatment with reserpine is not the result of depletion of noradrenaline from the mesenteric arterial wall, but may be due to interference with the mechanism specific to actions of angiotensin II. (Auth. Abs. Mod.)

80–1111. Hayem, A.; Scharfman, A.; Laine, A.; Lebas, J.; Kopaczewski, A. **Composition Proteique et Enzymatique des Liquides de Lavage Bronchique et Broncho-Alveolaire de Six Sujets Normaux. [Protein and Enzyme Contents of the Bronchial and Broncho-Alveolar Washing Fluids From Six Normal Subjects.]** *Bulletin Europeen de Physiopathologie Respiratoire* 15(4): 611-624, 1979, French.

Biochemical analysis of bronchoalveolar and bronchial lavage fluids was carried out to compare protein and enzyme contents in fluids from deep and distal levels of the bronchial tree of normal subjects. The fluids were obtained from six healthy men, aged 30 to 35. Two of the subjects had been smoking more than 20 cigarettes/day for over 10 years. One subject was an occasional smoker. The immunoelectrophoretic pattern of the bronchoalveolar fluid of the two heavy smokers showed the presence of α-globulins which were not detected in nonsmokers. In the bronchoalveolar fluids of two smokers and one nonsmoker, the IgG content exceeded 30 percent vs. 25.3-26.8 percent for three nonsmokers. In the same fluids of one heavy smoker and the occasional smoker, the percentage of IgA 7S was found to be 30 and 21, respectively, vs. 7-16 for the other four subjects, including one of the heavy smokers. In the bronchial lavage fluid, IgA 7S was detected only in the smoker with 30 percent IgA 7S in his bronchoalveolar fluid. Glycosidase activity was distinctly higher in the fluids of the occasional smoker and much higher in the fluids of the two smokers than in the nonsmokers.

80–1112. Hinman, L. M.; Stevens, C. A.; Matthay, R. A.; Gee, J.B.L. **Elastase and Lysozyme Activities in Human Alveolar Macrophages. Effects of Cigarette Smoking.** *American Review of Respiratory Disease* 121(2): 263-271, February 1980.

The elastase and lysozyme activities of cells obtained by bronchoalveolar lavage from normal smokers and nonsmokers were compared. After total and differential cell counts were obtained for the initial lavage cell population, the enzyme activities of the total lavage cell population, the culture vessel's adherent alveolar macrophage cell fraction, and the cell culture supernatant medium were determined. Data indicated that macrophages, particularly from smokers, synthesized a calcium-dependent activity against a synthetic elastase substrate, succinyl-trialanine-*p*-nitroanilide. This activity was enhanced in smokers and was distinct from the polymorphonuclear leukocyte elastase as measured with this synthetic substrate. Measurements using insoluble elastin labeled with ^3H demonstrated that smokers' macrophages also contained a serine-proteinase activity whose inhibitor profile resembled that of polymorphonuclear leukocyte elastase. Finally, macrophages from smokers secreted five times more lysozyme and contained more lactate dehydrogenase activity than did nonsmokers' macrophages. (Auth. Abs. Mod.)

80–1113. Hirota, N. **Intranuclear Rodlets in Undifferentiated Carcinomas of Salivary Glands in Strain A Mice in a Study Involving a Tobacco Specific Nitrosamine, *N*-Nitrosonornicotine.** *Cancer Letters* 6(6): 365-369, May 1979.

Undifferentiated carcinomas of the salivary glands were found in 2 of 44 (4.5 percent) strain A mice injected intraperitoneally with a tobacco-specific nitrosamine, *N*-nitrosonornicotine (NNN). The presence of intranuclear rodlets (INR) in the salivary carcinomas provided the first demonstration of such structures in a non-neuronal tumor in mice. Two types of rodlets were exhibited; one was composed of fibrillar filaments arranged in bundles, and the other was much thicker, branching in form. These INR appeared to be closely associated with nuclear chromatin or nucleoli. (Auth. Abs.)

80–1114. Hocking, W. G.; Golde, D. W. **The Pulmonary-Alveolar Macrophage. (First of Two Parts).** *New England Journal of Medicine* 301(11): 580-587, September 13, 1979.

Morphologic features, metabolic characteristics, and antimicrobial mechanisms of pulmonary alveolar macrophages (PAMs) are discussed and data from various human studies are reviewed. Marked morphological changes in PAMs were caused by cigarette smoke. Smokers had PAMs that were slightly larger in diameter than those from nonsmokers. Characteristic inclusions were seen in electron micrographs of PAMs obtained from smokers. The needle-like structures in the inclusions were platelike, hexagonal crystals similar to those of kaolinite, an aluminum silicate present in cigarette smoke from several different brands. Although found predominantly in tobacco smokers, these crystalline inclusions have also been seen in cells from marihuana smokers and nonsmokers.

80–1115. Hoidal, J. R.; Fox, R. B.; LeMarbre, P. A.; Takiff, H. E.; Repine, J. E. **Oxidative Metabolism of Alveolar Macrophages from Young Asymptomatic Cigarette Smokers. Increased Superoxide Anion Release and Its Potential Consequences.** *Chest* 77(2): 270-271, February 1980.

To study the mechanism by which alveolar macrophages (AM) may cause emphysema, the oxidative metabolism of AM from nonsmokers and young asymptomatic cigarette smokers was compared. Alveolar macrophages were obtained by bronchial lavage from 43 nonsmoking volunteers (ages 18 to 30 years) and 34 age-matched smokers (1 pack/day minimum, mean 7 pack/years). The first experiment compared oxygen (O_2) uptake, superoxide anion (O_2^-) release, and glucose ($1\text{-}^{14}C$) oxidation by AM from smokers and nonsmokers. It was found that oxygen uptake and glucose ($1\text{-}^{14}C$) oxidation by stimulated or unstimulated AM were similar in smokers and nonsmokers. In contrast, O_2^- release by AM from smokers was significantly greater ($p<0.05$) than for nonsmokers before or after stimulation. The second experiment evaluated possible mechanisms of altered O_2^- release by AM from smokers. Superoxide dismutase (SOD) activity of AM from smokers was markedly increased. The third experiment was designed to examine potential consequences of enhanced O_2^- release by AM from smokers. Macrophages from smokers killed human fibroblasts in culture and this cytocidal activity increased when the macrophages were stimulated by phorbol myristate acetate. The augmented cytocidal activity of stimulated AM was inhibited by addition of SOD and catalase. Superoxide dismutase and catalase inhibitable O_2 intermediates from smokers' AM promoted release of elastolytic agents from these phagocytes. These findings support direct and indirect methods by which increased O_2^- release by smokers' AM leads to tissue injury.

80–1116. Huber, G. L.; Pochay, V. E.; Pereira, W.; Shea, J. W.; Hinds, W. C.; First, M. W.; Sornberger, G. C. **Marijuana, Tetrahydrocannabinol, and Pulmonary Antibacterial Defenses.** *Chest* 77(3): 403-410, March 1980.

Although marihuana is now consumed extensively, little is known of its biologic effects on the lung. To study this problem, the intrapulmonary inactivation of an aerosolized challenge of *Staphylococcus aureus* was quantified in rats exposed to graded amounts of fresh marihuana smoke. Controls inactivated 85.1 ± 0.3 percent of the bacteria 6 hours after inoculation. Following an in vivo accumulative exposure to smoke from progressively increasing numbers of marihuana cigarettes for periods of 10 minutes each hour for 5 consecutive hours, intrapulmonary bacterial inactivation was impaired in a dose-dependent manner. Evaluation of the effects of parenterally administered delta-9-tetrahydrocannabinol (THC) or of exposure to fresh smoke from THC-extracted marihuana placebo cigarettes indicated that the cytotoxin in marihuana was not related to the primary psychomimetic component. Thus, marihuana smoke is toxic to the lung and impairs the pulmonary antibacterial defense system in a dose-dependent manner. (Auth. Abs.)

80–1117. Hugod, C.; Astrup, P. **Exposure of Rabbits to Carbon Monoxide and Other Gas Phase Constituents of Tobacco Smoke. Influence on Coronary- and Aortic Intimal Morphology.** *Muenchener Medizinische Wochenschrift* 122(Supplement 1): S18-S24, February 20, 1980.

Noncholesterol-fed rabbits were exposed to carbon monoxide (CO) at concentrations in air of either 200, 2,000, or 4,000 ppm. They were also exposed to 0.5 ppm hydrogen cyanide alone or in combination with 200 ppm carbon monoxide, or with 200 ppm carbon monoxide and 5 ppm nitric oxide, and eventually with 50 ppm carbonyl sulphide. Durations of continuous exposure were between 1/2 week and 12 weeks. Using the same criteria for intimal damage as in earlier morphological studies, no histotoxic effect on intimal/subintimal morphology of coronary arteries or the aorta could be demonstrated when a blind light-microscopic evaluation was performed. (Auth. Abs.)

80–1118. Hunninghake, G.; Gadek, J.; Crystal, R. **Mechanism by Which Cigarette Smoke Attracts Polymorphonuclear Leukocytes to Lung.** *Chest* 77(2): 273, February 1980.

Since alveolar macrophages (AM) interact with cigarette smoke in vivo and are capable of releasing a chemotactic factor (CF) for PMN, the present study was undertaken to determine if cigarette smoke might stimulate AM to release a CF for PMN in vitro. The findings in six cigarette smokers showed that alveolar macrophages obtained by bronchoalveolar lavage (BAL) spontaneously released CF for PMN (100 ± 15 PMN/hpf) and, as an in vivo correlate, their BAL contained 6 ± 1 percent PMN. In five nonsmoking controls, AM did not spontaneously release CF for PMN (5 ± 1 percent PMN/hpf) and their BAL contained <1 percent PMN. In addition, normal AM could be stimulated to release CF for PMN by exposing them in vitro to cigarette smoke (85 ± 10 PMN/hpf).

80–1119. Husum, B.; Wulf, H. C. **Sister Chromatid Exchanges in Lymphocytes in Operating Room Personnel.** *Acta Anaesthesiologica Scandinavica* 24(1): 22-24, February 1980.

Sister chromatid exchanges (SCE) and exchange points were counted in lymphocytes in peripheral blood drawn from hospital personnel exposed to anesthetics as well as from persons not exposed. A total of 38 healthy persons (15 of whom were smokers) were investigated, representing female nurse anesthetists, male physicians practicing anesthesia, female nurses from the intensive care unit, and female secretaries. The mean SCE number per cell for each person was used as the variable, and the Mann-Whitney U-test was applied to test for differences between groups. The group of secretaries seemed to differ from the other three groups, which appeared identical ($p<0.002$). Correlation of cigarette smoking and number of SCE could not be demonstrated ($r=0.255$, $n=38$). It was concluded that by this method there was no indication of a mutagen effect of long-term exposure to waste anesthetic gases such as halothane and nitrous oxide. (Auth. Abs. Mod.)

80–1120. Ianysheva, N. Ia.; Balenko, N. V.; Chernichenko, I. A. **O Blastomogennom Effekte Benz(*a*)pirena pri Drobnom Postuplenii v Organy Dykhaniya. [The Blastomogenic Effect of Benzo(*a*)pyrene Fractionalized Intake in Respiratory Organs.]** *Gigiyena i Sanitariya* 43(7): 8-11, 1978, Russian.

A single or serial total 0.005-25 mg dose of benzo(*a*)pyrene (BaP) was instilled intratracheally into white rats. The animals were under observation until their natural death. When a total dose of 2.5 mg BaP was instilled in 10 fractions, three times as many rats developed tumors as after a single 2.5 mg dose, and six times as many developed malignant tumors with some metastases. With a total dose of 0.5 mg BaP instilled in 10 fractions vs. a single dose, 28.2 vs. 5.9 percent of tumors and 15.7 vs. 0 percent of malignant tumors were found. The effect of the single 2.5 mg dose was 13.0 percent of total tumors and 4.3 percent of malignant tumors. Only 14.4 percent of rats developed benign tumors after taking 10-0.1 mg doses. The higher the degree of BaP dose fractionation, the slower was blastomogenic growth in the lungs. In contrast with a single 2.5 mg BaP dose, the same total dose administered in 10 fractions caused development of reticulosarcomas in addition to the epithelial tumors. A 25 mg dose in 10 fractions caused development of combined squamous cell, adenocarcinoma, and a nondifferentiated small cell carcinoma. There is, however, a threshold dose below which BaP fractionation does not have any adverse effect during the natural lifespan. For these experiments, the threshold dose was 0.02 mg.

80–1121. Klein-Szanto, A.J.P.; Topping, D. C.; Heckman, C. A.; Nettesheim, P. **Ultrastructural Characteristics of Carcinogen-Induced Nondysplastic Changes in Tracheal Epithelium.** *American Journal of Pathology* 98(1): 61-68, January 1980.

Nondysplastic hypotrophic and metaplastic epithelial alterations induced by dimethylbenz(*a*)anthracene in isogenic tracheal transplants in rats were studied by light and electron microscopy 3-24 months after cessation of a 4-week carcinogen exposure. Hypotrophic epithelium observed at all time points was characterized by the presence of nonciliated cells that adopted either cuboidal or squamous shapes, forming simple or bistratified epithelia. Most of these cells, as well as some metaplastic cells, exhibited features of mucin-secreting cells. The metaplastic epithelia showed nonkeratinizing squamous metaplasia, closely related to transitional metaplasia, and keratinizing squamous metaplasia, which presented either an atrophic or an acanthotic epithelium. Although many of these epithelia showed morphologic features of normal stratified epithelia, several nonkeratinizing squamous metaplasias and acanthotic keratinizing squamous metaplasias exhibited some irregularities, probably representing very early atypical ultrastructural features (i.e., perinuclear concentration of tonofilament bundles, the presence of dark and clear basal epithelial cells, interruptions and alterations of the basal lamina). These features were not observed in a group of early squamous metaplasias studied for comparative purposes 2 weeks after cessation of dimethylbenz(*a*)anthracene exposure, which were characterized by a combination of degenerative phenomena and increased cell proliferation. (Auth. Abs.)

80–1122. Kouri, R. E.; Rude, T. H.; Curren, R. D.; Brandt, K. R.; Sosnowski, R. G.; Schechtman, L. M.; Benedict, W. F.; Henry, C. J. **Biological Activity of Tobacco Smoke and Tobacco Smoke-Related Chemicals.** *Environmental Health Perspectives* 29: 63-69, April 1979.

Exposure to whole cigarette smoke from reference cigarettes results in the prompt (peak activity is 6 hours), but fairly weak (approximately twofold), induction of murine pulmonary microsomal monooxygenase activity in rats. This activity can be detected by using either benzo(*a*)pyrene (BaP) or ethoxyresorufin as substrates, and can be inhibited by treatment with cycloheximide or actinomycin D. Unlike the induction of pulmonary monooxygenases following intratracheal administration of 3-methylcholanthrene, these cigarette smoke-induced increases were not unequivocally linked to the Ah locus. Whole smoke condensate and fractions derived from these condensates can (a) induce pulmonary monooxygenase activity, (b) inhibit BaP metabolism in vitro, (c) be metabolized to forms mutagenic to *Salmonella typhimurium* tester strains TA1538 or TA98, (d) transform C3H 10T1/2 cells in vitro, and (*e*) enhance the carcinogenicity of BaP in murine pulmonary tissue. A potentially important observation is that whereas hepatic tissue is capable of activating whole cigarette smoke condensate to mutagenic forms in vitro, murine pulmonary tissue does not seem capable of such activation. Although these pulmonary-derived tissue homogenates have significant aryl hydrocarbon hydroxylase activity and can metabolize Aflatoxin B_1, 2-aminofluorene, and 7,8-dihydro-7,8-dihydroxybenzo(*a*)pyrene to mutagenic forms, these homogenates fail to activate both cigarette smoke condensate and the pro-mutagen, 6-aminochrysene. These results are discussed with reference to the concept that whole cigarette smoke may be both a potential "initiator" and "promoter" of lung cancer in mice, and that this latter property may be the most important in determining cancer risk. (Auth. Abs.)

80–1123. Le Mesurier, S. M.; Lykke, A.W.J.; Stewart, B. W. **Reduced Yield of Pulmonary Surfactant: Patterns of Response Following Administration of Chemicals to Rats by Inhalation.** *Toxicology Letters* 5(1): 89-93, January 1980.

Repeated exposure of rats to trichloroethylene, carbon tetrachloride, gasoline vapor, or cigarette smoke is followed by significant reduction in the recovery of pulmonary surfactant, inhibition being evident as early as 5 days after commencement of treatment. As compared to 19.75 ± 0.87 mg surfactant for controls at 5-15 days, cigarette smoke inhalation resulted in 16.08 ± 0.85 and 9.46 ± 0.14 mg for 5 and 15 days, respectively. The degree of reduction in surfactant recovery was dose-dependent and the kinetics of this reaction indicated the relative toxicities of the treatments. The results are discussed with reference to the use of surfactant recovery as an indicator of nonspecific injury to respiratory tissue. (Auth. Abs. Mod.)

80–1124. Lippold, O.C.J.; Wilson, C. G. **The Effect of Cigarette Smoking on Finger Tremor.** *British Journal of Clinical Pharmacology* 6(5): 457P-458P, November 1978.

The effect of cigarette smoking on finger tremor was studied in 35 subjects aged 17-63 years. For 13 of the smokers, urine samples were collected before and 2 hours after smoking and analyzed for nicotine and cotinine content. An increase in tremor of 1-12 Hz was seen in 30 of the 35 subjects during smoking. The five subjects who showed no change described themselves as "noninhalers." There was no apparent correlation between the amounts of nicotine or cotinine in the post-smoking urine samples and the magnitude of the increases in tremor produced by smoking.

80–1125. Lorenz, J.; Schmassmann, H.; Ohnhaus, E.; Oesch, F. **Activities of Polycyclic Hydrocarbon Activating and Inactivating Enzymes in Human Lungs of Smokers, Non-Smokers, Lung-Cancer and Non-Cancer Patients.** *Archives of Toxicology* (Supplement 2): 483-489, 1979.

Human lungs are an important target organ for environmental carcinogenic polycyclic hydrocarbons (PAH), which can be metabolized by microsomal monooxygenases (MO) to mutagenic epoxides. The concentrations within the cells of these reactive epoxides, which are suspected ultimate carcinogens, will be controlled not only by the MO activities, but also by the epoxide inactivating epoxide hydratase (EH) and glutathione-S-transferase (GT) activities. As no data exist on these enzyme activities in human lungs, a study was made as to whether a correlation exists between these epoxide forming and inactivating enzyme activities and the occurrence of lung cancer by measuring these enzyme activities in 47 samples from lungs of smokers, nonsmokers, lung cancer, and noncancer patients. Of the 37 smokers, 30 had lung cancer, compared to 2 of the 10 nonsmokers. Using standard assays, no MO activity could be detected, although very sensitive assays with benzo(*a*)pyrene, 7-ethoxycoumarin, 7-

ethoxyresorufin, and biphenyl as substrates were used while these activities in rat lung microsomes used as positive controls were always clearly measurable. Evidence was obtained for the presence of a diffusible MO-inhibitor in human lung microsomes. Finally 7-ethoxycoumarin-O-dealkylase activity could be measured after modification of the assay. The EH and GT activities were determined with benzo(*a*)pyrene 4,5-oxide; the latter was also measured with 2,4-dinitrochlorobenzene as substrate. Considerable interindividual variations in enzyme activities were observed. However, no significant differences were found when the enzyme activities in lungs from noncancer patients and nonsmokers were compared with those in lung cancer patients. The substrate specificity of human lung epoxide hydratase investigated using K-region epoxides of various PAH was similar to that in rat liver and in human, rat, and mouse skin. (Auth. Abs. Mod.)

80–1126. Marcus, A. H.; Czajkowski, S., Jr. **First Passage Times as Environmental Safety Indicators: Carboxyhemoglobin from Cigarette Smoke.** *Biometrics* 35(3): 539-548, September 1979.

The concentration of carbon monoxide (CO) in the blood of a cigarette smoker varies in response to the frequency and dose of CO delivered by the cigarettes he smokes and by the rate at which CO washes out of his blood. Moments of first passage times or exit times above a nominal threshold can be calculated using a stochastic differential equation that takes into account certain random variations in smoking intervals, CO doses, and washout rate. Almost any additional source of variation decreases in length of time a smoker may expect to smoke until a threshold value is exceeded. One possible means of reducing CO hazards is to smoke less from each cigarette, even when this may be partially compensated by increased frequency of smoking. (Auth. Abs. Mod.)

80–1127. Mitra, G.; Poddar, M. K.; Ghosh, J. J. **Effect of Tobacco-Leaf Extract Administration on Liver, Lung, Intestine, and Serum Enzymes.** *Toxicology and Applied Pharmacology* 52(2): 262-266, February 1980.

Tobacco leaf extract induced benzpyrene hydroxylase activities of liver and lung of male albino rats after 7, 30, and 62 days of oral administration. Aniline hydroxylase activity of liver and lung showed an increase after 30 days of administration, while the *N*-demethylase activity of liver showed a small increase at the first phase and a decrease after 62 days. Of the lysosomal enzymes investigated, β-glucuronidase activity of both liver and lung showed a significant increase. Increased activities of hepatic lysosomal acid phosphatase was observed at 7 and 30 days of leaf extract administration. A decrease was observed in sucrase and maltase activities of intestinal brush border after 7 and 30 days of tobacco leaf extract administration. No significant change in serum enzymes was observed under the conditions of the present study. (Auth. Abs.)

80–1128. Pelkonen, O.; Moilanen, M.-L. **The Specificity and Multiplicity of Human Placental Xenobiotic-Metabolizing Monooxygenase System Studied by Potential Substrates, Inhibitors and Gel Electrophoresis.** *Medical Biology* 57(5): 306-312, October 1979.

The specificity of the placental monooxygenase system to metabolize foreign compounds was studied by using different potential substrates and inhibitors and by performing electrophoresis of placental microsomes. Placental preparations from smokers catalyzed benzo(*a*)pyrene (BaP) hydroxylation, 7-ethoxycoumarin *o*-deethylation, and 2,5-diphenyloxazole hydroxylation, but not biphenyl hydroxylation at 2-, 3-, or 4-carbon, aldrin epoxidation to dieldrin or coumarin hydroxylation, or to aminopyrine N-demethylation. Enzyme activities were inhibited by α-naphthoflavone, but to a much lesser extent by SKF 525-A or metyrapone. Correlations between the metabolism of BAP, 7-ethoxycoumarin, and 2,5-diphenyloxazole were highly significant. There was a clear difference in Michaelis-Menten constant of 7-ethoxycoumarin *o*-deethylation between placentas from smokers and nonsmokers. Gel electrophoresis revealed that protein bands of placental microsomes in the region of cytochrome P-450 enzymes were less prominent than those of rat liver microsomes, a finding that accorded with the relative amounts of cytochrome P-450. There were no consistent differences in the electrophoretic pattern between placentas of variable benzo(*a*)pyrene hydroxylase activities. Results show that the human placental monooxygenase system is restricted in substrate specificity, that there may be a qualitative difference between smokers and nonsmokers, and that the increase in several enzyme activities by cigarette smoking cannot be detected by the standard gel electrophoresis. (Auth. Abs.)

80–1129. Polak, E. **Le Papier a Cigarette. Son Role Dans la Pollution des Lieux Habites. Tabagisme Passif: Notion Nouvelle Precisee. [Cigarette Paper. Its Role in Pollution of Dwellings. Passive Smoking: The New Concept Refined.]** *Bruxelles Medical* 57(8): 335-340, August 1977, French.

Two experiments were carried out to determine the role of commercial cigarette paper impregnated with potassium nitrate or magnesium citrate in polluting dwellings and in affecting the health of nonsmokers. In the first experiment, 10 smokers smoked 4 commercial cigarettes during 1 hour at the rate of one puff/min in a 60 m³ room in the presence of 5 nonsmokers. The experiment was repeated with four cigarettes rolled in untreated paper. Carbon monoxide (CO) content in the air after the first and the second experiments was found to be 23 and 18 ppm, respectively. In nonsmokers' blood, the average increase of carboxyhemoglobin (COHb) level due to commercial (combustible paper) and rolled cigarette smoke was 0.30 and 0.10 percent/hour, respectively. In another experiment whose focus was lung function, six subjects smoked six cigarettes each in 1 hour in a 180 m³ room in the presence of seven nonsmokers. In smokers, average decreases of 10 percent of their vital capacity (VC), 7 percent of their midexpiratory volume (MEV_1) in 1 sec, and

4.5 percent of the Tiffeneau ratio were recorded. In passive smokers, VC and MEV_1 decreased an average of 5 and 1 percent, respectively, while the Tiffeneau ratio decreased by over 10 percent in two cases and remained either the same or slightly higher in five cases.

80–1130. Prough, R. A.; Capdevila, J.; Lubet, R. A. **Metabolic Activation of Benzo(*a*)pyrene and 9-Hydroxybenzo(*a*)pyrene by Tissue Fractions From Rat Liver and Lung.** *Biochemical Society Transactions* 7(1): 122-124, 1979.

Microsomal fraction-mediated mutagenesis in *Salmonella typhimurium* and DNA alkylation from benzo(*a*)pyrene and 9-hydroxybenzo(*a*)pyrene was studied in rats using tissue fractions from liver and lung. Results indicate that 9-hydroxybenzo(*a*)pyrene is a better premutagen and prealkylating agent than is benzo(*a*)pyrene when metabolically activated by tissue fractions from 5,6-benzoflavone-treated rats.

80–1131. Putz, V. R.; Johnson, B. L.; Setzer, J. V. **A Comparative Study of the Effects of Carbon Monoxide and Methylene Chloride on Human Performance.** *Journal of Environmental Pathology and Toxicology* 2(5): 97-112, May-June 1979.

The purpose of this investigation was to determine whether similar carboxyhemoglobin (COHb) values obtained from inhaling carbon monoxide or methylene chloride have the same behavioral effects. The effects were assessed by the performance of 12 humans (six males and six females, all of whom were nonsmokers) on a visual-manual, dual-task, and an auditory vigilance task. The results indicated that both substances in concentrations sufficient to produce 5 percent COHb significantly impaired human performance under difficult or demanding task conditions. The conclusion was that carbon monoxide, the main metabolite of methylene chloride, was responsible for the observed performance decrements. (Auth. Abs. Mod.)

80–1132. Queval, P.; Beaumatin, J.; Morin, M.; Courtois, D.; Lafuma, J. **Inducibility of Microsomal Enzymes in Normal and Pre-Cancerous Lung Tissue. Synergistic Action of 5-6 Benzoflavon or Methyl-Cholanthrene in Radiation Induced Carcinogenesis.** *Biomedicine* 31(6): 182-186, October 1979.

The inducibility of pulmonary microsomal enzymes and the biological effects of chronic induction by 5-6 benzoflavone and methylcholanthrene were compared in normal and precancerous rats. Two important points were illustrated: (1) a dose of 6,000 working level month resulted in a permanent modification of the pulmonary microsomal enzymatic pool and (2) 5-6 benzoflavon was a strong cofactor of radiation-induced carcinogenesis. This synergistic action was demonstrated by a much shorter latency; moreover, neoformed tissue and epidermoid cancers with a particular morphology were observed. In all the cases cancers appeared within 100 days following the beginning of the chemical treatment. (Auth. Abs.)

80–1133. Reddy, M. S.; Naik, S. R.; Bagga, O. P.; Chuttani, H. K. **Effect of Chronic Tobacco-Betel-Lime "Quid" Chewing on Human Salivary Secretions.** *American Journal of Clinical Nutrition* 33(1): 77-80, January 1980.

Salivary flow rates by mechanical stimulation with forced spitting method and by chemical stimulation with 10 percent citric acid were determined in 25 healthy adult subjects with a history of chronic tobacco-betel-lime "quid" chewing and in 25 healthy control adults with no history of chewing. The chewers secreted more saliva as compared to nonchewers on chemical ($p<0.05$), but not on mechanical stimulation. The salivary amylase and potassium levels were significantly lower in chewers as compared to nonchewers ($p<0.05$ and $p<0.01$, respectively). Salivary sodium was also lower in chewers, but the difference was not statistically significant. These reductions were thought to be due to increased salivary flow with its dilutional effect. There was no difference between the two groups with respect to salivary pH. The salivary flow rates by either method had significant positive correlation with the duration of chewing ($p<0.02$), but not with the amount of tobacco chewed. Salivary potassium was inversely correlated with the amount of tobacco chewed ($p<0.01$). It was concluded that chronic tobacco-betel-lime quid chewing induces excessive secretion of more watery saliva leading to a concomitant decrease in enzyme and electrolyte content. One or more of the following factors were considered to be operating in causing increased salivary flow in chewers—effect of nicotine or tobacco on other constituents of the quid, chronic salivary gland hyperplasia, or chronic hypertrophy of the muscles of mastication. (Auth. Abs. Mod.)

80–1134. Reznick-Schueller, H. M.; Tomaszewski, J. E. **Binding of Radioactivity After Transplacental Administration of Tritiated *N*-Nitrosodiethylamine to Syrian Golden Hamsters.** *Journal of the National Cancer Institute* 64(2): 373-375, February 1980.

Tritiated *N*-nitrosodiethylamine (DEN) was administered to two groups of female Syrian golden hamsters on days 11 and 15 of pregnancy. Binding of radioactivity was measured in maternal and fetal organs after various intervals by liquid scintillation counting of dehydrated and combusted tissues. No bound activity was found in the fetal tracheas on day 11 of gestation, when transplacental administration of DEN is known to be noncarcinogenic for the offspring. On day 15, when DEN administration caused a 95 percent incidence of tracheal tumors in the offspring, bound radioactivity was found in the fetal tracheas. In the respiratory tracts of the mothers, the distribution of bound radioactivity correlated with the distribution of target cell types. Binding was high in segmental bronchi and bronchioles which contain numerous Clara cells, the major source of DEN-induced pulmonary tumors. No binding occurred in the main bronchi, which do not possess Clara cells. (Auth. Abs.)

80–1135. Rodgers, R. J. **Effects of Nicotine, Mecamylamine, and Hexamethonium on Shock-Induced Fighting, Pain Reactivity, and Locomotor Behaviour in Rats.** *Psychopharmacology* 66(1): 93-98, November 1979.

Three series of experiments were performed to evaluate possible nicotinic cholinergic influences on fighting behavior in rats. Each series consisted of three tests (naive animals in each test): shock-induced fighting, pain threshold estimation, and locomotor activity. In the first series, nicotine (0.25-1.00 mg/kg) was found to produce a dose-dependent inhibition of fighting without altering shock thresholds. However, the highest dose used also significantly reduced rearing in the activity test. In the second series, mecamylamine (a centrally active antinicotinic) produced a facilitation of fighting at low doses (2.5 mg/kg) and an inhibition at higher doses (10 mg/kg). While these effects were unrelated to changes in shock thresholds, the high dose resulted in a reduction in both horizontal activity and rearing. Finally, as a control for possible peripheral effects of nicotinic blockade, a third series examined the behavioral effects of hexamethonium. Low doses of this compound (2.25-4.5 mg/kg) had little effect on fighting while higher doses (9-18 mg/kg) attenuated these responses. Interestingly, although hexamethonium had no effect on shock thresholds, the highest dose (18 mg/kg) produced a facilitation of horizontal activity. Results are discussed in relation to the hypothesis of central nicotinic cholinergic inhibition of agonistic behavior. (Auth. Abs.)

80–1136. Rodgers, R. L.; Moore, J. I.; Hornbrook, K. R. **Comparison of the Cardiostimulatory Effects of Acetylcholine and Nicotine on the Working Guinea-Pig Heart.** *Naunyn-Schmeideberg's Archives of Pharmacology* 310(2): 97-101, December 1979.

The isolated working guinea-pig heart was used to compare the cardiostimulatory effects of acetylcholine and nicotine observed in the presence of atropine. Both agonists increased aortic pressure, left ventricular pressure, cardiac output, and ventricular cyclic AMP levels. These responses were qualitatively and quantitatively similar to the effects of exogenous norepinephrine. Hexamethonium treatment abolished the responses to acetylcholine and to nicotine. However, several differences in the responses of the two agonists were also observed with respect to (1) the effect of propranolol pretreatment, (2) selective effects on coronary and aortic flow rates, (3) coefficients of correlation between ventricular cyclic AMP and changes in left ventricular pressure, and (4) the "autoinhibition" effect. The results support the view that the cardiostimulatory effects of acetylcholine are due entirely to endogenous catecholamine release, but that the effects of nicotine may involve an additional action. (Auth. Abs. Mod.)

80–1137. Russell, M.A.H.; Jarvis, M. J.; Feyerabend, C. **A New Age For Snuff?** *Lancet* 1(8166): 474-475, March 1, 1980.

The absorption of nicotine by cigar smokers and snuff users was studied by measuring blood nicotine levels during noninhaled cigar smoking and after taking snuff. The rate of nicotine absorption from noninhaled cigar smoking was slow, which may explain why many cigarette smokers continue to inhale when they switch to cigars. In contrast, the rate of absorption from snuff was extremely rapid. Peak levels which matched those of cigarette smoking were reached within 5 min. It is concluded that snuff could be an acceptable and less harmful substitute for cigarette smoking. (Auth. Abs. Mod.)

80–1138. Spohr, U.; Hengen, N.; Harenberg, J.; Walter, E.; Augustin, J.; Comberg, H. U.; Moerl, H.; Weber, E. **Nikotinwirkungen nach Zigarettenrauchen auf Kreislauf und Stoffwechsel unter Berucksichtigung der Plasmanikotin- und CO-Hb-Spiegel. [The Effects of Nicotine on Circulation and Metabolism After Cigarette Smoking With Reference to the Plasma Levels of Nicotine and COHb.]** *Muenchener Medizinische Wochenschrift* 122(Supplement 1): S25-S32, February 20, 1980, German.

The effects of smoking cigarettes with different nicotine contents (1.5 mg and 0.08 mg nicotine/cigarette) were investigated in six healthy habitual smokers. Carboxyhemoglobin (COHb) and plasma nicotine levels were determined at the same time as circulatory parameters, heart rate, and blood pressure, together with metabolic parameters like blood sugar, lactate, free fatty acids, and plasma dopamine-β-hydroxylase (DBH) and cortisol. With the exception of COHb, the parameters investigated were shown to be dependent on the nicotine levels. For blood pressure, heart rate, and lactate the peaks appeared simultaneously with the maximum nicotine levels, whereas for DBH, cortisol, blood sugar, and free fatty acids there was a delayed reaction in comparison with the nicotine levels. The parameters investigated are not affected by COHb with levels up to 5.6 ± 0.5 percent. These results show that after cigarette smoking, nicotine causes considerable changes in the circulation and metabolism. (Auth. Abs.)

80–1139. Tsibris, J.C.M. **Carcinogen Activation by Human Uterine Enzymes.** *Annals of Clinical and Laboratory Science* 9(3): 236-242, May-June 1979.

Three topics were briefly reviewed relating to carcinogenesis of estrogen-responsive tissues: (a) enzymology of benzo(*a*)pyrene (BaP) activation by human tissues (placenta, ovary, myometrium), (b) microsomal activation of estrogens to estrogen arene oxides, and (c) estrogen and progesterone receptor studies in endometrial carcinoma. A working hypothesis on the etiology of gynecologic tumors stated that environmental chemicals, such as cigarette smoke and polycyclic and polyhalogenated hydrocarbons, induce special forms of cytochrome P-450 monooxygenase and related enzyme systems which can activate endogenous or prescribed estrogens and nonsteroid antiestrogens to act as initiators and/or promoters of neoplasia in estrogen-dependent organs. The role of estrogen receptors was perceived as a cellular device which transports the activated estrogen to a specific site on chromatin. (Auth. Abs. Mod.)

80–1140. Van Doorn, R.; Bos, R. P.; Leijdekkers, C.-M.; Wagenaas-Zegers, M.A.P.; Theuws, J.L.G.; Henderson, P. T. **Thioether Concentration and Mutagenicity of Urine from Cigarette Smokers.** *International Archives of Occupational and Environmental Health* 43(3): 159-166, June 15, 1979.

Urinary thioether compounds, such as mercapturic acids, can be considered as nontoxic end products of potentially alkylating agents. On the other hand, urinary mutagenicity may represent the excretion of potential mutagens that have not definitively been detoxified by the organism. It is suggested that a combined urinary thioether and *Salmonella typhimurium* mutagenicity test may be useful in monitoring people occupationally exposed to potentially alkylating compounds, in particular to mixtures of these chemicals. Exposure to cigarette smoke, containing several known mutagens and carcinogens, is expected to interfere with the test results. The excretion of mutagens and thioethers was determined in the urine of smokers and nonsmokers. Persons smoking up to 10 cigarettes/day did not show a significant increase in the mutation frequency when compared with nonsmokers. Urine mutagenicity in the group of persons smoking 11-20 cigarettes/day and in the group of persons smoking more than 20 cigarettes daily significantly increased ($p<0.05$) when compared with the preceding group. Smoking up to about 10 cigarettes/day caused no statistically significant change in the mutation frequency whereas the urinary thioether excretion shows a remarkable increase ($p<0.01$). On the other hand, for the heavy smokers it appeared that the urinary mutation frequency increased strongly, while there was hardly any increase in the urinary thioether excretion. (Auth. Abs. Mod.)

80–1141. Wehner, A. P.; Stuart, B. O.; Sanders, C. L. **Inhalation Studies with Syrian Golden Hamsters.** *Progress in Experimental Tumor Research* 24: 177-198, 1979.

The Syrian golden hamster has been used for chronic inhalation studies in which investigations were made of the biological effects of cigarette smoke, chrysotile asbestos, nickel oxide (NiO), cobalt oxide (CoO), the latter three materials in combination with cigarette smoke, talc baby powder, radon daughters, uranium ore dust, diesel engine exhaust, radon daughters in combination with uranium ore dust, and radon daughters in combination with uranium ore dust and diesel engine exhaust, asbestos cement, fly ash, and plutonium dioxides. Exposure to asbestos (23μg/l respirable aerosol concentration, 7 hours/day, 5 days/week) for 11 months resulted in asbestosis in all animals, in an increased incidence of lung adenomas, indicating an early neoplastic response, and in a shorter life span. Life span exposures to NiO (53μg/l) and to CoO (10μg/l) resulted in pneumoconiosis. Life span exposure to cigarette smoke (3 times/day, 5 days/week, in Hamburg II smoking machines) significantly decreased body weight ($p<0.01$) and increased the incidence of tumors ($p<0.05$) and epithelial lesions of the larynx ($p<0.01$). The increased incidences in the smoke-exposed hamsters were not necessarily caused by the smoke exposures, but might be explained by the significantly longer life span of the smoke-exposed hamsters. (Auth. Abs. Mod.)

See also, **80–1246**

MORTALITY AND MORBIDITY

80–1142. Dyer, A. R.; Stamler, J.; Paul, O.; Lepper, M.; Shekelle, R. B.; McKean, H.; Garside, D. **Alcohol Consumption and 17-Year Mortality in the Chicago Western Electric Company Study.** *Preventive Medicine* 9(1): 78-90, January 1980.

The association between baseline alcohol intake and mortality from all causes and specific causes based on 17 years of followup experience was analyzed for 1,832 white males originally aged 40-55 from the Chicago Western Electric Company study. Alcohol consumption was expressed as number of drinks per day for all intake combined, including hard liquor, beer, and wine, based on the maximum intake reported on three questionnaires. With mortality rates adjusted only for age, total intake was associated with increased risk of death from all causes, the cardiovascular diseases, coronary heart disease, cancer, and other causes at the level of six or more drinks per day. Although some of the increased mortality in this six or more per day group was explainable by their heavier cigarette usage (18.7 cigarettes/day compared to 10.2/day for those consuming <6 drinks a day), there remained an excess that could not be explained. There was no increase in mortality with increasing alcohol consumption below this level. Only the associations between alcohol intake and death from the cardiovascular diseases and coronary heart disease failed to persist after adjustment for other risk factors, such as smoking and blood pressure. When the deaths were divided into those occurring within the first 10 years of followup and those occurring more than 10 years after entry, the association between baseline alcohol intake and mortality was generally stronger for those deaths occurring more than 10 years after entry. (Auth. Abs. Mod.)

80–1143. Garrow, J. S. **Weight Penalties.** *British Medical Journal* 2(6199): 1171-1172, November 10, 1979.

The literature on the relationship between obesity and mortality is reviewed. A study of 4.5 million insured lives over the 1935-1954 period indicated that there is a "desirable range" of weight for men and women which is associated with greatest longevity. An American Cancer Society study showed that men and women smoking 20 cigarettes or more a day had by far the highest mortality from all causes in all weight categories, while nonsmokers showed low mortality rates. Studies of longshoremen and civil servants showed that smokers had increased mortality, especially from heart attack and respiratory disease. It is concluded that the effect of smoking on mortality seems to be separate from that of overweight.

80–1144. Lee, P. N. **Has the Mortality of Male Doctors Improved With the Reductions in Their Cigarette Smoking?** *British Medical Journal* 2(6204): 1538-1540, December 15, 1979.

From 1951 to 1971, male doctors reduced their cigarette smoking more than did men in other social classes. In 1970-1972, 665 male doctors under age 65 died. Had they shown the same improvements in cause-specific death rates over the 20 years as men in other classes, 699 deaths would have been expected. This saving of 34 deaths in the doctors comprised savings from coronary heart disease (83), stroke (16), and lung cancer (8) balanced by 60 losses from three stress-related causes—namely, accidents and poisonings (30), suicide (26), and cirrhosis of the liver (4)—plus 13 from other causes. As a relative reduction in mortality from heart disease in doctors (as compared with that in other social classes) also occurred during 1931-1951 before they began to give up smoking, some of the saving in heart disease deaths in 1951-1971 was probably not related to changes in smoking habits. The relative worsening in mortality from stress-related diseases may have been due partly to a possible adverse effect of giving up smoking if smoking had acted to reduce stress. From these findings, the benefits of giving up smoking may not be so great as has commonly been assumed. (Auth. Abs. Mod.)

80–1145. Meurman, L. O.; Kiviluoto, R.; Hakama, M. **Combined Effect of Asbestos Exposure and Tobacco Smoking on Finnish Anthophyllite Miners and Millers.** *Annals of the New York Academy of Sciences* 330: 491-495, 1979.

A 1974 study of the morbidity and mortality among workers employed at two asbestos quarries in Finland has been supplemented with additional information on smoking habits. The present study data indicate that the relative risks of lung cancer are as follows: nonsmoker not exposed to asbestos, 1; nonsmoking asbestos worker, 1.6; smoker not exposed to asbestos, 12; and asbestos worker who smokes, 19. The number and cause of deaths by age, exposure, and sex are tabulated for the years 1976-1977. For smokers vs. nonsmokers, there were 22 vs. 1 lung cancer deaths, 35 vs. 7 asbestosis deaths, and 95 vs. 24 deaths from all causes.

See also, **80–1149, 80–1207, 80–1208, 80–1211, 80–1221**

NEOPLASTIC DISEASES

80–1146. Antman, K. H.; Blum, R. H.; Greenberger, J. S.; Flowerdew, G.; Skarin, A. T.; Canellos, G. P. **Multimodality Therapy for Malignant Mesothelioma Based on a Study of Natural History.** *American Journal of Medicine* 68(3): 356-362, March 1980.

Forty patients with malignant mesothelioma were studied to assess the efficacy of current treatment concepts and to examine clinical features at diagnosis which correlate with prognosis. Asbestos exposure and smoking history were reported in 63 and 82 percent, respectively, of the patients. Women, patients who presented with limited disease, and those who had had symptoms for more than 6 months before diagnosis survived significantly longer ($p \leq 0.02$). No prognostic significance could be found for a history of smoking or asbestos exposure, pathologic subtype, or pleural vs. peritoneal presentation. Thirty of the 40 patients underwent open thoracotomy or laparotomy for diagnosis. The median survival of the 10 patients in whom a subtotal resection was attempted was 15 months, contrasted to 8.5 months for the 24 patients who underwent only an open biopsy. Five patients received aggressive radiotherapy, four immediately following subtotal surgical resection and one after biopsy alone. These patients survived a median of 9.21 months, with one patient disease-free at 52 months after diagnosis. Twenty-nine patients received chemotherapy. A partial response occurred in 9 of 22 (40 percent) previously untreated patients given Adriamycin-containing regimens. Patients who received chemotherapy survived a median of 15 months. In contrast, five patients (four who presented with limited disease) who accepted only supportive care survived a median of only 4.2 months after diagnosis. The treatment of mesothelioma is often based on protocols designed for sarcomas. Unlike patients with sarcoma, who generally die of metastatic disease, 78 percent of the patients who could be evaluated died of complications of their local primary disease. It is concluded that mesothelioma is sufficiently different from sarcoma to justify its treatment as a separate disease entity. Otherwise healthy patients with disease limited to a single hemithorax should be offered maximal tumor resection, followed by postoperative radiation therapy to areas of residual disease, followed by chemotherapy with a regimen containing Adriamycin. (Auth. Abs. Mod.)

80–1147. Bader, M.; Sterling, T. D. **Smoking Patterns by Occupation, Industry, Sex and Race. (Letters).** *Archives of Environmental Health* 34(3): 189, May-June 1979.

The greater prevalence of lung cancer among blacks than whites has been ascribed to the larger percentage of blacks in occupations where they are exposed to toxic airborne particles rather than to any difference in smoking habit (*Archives of Environmental Health* 33(6): 313-317, November-December 1978). The author of this letter points out that the higher prevalence may be due to the fact that blacks smoke different brands of cigarettes (e.g., nonfilter, high tar and nicotine) than whites, that they smoke them more completely, and that, in addition, blacks have a 12-14 percent smaller vital capacity of their lungs. It is concluded that occupational exposure may be a factor in the increased incidence of lung cancer among blacks, but that the data presented warrant no such conclusion. In response, the author of the original article reiterates that data show blacks are overrepresented in the "dirty" occupations and that occupation—not smoking—bears the major responsibility for lung cancer in human populations. See also 79-0329.

80–1148. Burch, P.R.J. **Smoking and Lung Cancer. Tests of a Causal Hypothesis.** *Journal of Chronic Diseases* 33(4): 221-238, 1980.

The problem of estimating dose—response relations from epidemiological studies is discussed bearing in mind that association, as described by mortality ratios, might arise through a combination of causal and constitutional factors. A sound theory of carcinogenesis is a prerequisite to any such attempt. Difficulties inherent in one model are avoided by the precipitator hypothesis which is further tested in this paper. The hypothesis assumes that the carcinogenic risk is linear with the average rate of smoking at an interval of T years before death, and that the doubling dose-rate of D cigarettes/year is constant with respect to age over the range tested. Earlier studies indicate that the expected value of T for the precipitation of lung cancer is about 2.5 years. Equations are derived to enable the hypothesis to be tested against secular changes in cigarette consumption and in mortality from lung cancer. The Registrar General's sex- and age-specific death rates for lung cancer in England and Wales, 1950-1954 to 1963-1967; 1968-1971 to 1972-1975; 1972-1973 to 1974-1975; and 1974-1975, are analyzed in relation to the sex- and age-specific statistics for cigarette consumption in the United Kingdom. Values for T of 2.5, 5, and 10 years are tried. One set of data only (males, 1972-1973 to 1974-1975, $T = 2.5$ years) yields a significantly positive value for D and can be said to agree with the hypothesis that the association between smoking and lung cancer is part causal and part constitutional. Attempts are made to correct those recorded changes in mortality that depart consistently from theory. Various trends—some increases and some decreases in death rates—are too large to be accommodated by a linear precipitator theory; others are consistent with the null hypothesis of no causal effect. Errors of death certification are probably responsible for most of the anomalies. In view of the dubious reliability of the potentially critical data analyzed here and elsewhere, it is questionable whether any firm conclusions can as yet be drawn about dose—response relations for cigarette smoking and lung cancer. (Auth. Abs.)

80–1149. Edling, C. **Lungcancermortaliteten i Sverige 1964-1976. [Lung Cancer Mortality in Sweden 1964-1976.]** *Lakartidningen* 75(41): 3659-3661, October 1978, Swedish.

Estimates of lung cancer mortality in Sweden have been made for the 1964-1976 period in order to provide a basis for future health care planning for the Health Care Planning and Rationalization Institute. Mortality rates have continuously increased for both men and women during this time period. In 1965 the national age standardized lung cancer mortality rate per 10^{-5} person years was 42 for men and 11 for women. In 1975 the corresponding figures were 64 and 20, respectively. Local differences in lung cancer mortality rates indicate that smoking is not the only etiological agent. Estimates were made of crude mortality rates in hypothetical populations with various percentages of the given population being nonsmokers, moderate smokers, and heavy smokers. The risks for moderate and heavy smokers were assumed to be 10 and 20 times the risks for nonsmokers. For example, a population with 70 percent nonsmokers and 30 percent moderate smokers would have an estimated mortality rate 0.57 times that of a population with 50 percent nonsmokers, 40 percent moderate smokers, and 10 percent heavy smokers.

80–1150. Friedl, H. P.; Wrba, H. **Lungenkrebsrisiko und "Raucherprophylaxe" in Osterreich. [Risk of Lung Cancer and "Smoker's Prophylaxis" in Austria.]** *Onkologie* 1(3): 124-127, June 1978, German.

Data are presented on the smoking habits of the Austrian population and mortality rate from lung cancer in Austria. In 1972, 45.7 percent of the men and 13.4 percent of the women were smokers. Only 11 percent of all smokers vs. 41 percent of nonsmokers believed that smoking was dangerous to their health. However, 42 percent of the regular smokers have tried at least once to stop smoking. About 90 percent of all smokers and ex-smokers began smoking regularly before they were 25 years old and nearly 80 percent started before they were 20 years old. This indicates the importance of health education for youth as part of an antismoking program. An average reduction of 51 percent in smoke components and 66 percent in the nicotine content of the average Austrian cigarette was noted from 1960 to 1976. This trend toward consumption of "low-risk" cigarettes is related to lower mortality from lung cancer in men over the past 20 years (1953 to 1973). A slight reduction of the mortality rate was observed in the lower age bracket (40-60 years), while the rate increased fourfold in the 60-85 age bracket. These findings are tentatively correlated with a reduction in the carcinogenic content of cigarettes.

80–1151. Higginson, J.; Muir, C. S. **Environmental Carcinogenesis: Misconceptions and Limitations to Cancer Control.** *Journal of the National Cancer Institute* 63(6): 1291-1298, December 1979.

The objectives of this paper are (1) to stress that extensive information is already available on cancer causation in man and to summarize additional inferences concerning possible carcinogenic mechanisms and etiology that may be drawn from epidemiologic data; (2) to emphasize the complexities inherent in the concept of "environment," particularly carcinogenic risk factors and lifestyle, and their significance for cancer prevention; and (3) to propose that more research is needed in human cancer on the role of cocarcinogenesis, including promotion. Various personal habits (e.g., tobacco, alcohol, occupation, lifestyle) as etiologic factors are considered for the United Kingdom and Greater Bombay, India. In the United Kingdom, 80-85 percent of lung cancers are thought to be caused by cigarette smoking. Smoking is also implicated in cancer of the mouth, larynx, pharynx, esophagus, cardiac end of stomach, bladder, and probably the pancreas. The proportion of cancers due to cigarette smoking is somewhat lower in Bombay, where tobacco use appears to have its impact through the betel quid on the oral cavity, pharynx, and upper esophagus. In the United Kingdom, excessive alcohol consumption causes cancer of the mouth, pharynx, esophagus, and liver. Inasmuch as most persons with cancers of the mouth and esophagus both smoke and drink heavily, they have been assigned to the tobacco-alcohol category. Findings in nonsmoking, "teetotal" Mormons and Seventh-Day Adventists in the United States support these estimates. Alcohol consumption is relatively rare in Bombay.

80–1152. Howe, G. R.; Burch, J. D.; Miller, A. B.; Cook, G. M.; Esteve, J.; Morrison, B.; Gordon, P.; Chambers, L. W.; Fodor, G.; Winsor, G. M. **Tobacco Use, Occupation, Coffee, Various Nutrients, and Bladder Cancer.** *Journal of the National Cancer Institute* 64(4): 701-713, April 1980.

In a Canadian population-based case—control study of 480 male and 152 female case—control pairs, the relative risk for development of bladder cancer for ever used vs. never used cigarettes was 3.9 for males and 2.4 for females, with a dose—response relationship in both sexes. A reduced risk was associated with the use of filter cigarettes compared to nonfilter cigarettes. For three measures of cigarette smoking: average frequency (cigarettes/day), duration (years), and estimated lifetime consumption (packs), both sexes had a highly significant dose—response relationship; the heaviest male smokers showed approximately a fivefold increase in risk, and the female smokers showed a threefold increase relative to that seen in lifetime nonsmokers. All chi-squares for trend were statistically highly significant ($p<0.01$). After control for cigarette usage, a significant risk (2.0) was noted for male pipe smokers. Heavy inhaling gave an increased risk of 8.8 compared to noninhalers who smoked the same amount of pipe tobacco. Data were insufficient for the ascertainment of any dose—response relationship for cigars and cigarillos. A relative risk estimate for males who ever smoked cigars or cigarillos vs. those who never smoked was 1.2. The relative risk for males reporting using chewing tobacco at any time vs. those never using chewing tobacco was 0.9. For male ex-smokers the risk after 15 years of no smoking was less than one-half that of current male smokers. Bladder cancer risk was found for workers in the chemical, rubber, photographic, petroleum, medical, and food processing industries among males, and for workers occupationally exposed to dust or fumes among both sexes. Bladder cancer risk was elevated for males consuming all types of coffee, regular coffee and instant coffee, and for females consuming instant coffee, but no dose—response relationship was found. Risk was found for males consuming water from nonpublic supplies, but not for females. Reevaluation of the data on artificial sweeteners confirmed a significant bladder cancer risk in males and a dose—response relationship. The cumulated population attributable risk for bladder cancer was 90 percent for males from cigarette smoking, industrial exposure, and exposure to nonpublic water supplies and 29 percent for females from cigarette smoking, industrial exposure, and instant coffee consumption. (Auth. Abs. Mod.)

80–1153. Khan, M. Y.; Maltzman, B.; Jonnard, R. **Carcinoma of Esophagus. Autopsy Reports.** *New York State Journal of Medicine* 80(4): 575-579, March 1980.

A retrospective study of etiologic factors in 68 well-documented autopsied cases of esophagocarcinoma is presented. Dysphagia, weight loss, and anemia were the most common presenting symptoms; 70 and 75 percent of patients died within 6 months following diagnosis, and 9 months following the onset of symptoms, respectively. Among the associated findings, dental deterioration and poor oral hygiene were present in 90 percent of cases. A strong statistical association between esophageal neoplasms and heavy smokers/drinkers was observed, and the association indicates a synergistic effect of these two factors. Race and occupation showed only a weak correlation statistically and could very well be attributed to socioeconomic factors alone, and not to any racial factors per se. On the other hand, correlation between race and age is weak, but is probably significant in both racial groups. (Auth. Abs. Mod.)

80–1154. Lancet. **What Proportion of Cancers are Related to Occupation?** *Lancet* 2(8102): 1238-1240, December 9, 1978.

An examination of reported attempts to quantify the relative risks of various cancers in workers exposed to particular substances indicates that occupational cancers comprise anywhere from 1 to 5 percent of the total cancer toll. A recent National Institutes of Health study maintains that estimates such as these often do not take into account the multiple causation of many cancers, and also do not adequately follow up retired workers. Although the NIH report received wide publicity, its framework is remarkably insubstantial in view of other research. There is no review of the full range of epidemiological studies that have been published on this subject; no data are presented concerning the degree of exposure in the studied workers, and risks are estimated from only one study. The British Registrar General's Decennial Supplement on Occupational Mortality, another source of occupational cancer data, tabulates cancer mortality by social class. A calculation of smoking data, by social class and risk of lung cancer for various levels of smoking, suggests that manual laborers have a lung cancer risk that is 22 percent higher than that of administrative and clerical workers. The NIH report suggests that a considerable proportion of smokers' premature deaths are as much due to other agents as to smoking.

80–1155. Lemoine, J. M. **Modalites du Depistage Presymptomatique des Cancers Bronchogeniques. [Modality of Early Diagnosis of Bronchogenic Cancers.]** *Broncho-Pneumologie* 29(3): 265-272, May-June 1979, French.

A prospective diagnostic study of 2,000 patients was launched in 1972 in order to support a proposal for a target population classification for the study of bronchogenic carcinomas and correlated diseases. Total tobacco consumption is considered to be one of the main factors in the selection of individuals for a systematic detection study, age and sex being the other factors. Of the 2,000 patients examined by bronchoscopy, 79 percent were men, 94 percent of whom had bronchogenic cancer and had smoked at least 150,000 cigarettes. It was found that 83 and 92 percent of the male smokers with bronchogenic carcinoma were 45-74 and 40-79 years old, respectively. Among the women with bronchogenic and metastatic carcinomas, 68 and 96 percent were inveterate smokers, respectively. Of the chronic bronchitics, 76 and 27 percent of the men and women, respectively, had smoked more than 150,000 cigarettes. Of the patients with coronary occlusions, approximately one-half are smokers.

80–1156. Lemoine, J. M. **Reunion Commune des Groupes d'Etudes sur les Cancers Bronchogeniques et sur les Bronchites Chroniques de l'A.I.B.P. a Toulouse le 3-5-1978. [Joint Meeting of the Study Groups of the A.I.B.P. on Bronchogenic Cancers and Chronic Bronchitis in Toulouse 3-5-1978.]** *Broncho-Pneumologie* 29(3): 264-265, May-June 1979, French.

A meeting on chronic bronchitis and bronchogenic cancer took place in Toulouse, prompted by the data that these diseases have been diagnosed in men over 40 who are heavy smokers. Discussed are early detection of bronchogenic cancers and a more precise classification of the target population extended to include cases of chronic bronchitis and coronary artery obstruction.

80–1157. Longnecker, D. S.; Shinozuka, H.; Dekker, A. **Focal Acinar Cell Dysplasia in Human Pancreas.** *Cancer* 45(3): 534-540, February 1, 1980.

A series of 108 prospectively collected human pancreata were evaluated histologically for the presence of acinar cell and ductal lesions. Foci of dysplastic acinar cells were present in 44 percent of the series. Some of the focal acinar cell abnormalities were similar to atypical acinar cell nodules which have been described in carcinogen-treated experimental animals. The incidence of nodules was higher among patients with a history of cigarette smoking (46.8 percent), particularly heavy (67->100 pack-years) smokers (83.3 percent), than among nonsmokers (40 percent), and among patients with a history of alcohol abuse than among abstainers. The presence of dysplastic acinar cell nodules in a pancreas seemed unrelated to the presence of cancer in other sites, or diabetes. Ductal epithelial abnormalities were more frequent than focal acinar cell dysplasia. (Auth. Abs. Mod.)

80–1158. Morrison, A. S.; Buring, J. E. **Artificial Sweeteners and Cancer of the Lower Urinary Tract.** *New England Journal of Medicine* 302(10): 537-541, March 6, 1980.

The relationship between cancer of the lower urinary tract and the use of artificial sweeteners was evaluated in a case-control study of 592 patients with lower urinary tract cancer (94 percent of whom had a bladder tumor) and 536 controls chosen from the general population of the study area. A history of use of artificial sweeteners and exposure to other known or suspected risk factors was determined by interview. In those who had used dietetic beverages and in those who had used sugar substitutes, the relative risk of lower urinary tract cancer was estimated as 0.9 (0.7 to 1.2, 95 percent confidence interval), as compared with 1 in nonusers of artificial sweeteners. Among men, the relative risk was 0.8 (0.6 to 1.1) in those who had used dietetic beverages and 0.8 (0.5 to 1.1) in those who had used sugar substitutes. Among women, the corresponding relative risks were 1.6 (0.9 to 2.7) and 1.5 (0.9 to 2.6). Increasing frequency or duration of use of artificial sweeteners was not consistently associated with increasing relative risk. Both cigarette smoking and occupational history were associated with the rate of bladder cancer in this study. In both men and women, smokers had a lower relative risk than nonsmokers (0.7 vs. 0.9 in men and 1.2 vs. 2.6 in women using dietetic beverages and 0.6 vs. 1.1 in men and 1.5 vs. 2.1 in women using sugar substitutes). This study suggests that, as a group, users of artificial sweeteners have little or no excess risk of cancer of the lower urinary tract. (Auth. Abs. Mod.)

80–1159. Mueller, K.-M.; Mueller, G. **Praneoplasien des Bronchialsystems bei Rauchern und Nichtrauchern. [Preneoplasms of the Bronchial System in Smokers and Nonsmokers.]** *Muenchener Medizinische Wochenschrift* 122(Supplement 1): S14-S17, February 20, 1980, German.

Parallel to the rapidly rising frequency of bronchial carcinoma with almost 27,000 deaths in 1978, preneoplasms of the bronchial system are now almost daily observations for the pathologist. The question of cancerous prestages of the bronchial mucosa is closely coupled with the histogenesis of malignant neoplasms. Besides epithelial anomalies such as basal cell hyperplasia and squamous epithelial metaplasias, precancerous epithelial dysplasias of Grades I to III and carcinoma in situ must be differentiated. From extensive histological investigations, the relationship between smoking, smoking habits, and carcinogenic constituents in the condensate of cigarettes on one hand, and both frequency and severity of preneoplastic and neoplastic changes in the bronchial system on the other, is well known. In a study of epithelial anomalies and preneoplasms in the lungs of 200 male patients, squamous epithelial metaplasia was found in 60-80 percent of heavy smokers, but in nonsmokers only if they also had chronic bronchitis. (Auth. Abs. Mod.)

80–1160. National Cancer Institute. **Aspects of Cancer Research 1971-1978: Editorials from the Journal of the National Cancer Institute.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute Monograph 52, NIH Publication No. 79-1863, September 1979, 538 pp.

This monograph contains editorials from the Journal of the National Cancer Institute which has published this series since 1971. For this volume, the authors of each editorial were invited to submit a short addendum which appears after the editorial. Among the various aspects of cancer considered are (1) general epidemiology, (2) chemical and physical agents, (3) drugs and hormones, (4) microbiological agents, (5) lung cancer (and smoking), (6) large bowel cancer, and (7) treatment.

80–1161. Public Health Reports. **Risk of Bladder Cancer Higher Among Heavy Users of Cigarettes and Artificial Sweeteners.** *Public Health Reports* 95(2): 196, March-April 1980.

The National Cancer Institute and the Food and Drug Administration have released the preliminary findings of a study in which the relationship between the use of artificial sweeteners, heavy cigarette smoking, and the incidence of bladder cancer in humans was examined. Although the preliminary results showed no increased risk of bladder cancer among users of artificial sweeteners in the overall study population, there was evidence that the risk was increased by 60 percent in heavy users (≥ 6 servings of a sugar substitute or ≥ 2 8-ounce diet beverages a day) and in women who consumed sugar substitutes or diet beverages two or more times a day. Persons who were both heavy smokers and heavy users of artificial sweeteners showed a higher risk of bladder cancer than heavy smokers who did not use sweeteners.

80–1162. Roos, B. A.; Lindall, A. W.; Baylin, S. B.; O'Neil, J. A.; Frelinger, A. L.; Birnbaum, R. S.; Lambert, P. W. **Plasma Immunoreactive Calcitonin in Lung Cancer.** *Journal of Clinical Endocrinology and Metabolism* 50(4): 659-666, April 1980.

Plasma calcitonin (CT) was increased in 135 untreated eucalcemic men with lung cancer and a control/smoker population. CT levels were determined by radioimmunossay (RIA) and validated by immunoextraction. Plasma immunoreactive CT moieties were purified by immunoadsorbent chromatography, treated with mercaptoethanol and urea, and characterized by gel filtration. Artifacts in human CT RIAs of cancer patient plasmas were detected by parallel plasma incubations in a salmon CT RIA system, which does not detect human CT, and by immunoprecipitation of tracer at the end of RIA incubations. Heating fresh plasmas to 65°C for 1.5 hours reduced RIA artifacts without loss of CT moieties. RIA of plasma samples showed elevated plasma immunoreactive calcitonin (iCT) in the lung cancer patients. The mean iCT concentration and percentage of patients with levels >100 pg/ml were epidermoid, 64 pg/ml (11 percent); small cell, 143 pg/ml (19 percent); adenocarcinoma, 334 pg/ml (42 percent); and anaplastic carcinoma, 158 pg/ml (21 percent). Mean plasma iCT levels in smokers (range, <25 to 126 pg/ml) and nonsmokers (range, <25 to 97 pg/ml) were 40 and 35 pg/ml, respectively. Unequivocal hypercalcitoninemia (heat stable, causing no inhibition of antibody-tracer binding in the salmon CT RIAs, and immunoextractable with human CT antibodies) was not found in any of the apparently hypercalcitoninemic plasmas from persons with epidermoid or anaplastic lung cancer. By contrast, unequivocal hypercalcitoninemia was found in 27 percent (15 of 55 samples) of the plasma samples from patients with small cell carcinoma or adenocarcinoma. Most of the immunoreactive CT recovered from small cell and adenocarcinoma lung cancer plasmas with unequivocally elevated CT levels is much larger than the CT monomer. (Auth. Abs. Mod.)

80–1163. Veber, O. A.; Dolgintsev, V. I. **Znachenie Sanitarno-Prosvetitel'noi Raboty v Profilaktike Raka Zheludka. [Importance of Health Education in Prophylaxis of Stomach Cancer.]** *Sovetskoe Zdravookhranenie* (5): 26-29, 1979, Russian.

Results of an epidemiological study of stomach cancer in the Tumen region indicate that smoking, alcohol abuse, and inadequate nutrition may be risk factors for precancerous and cancerous stomach conditions. The data show that smoking incidence is the same in male stomach cancer patients as in healthy male subjects. However, the cancer patients had not only smoked on an empty stomach 1.6 times more often than the healthy subjects, but had started smoking at 15 years 1.8 times more often than the healthy subjects. Among the patients, 26 percent were heavy smokers (>20 cigarettes/day) who had started smoking at 15 years, while 2.8 times fewer healthy subjects were heavy smokers. Only 3.3 percent of the female patients were smokers. Irregular nutrition combined with alcohol abuse and smoking on an empty stomach was 15.9 times more frequent in cancer patients than in healthy subjects. Based on these data, a differential approach to health education is recommended for a variety of audiences (e.g., children, adolescents, women, and industrial workers). Positive results are expected from prohibition of smoking in public places, limitation of tobacco product advertisement, establishment of "nonsmoking collectives," and health education (particularly for young people).

80–1164. Wynder, E. L.; Stellman, S. D. **Artificial Sweetener Use and Bladder Cancer: A Case-Control Study.** *Science* 207(4436): 1214-1216, March 14, 1980.

In a case—control study of 302 male and 65 female bladder cancer patients and an equal number of other patients matched to them in age, sex, hospital, and hospital room status, no association was found between use of artificial sweeteners (AS) or diet beverages (DB) and bladder cancer. No dose-response was observed with respect to quantity or duration of use or to the two combined. No evidence was found to suggest that artificial sweeteners or diet beverages promote the tumorigenic effect of tobacco smoking; in AS consumers, the risk ratios for bladder cancer of long-term (≥10 years) male and female cigarette smokers were 0.56 and 1.04, respectively, and similar results were obtained for DB. No statistically significant changes occurred when smokers were further classified by the number of cigarettes smoked per day. Artificial sweetener and diet beverage use strongly reflected socioeconomic status among controls with various diagnoses. (Auth. Abs. Mod.)

See also, **80–1125**

NON—NEOPLASTIC RESPIRATORY DISEASES

80–1165. American Hospital Association. **Staff Manual for Teaching Patients About Chronic Obstructive Pulmonary Diseases.** Chicago, American Hospital Association, 1979, 414 pp.

This manual provides information for hospital staff members in helping patients manage their chronic obstructive pulmonary diseases (COPD). The topics covered include the disease process, COPD diagnosis and therapies, and patient education. The relationship between smoking and COPD is briefly discussed. Smoking is the most common factor contributing to COPD. Methods for encouraging patients to stop smoking are described.

80–1166. Bergstermann, H.; Emslander, H.-P.; Roeder, W. **Bestimmung des CO-Transferfaktors der menschlichen Lunge mit einem stabilen CO_{18}-Isotop; klinische Erfahrung bei restriktiven und obstruktiven Ventilationsstorungen. [Determining the CO Exchange Ratio of the Human Lung Using a Stable CO_{18} Isotope; Clinical Experience with Restrictive and Obstructive Ventilation Disorders.]** *Verhandlungen der Deutschen Gesellschaft fuer Innere Medizin* 83: 1465-1467, April 1977, German.

The single-breath pulmonary diffusing capacity for carbon monoxide (DL_{CO}-SB)/m² body surface was determined in 157 healthy adults, 20-70 years old, by the standard end-tidal sampling and mass spectroscopic methods using O_{18}. The population sample consisted of 85 male and 72 female subjects. Significantly higher respiratory exchange ratios for CO were found for the 42 male nonsmokers, while the ratios for the 43 male smokers were significantly lower. The exchange ratio for the 44 female nonsmokers was also significantly higher than for the 28 female smokers. In 32 patients with obstructive ventilation disorders and an average airway resistance of 6.6 cm H_2O/l/sec, DL_{CO} determined by the standard method was 44.6 percent of the normal value, whereas the exchange ratio determined with CO_{18} was 75.8 percent of the normal value. In 28 patients with restrictive ventilation disorders and vital capacity 49.5 percent lower than normal for the same age, no significant difference was detected between the DL_{CO} values determined by the two methods. In contrast, in six patients with restrictive ventilation disorders, the standard deviation decrease from 16.2 to 6.5 percent was statistically significant as determined in four consecutive comparative measurements of DL_{CO}. In the case of obstructive ventilation disorders, more accurate results were obtained with mass spectrometry.

80–1167. Chan-Yeung, M.; Schulzer, M.; MacLean, L.; Dorken, E.; Grzybowski, S. **Epidemiologic Health Survey of Grain Elevator Workers in British Columbia.** *American Review of Respiratory Disease* 121(2): 329-338, February 1980.

Health surveys of 610 workers (grain elevator, sawmill, and civic) were performed using medical and occupational questionnaires, spirometry, skin tests, α_1-antitrypsin determinations, precipitin studies, and dust concentration measurements. The prevalence of eye, nasal, and chest symptoms was higher in grain elevator workers than among the control groups of sawmill and civic workers. In all three groups of workers, the frequency of chest symptoms was greater among smokers than nonsmokers. Current smokers in all three groups had lower pulmonary function values than did nonsmokers. No potentiating effect between exposure to grain dust and cigarette smoking was found; the effect of these variables on pulmonary function was only additive. Measurements of pulmonary function were highly dependent on age, regardless of work group and smoking habits. No significant difference was found in mean pulmonary function values between atopic and nonatopic subjects in the three work groups, regardless of smoking habits.

80–1168. Charan, N. B.; Hildebrandt, J.; Butler, J. **Alveolar Gas Compression in Smokers and Asthmatics.** *American Review of Respiratory Disease* 121(2): 291-295, February 1980.

To correlate the degree of alveolar gas compression with the severity of airway obstruction, plots were made of separately exhaled and plethysmographic maximal expiratory flow volume (MEFV) curves in each of four healthy nonsmokers, four asymptomatic smokers, and four asymptomatic asthmatics. Spirometry, lung volumes, single breath nitrogen plateau (Phase III), and closing volumes (Phase IV) were normal in the smokers and nonsmokers, and they were either normal or showed evidence of mild airway obstruction in the asthmatics. Maximal expiratory flow volume curves were plotted by using exhaled flow against exhaled volume, and by using plethysmographic flow against plethysmographic volume. These curves were utilized to construct isovolume-pressure-flow curves of esophageal pressure (Pes) against exhaled flow and against plethysmographic flow for each subject. Differences in the flow (plethysmographic minus exhaled) were calculated from these curves at 75 percent vital capacity (VC), 50 percent VC, and 25 percent VC at the same Pes in all three groups. At 75 percent VC, the mean flow difference in the asthmatic group was significantly higher than in normals ($p<0.05$); at 50 percent VC, it was significantly higher both in the asthmatics and the smokers ($p<0.05$); at 25 percent VC, there was no significant difference among the three groups. The mean plethysmographic forced vital capacity (FVC) exceeded exhaled FVC in all three groups, but the difference was significantly higher in smokers and asthmatics than in nonsmokers ($p<0.05$). These findings suggest that significant alveolar gas compression may occur at 75 percent VC in mild asthmatics, whereas in smokers it occurs at the middle portion of the VC. (Auth. Abs.)

80–1169. Cohen, B. H.; Menkes, H. A.; Bias, W. B.; Chase, G. A.; Diamond, E. L.; Graves, C. G.; Levy, D. A.; Meyer, M. B.; Permutt, S.; Tockman, M. S. **Multiple Factors in Airways Obstruction.** *Chest* 77(2): 257-259, February 1980.

A number of risk factors for chronic obstructive pulmonary disease (COPD) (e.g., smoking habits, family history, socioeconomic status, age, sex, race, and alcohol intake) were studied in 2,539 subjects, none of whom were patients. The greatest difference of means (6.6) was found when comparisons of subjects more than 45 years old were made with subjects 20-44 years old. Smokers of $\geq$ 1 pack of cigarettes/day had a mean forced expiratory volume in 1 sec percent 4.6 less than never-smokers. Other risk factors important to the development of COPD included age, socioeconomic status, family relationship, sex, and secretor status.

80–1170. de Hamel, F. A.; O'Donnell, T. V. **Stopping Smoking. Symptoms and Objective Assessment in Ex-Smokers.** *New Zealand Medical Journal* 90(649): 456-459, December 12, 1979.

The results of a respiratory questionnaire and spirometric tests of 133 ex-smokers out of 649 male public servants aged 15-64 years have been analyzed. When the population was adjusted for age, height, and the presence or absence of chronic respiratory symptoms, the forced expiratory volume in 1 sec of the ex-smokers had not returned to normal but fell between that of the light ($<$10/day) and moderate cigarette smokers. Factors contributing to this have been investigated. Factors affecting the FEV$_1$ of the ex-smokers were found to include duration of smoking, quantum smoked, and years since smoking ceased. An interactive effect has been demonstrated between quantum smoked and duration of smoking such that ex-smokers who used to smoke 10 or more cigarettes a day for more than 10 years had the lowest lung function, but duration of smoking was not important if less than 10 cigarettes a day had been smoked. Only just over a third of the ex-smokers gave health-related reasons for giving up smoking, a proportion which is considerably lower than has been found in other studies. Possible reasons for this difference are discussed. As in other studies less than 10 percent gave up smoking because of its cost. Respiratory screening to include questions on cough, phlegm, and wheeze together with measurement of the FEV$_1$ is an important way of demonstrating to individual smokers evidence of any damage caused by their smoking. (Auth. Abs.)

80–1171. Dosman, J. A.; Cotton, D. J.; Graham, B. L.; Hall, D.; Li, R.; Froh, F.; Barnett, G. D. **Sensitivity of Variables Derived from the Single Breath Nitrogen Test in Smokers.** *Chest* 77(2): 286-289, February 1980.

In an evaluation of the sensitivity of two lung function tests (1) slope of the alveolar plateau ($\Delta N_2/L$) and (2) closing volume (CV/VC) which are derived from the single breath nitrogen curve, forced expiratory volume in 1 sec (FEV$_1$)/forced vital capacity (FVC) times 100 was used as a reference test. For this analysis, information on occupational history, respiratory symptoms, previous chest illness, and smoking habits was obtained from a study population consisting of adults aged 25 to 59 from a nonpolluted rural community. There were 418 nonsmokers consisting of 138 men and 280 women, 456 current smokers consisting of almost equal numbers of men (241) and women (215), and 275 ex-smokers. Nonsmokers included those who had never smoked any kind of tobacco regularly or had smoked a total of less than one-half pack year (i.e., <1 pack/day for 6 months). Of the smokers, 7.0 percent (female) and 7.9 percent (male) had abnormal FEV_1/FVC times 100. For men, the sensitivity of $\Delta N_2/L$ was 63.2 percent and for women, 66.7 percent. The sensitivity of CV/VC was low for both men and women. However, the combination of $\Delta N_2/L$ and/or CV/VC improved sensitivity to 68.4 percent for men and 80.0 percent for women.

80–1172. Evans, H. E.; Bognacki, N. **α_1-Antitrypsin Deficiency and Susceptibility to Lung Disease.** *Environmental Health Perspectives* 29: 57-61, April 1979.

α_1-Antitrypsin (AAT), the major component of α_1-globulin, is capable of inhibiting proteolytic enzymes in polymorphonuclear leukocytes (PMN) and pulmonary alveolar macrophages (PAM). It is a glycoprotein synthesized in the liver with a molecular weight of 50,000 daltons, a half-life of 3-6 days, and a sedimentation constant of 3.3 S. Enzymes inhibited include elastase, collagenase, thrombin, kallikrein, and chymotrypsin. Serum AAT concentration is determined by multiple codominant alleles. The normal phenotype is M, the most severe deficiency state, Z; intermediate levels are associated with the MZ phenotype. Ethnicity is a major factor in determining phenotypic prevalence. The homozygous Z state occurs in 1/4000-1/8000 Caucasians. Numerous disease processes, mainly involving lungs and liver, have been associated with AAT deficiency. Establishing an unequivocal etiologic relationship, however, between AAT deficiency and lung disease has been difficult. Studies in this area have varied widely in the number of individuals included, whether phenotypic analysis, or only quantitative determinations, were carried out, in the range of clinical and pulmonary function studies performed, and whether the population was community or hospital based. Other confounding variables include the degree of industrial exposure, cigarette smoking, a history of childhood lung disease, and levels of proteolytic enzymes in PAMs and PMNs. Cigarette smokers have more PAM cells and each cell has more proteolytic capacity than is the case for nonsmokers. It has been estimated that about 80 percent of those with homozygous Z phenotype will develop obstructive pulmonary disease. The prevalence of lung disease in other non-M phenotypes, especially among heterozygotes is uncertain. Those investigations which support, and others which refute, an association are reviewed. Further investigations, especially those emphasizing levels of proteolytic enzymes, are needed to clarify the interrelationship of these variant phenotypes, environmental risks, and the predisposition to chronic obstructive lung disease. (Auth. Abs. Mod.)

80–1173. Fairshter, R. D.; Wilson, A. F. **Relative Sensitivities and Specificities of Tests for Small Airways Obstruction.** *Respiration* 37(6): 301-308, 1979.

This study compared several physiologic maneuvers for detecting small airways obstruction (SAO). Volume of isoflow best separated young smokers (average age—27.6; average cigarette consumption—9.3 pack years) from nonsmokers; this was true for comparisons of both population means and incidence of individual abnormality. However, assessment of individual abnormality by any test was greatly influenced by the criteria used to define abnormality. The best combination of test sensitivity and specificity was achieved by using the 95 percent confidence limits derived from a control population from the same laboratory. Even so, test results did not correlate significantly with pack years of cigarette smoking. Then, although tests for SAO are very sensitive, their prognostic value in young smokers is uncertain since abnormality presumably should correlate with extent of smoking. (Auth. Abs.)

80–1174. Gee, J.B.L.; Hinman, L. M.; Stevens, C. A.; Matthay, R. A. **Human Alveolar Macrophage and Obstructive Lung Disease.** *Chest* 77(2): 271-272, February 1980.

The potential role of alveolar macrophages (AM) in tissue injury in the lung depends on two factors: the degree of their activation, and their secretory activity, particularly with respect to elastase. Alveolar macrophages were obtained from smokers and nonsmokers by bronchoalveolar lavage. Cell counts and cell protein were measured prior to tissue culture of the AM in serum-free medium. Alveolar macrophage activation in smokers was demonstrated by increased cellular adherence, a fivefold increase in the secretion of lysozyme, and a twofold increase in intracellular angiotensin I convertase activities. Elastase activities in AM were measured with two substrates: succinyl-l-alanyl-alanine-p-nitrogen dioxide anilide, and H^3-labeled elastin. It was found that AM show elastase activity but also adsorb and/or ingest polymorphonuclear monocyte elastase.

80–1175. Gibson, J.; Gallagher, H.; Johansen, A.; Webster, I. **Lung Function in an Australian Population: I. Spirometric Standards for Non-Smoking Adults.** *Medical Journal of Australia* 1(7): 292-295, April 7, 1979.

Relationships between age, height, weight, and spirometry with forced expiratory volume at 0.5 sec, forced expiratory volume at 1 sec (FEV_1), forced vital capacity (FVC), and FEV_1/FVC are described for a sample of 6,275 female and 6,511 male nonsmoking adults living in New South Wales. Prediction equations for ventilatory function tests are derived from nonsmokers and compared with other standards that are available in a population sample of 31,172 subjects. It is concluded that the standards described in this paper are more appropriate for use in Australian populations. Nomograms for predicting FEV_1 and FVC in females and males are provided. (Auth. Abs.)

80–1176. Haslam, P. L.; Turton, C.W.G.; Heard, B.; Lukoszek, A.; Collins, J. V.; Salsbury, A. J.; Turner-Warwick, M. **Bronchoalveolar Lavage in Pulmonary Fibrosis: Comparison of Cells Obtained With Lung Biopsy and Clinical Features.** *Thorax* 35(1): 9-18, January 1980.

Bronchoalveolar lavage, open lung biopsy, and cell extraction from the biopsy material have been studied in 21 symptomatic patients (seven smokers, eight ex-smokers, and six nonsmokers) with progressive pulmonary fibrosis. The total and differential cell counts between the three different samples have been compared as well as the influence on them of smoking and their correlation with steroid responsiveness and later progress. The total cell counts in lung lavage were greater in smokers compared to nonsmokers ($p = 0.03$), the alleged ex-smokers holding an intermediate position. The same order of total cell counts was observed in those with cryptogenic fibrosing alveolitis (CFA) (mean 0.51×10^6/ml) and those with asbestos exposure (mean 0.53×10^6/ml). As for the differential cell counts, there was no significant difference when smokers with CFA were compared with nonsmokers. There was no correlation between semiquantitative scores of cell types observed within alveolar spaces and in alveolar walls and the differential or total cell counts obtained from extraction or lung lavage samples. There was, however, some correlation between differential counts obtained from lung lavage and extractions (neutrophils $p < 0.02$, eosinophils $p < 0.07$, lymphocytes $p < 0.08$), suggesting that lung lavage reflects the cellularity of the peripheral parts of the lung in patients without overt bronchial disease. Steroid responsiveness related to the percentage of lymphocytes found in extraction samples ($p < 0.01$) and was associated with a complementary fall in the percentage of macrophages ($p < 0.02$). There was no relationship between steroid response and the numbers of neutrophils or eosinophils in extracted samples. (Auth. Abs. Mod.)

80–1177. Higenbottam, T.; Shipley, M. J.; Clark, T.J.H.; Rose, G. **Lung Function and Symptoms of Cigarette Smokers Related to Tar Yield and Number of Cigarettes Smoked.** *Lancet* 1(8165): 409-412, February 23, 1980.

In a survey of over 18,000 male civil servants (7,600 cigarette smokers, 638 pipe and cigar smokers, 3,452 nonsmokers, and 6,693 ex-smokers), tar yield and number of cigarettes smoked daily were related to lung function and respiratory symptoms. Both phlegm production and airflow obstruction among smokers increased with daily cigarette consumption. Tar yield influenced phlegm production but not the degree of airflow obstruction. However, those smoking low-tar cigarettes (>20/day) had the same phlegm production as that of high-tar cigarette smokers. Ex-smokers had better lung function than current smokers with comparable total cigarette consumption; the difference between ex-smokers and current smokers was not related to the length of time that ex-smokers had abstained from smoking. (Auth. Abs. Mod.)

80–1178. Holma, B.; Kjaer, G. **Alcohol, Housing, and Smoking in Relation to Respiratory Symptoms.** *Environmental Research* 21(1): 126-142, February 1980.

An evaluation of the relative effects of housing standards, alcoholic beverage consumption, and tobacco smoking on the appearance of respiratory symptoms in the adult population in older residential districts in Central Copenhagen indicates that regular daily intake of alcoholic beverages, especially beer, and residence in damp and cold dwellings have more bearing on these symptoms than tobacco smoking. Within these limitations coughing in the morning was significantly correlated to the number of cigarettes smoked per day ($p < 0.05$), and phlegm production and wheezing in the chest were significantly related to the smoking of cigars ($p < 0.01$). The results are based on 881 interviews comprising 109 questions of medical, hygienic, social, and housing conditions. The evaluation of the results is primarily based on the use of a statistical structure search program identifying and ranking predictors for the health of the different population groups. (Auth. Abs. Mod.)

80–1179. Holtz, B.; Bake, B.; Sixt, R. **Prediction of Postoperative Hypoxemia in Smokers and Non-Smokers.** *Acta Anaesthesiologica Scandinavica* 23(5): 411-418, October 1979.

Age, weight, spirometric variables, peak expiratory flow, and airway closure assessments were correlated to postoperative gas exchange in 40 cholecystectomized subjects grouped according to smoking history. Partial pressure of oxygen in arterial blood (Pa_{O_2}) was significantly lower in the smoking group both pre- and postoperatively, but the decrease in Pa_{O_2} following operation was of the same magnitude irrespective of smoking history. In nonsmokers, the preoperative relationship between expiratory reserve volume and closing volume showed the highest correlation to postoperative alveolar-arterial oxygen difference $P(A\text{-}a)_{O_2}$ ($p < 0.001$) and to the increase of $P(A\text{-}a)_{O_2}$ following operation ($p < 0.001$), whereas age was found to be the best preoperative predictor of postoperative gas exchange in smokers. (Auth. Abs.)

80–1180. Jolly, D. T. **Acute Tonsillopharyngitis in Native and White Families.** *Canadian Family Physician* 26: 59, 61-62, 64, 66, January 1980.

Native and white families in whom a diagnosis of tonsillopharyngitis had been documented during 1975 or 1976 were surveyed during 1978 whenever a family member presented to the Southwest Middlesex Health Center in Ontario. Families who had no record of tonsillopharyngitis during 1975 and 1976 were selected as controls. The relationship between smoking and allergic disorders within the family and the diagnosis of tonsillopharyngitis was determined. The presence of smoking family members and family members with allergic disorders was noted significantly more often in study than in control families ($p = 0.01$ and $p < 0.02$, respectively). Similarly, families which had an average of two or more persons per bedroom were found significantly more frequently in study families with tonsillopharyngitis. Native patients were diagnosed as having tonsillopharyngitis significantly more frequently than white patients. In addition, native patients had a significantly greater number of positive bacterial throat cultures than did white patients. (Auth. Abs. Mod.)

80–1181. Kauffmann, F.; Querleux, E.; Drouet, D.; Lellouch, J.; Brille, D. **Evolution du VEMS en 12 ans et Tabagisme Chez 556 Travailleurs de la Region Parisienne. [Twelve Year FEV_1 Changes and Smoking Habits Among 556 Workers in the Paris Area.]** *Bulletin Europeen de Physiopathologie Respiratoire* 15(5): 723-737, 1979.

The aim of the study was to analyze the effects of smoking, in particular to show its causal role in the development of airflow obstruction, and to look at changes in smoking habits. The study was conducted among 556 men, aged 30-54 in 1960, surveyed twice, in 1960 and in 1972. The hypothesis of tobacco as a causal factor of airflow obstruction is strengthened, following this study, by three results: (1) forced expiratory volume in 1 sec (FEV_1) slope was related to tobacco consumption, even after adjustment for FEV_1 level (42 ml/year for nonsmokers, 51 ml/year for heavy smokers); (2) FEV_1 loss with age increased with the amount of tobacco consumption: one pack a day smoked for 25 years was equivalent to an aging of 5 years; (3) FEV_1 loss decelerated if the subject gave up smoking, thus preventing any further risk. What appeared to be a spontaneous regulation in smoking habits was observed. The men who stopped smoking were those with low respiratory status. In this population, men who were ex-smokers in 1960 and maintained this status until 1972 had a FEV_1 slope similar to that of the nonsmokers. (Auth. Abs.)

80–1182. Korovina, O. V.; Soboleva, L. G.; Lifshits, N. A.; Afanasova, L. N.; Grishina, L. G. **Nespetsificheskiye Zabolevaniya Organov Dykhaniya na Predpriyatii Tyazhelogo Mashinostroyeniya. [Nonspecific Diseases of the Respiratory Organs at a Heavy Machine Building Factory.]** *Sovetskaya Meditsina* (4): 100-105, April 1979, Russian.

A pneumotachometric examination of workers in a heavy machine building plant revealed bronchial obstruction in one-third of the workers. The incidence of obstruction was 43.7 percent in smokers vs. 36.6 percent in nonsmokers. The obstruction was particularly evident in 25.4 percent of the smokers and 18.9 percent of the nonsmokers. It was found that a combination of factors, such as smoking, atmospheric pollution, repeated virus infections, alcohol abuse, occupational and bacterial sensitization, were responsible for bronchial tree lesions, one of the main causes of frequent respiratory diseases in workers in a heavy machine building factory.

80–1183. Krzyzanowski, M. **Changes of Ventilatory Capacity in an Adult Population During a Five Year Period.** *Bulletin Europeen de Physiopathologie Respiratoire* 16(2): 155-170, 1980.

A followup study on chronic nonspecific respiratory disease was performed in Cracow on a probability sample of males and females aged 19-70 years. In 2,572 persons from the sample, complete interviews, as well as spirometric examinations, were performed twice, in 1968 and 1973. The relationships between the rate of forced expiratory volume in 1 sec (FEV_1) decrease in the 5-year period and the occurrence of various respiratory symptoms, age, sex, initial FEV_1 level, body height, smoking habit, education, air pollution level in the place of residence, were studied by multivariate analysis methods. It was found that the rate of FEV_1 decrease relates in the first place to the age of subjects under study, as well as to the changes of dyspnea and chronic bronchitis occurrence during the study period. A clear, but weaker, relationship with smoking habit was also found. The decrease in FEV_1 was faster in subjects who had been smoking longer and a slowing down of this process appeared in ex-smokers. However, no relationship could be found between the decreased FEV_1 rate and the number of cigarettes smoked daily. The greatest adjusted mean of the decreased FEV_1 indices (11.4) was computed for those smoking in both 1968 and 1973. The smallest adjusted mean decreased FEV_1 index (8.0) was obtained for the ex-smokers who gave up smoking before 1968. The education level, treated here as an index of socioeconomic conditions, was related to the rate of decrease in FEV_1 to a similar extent as the smoking habit. All variables considered in the analysis permitted an explanation of approximately 10 percent of the total variability of the FEV_1 changes. This suggests the existence of other factors having a bearing on the rate of decrease in ventilatory capacity. (Auth. Abs. Mod.)

80–1184. Liard, R.; Perdrizet, S.; Correman, J.; Bidou, S. **Smoking and Chronic Respiratory Symptoms: Prevalence in Male and Female Smokers.** *American Journal of Public Health* 70(3): 271-273, March 1980.

The relationship between respiratory symptoms and smoking habits, according to sex, was studied in 899 adults (average age 39, 55 percent male) in a Paris industrial medical center. The proportion of cigarette smokers was higher among males than females (57.0 vs. 41.4 percent). There were 94 ex-smokers (70 males and 24 females) in the group. Among males the proportion of heavy smokers (more than a pack a day) was higher than among females (41.0 vs. 25.8 percent). The relative risk of having chronic bronchitis among smokers, compared to nonsmokers, was higher in females (3.3) than in males (1.6). The prevalence of chronic respiratory symptoms, dyspnea, and wheezing was more closely associated with the number of cigarettes smoked per day in females than in males. No confounding factor was found to be responsible for these results. (Auth. Abs. Mod.)

80–1185. Loke, J.; Farmer, W.; Matthay, R. A.; Putman, C. E.; Smith, G.J.W. **Acute and Chronic Effects of Fire Fighting on Pulmonary Function.** *Chest* 77(3): 369-373, March 1980.

The acute and chronic effects of firefighting on pulmonary function were studied in 54 firefighters from Connecticut (32 smokers and 22 nonsmokers). Baseline studies of maximum expiratory flow-volume curves while breathing air and a mixture of 80 percent helium and 20 percent oxygen revealed obstruction of the small airways in 9 of 26 smokers (35 percent) and 2 of 15 nonsmokers (13 percent). Cigarette smoking appears to be a major contributor to obstruction of the airways in firefighters. In the nonsmoking group, disease of the small airways was present only in firefighters with at least 25 years of firefighting, and none of them had respiratory complaints. In seven firefighters retested immediately following mild exposure to a fire in a building, no significant changes in pulmonary function were noted. One firefighter (a cigarette smoker) trapped in a fire in a basement had a high level of carboxyhemoglobin (42 percent) and developed a severe obstructive ventilatory defect which persists 2-1/2 years after the fire. (Auth. Abs. Mod.)

80–1186. Mohsenifar, Z.; Tashkin, D. P. **Effect of Carboxyhemoglobin on the Single Breath Diffusing Capacity: Derivation of an Empirical Correction Factor.** *Respiration* 37(4): 185-191, 1979.

The effect on measured single breath diffusing capacity for carbon monoxide (DL_{CO}) of different blood concentrations of carboxyhemoglobin (COHb) produced by breathing carbon monoxide (CO) was studied in 19 healthy nonsmoking subjects. The DL_{CO} calculated without adjustment for blood COHb was found to be spuriously reduced by approximately 1 percent of the observed baseline value for each percent increase in blood COHb saturation. When measuring DL_{CO} of subjects who are likely to have elevated COHb blood levels, it is recommended that either blood COHb concentration or equilibrated alveolar carbon monoxide pressure be determined in order to correct the DL_{CO} for the effect of COHb. (Auth. Abs.)

80–1187. Nyqvist, B. **Luftvagssymtom hos Asfaltarbetare—Ytterligare en Yrkesbronkit? [Respiratory Tract Symptoms in Asphalt Workers—Another Occupational Bronchitis?]** *Lakartidningen* 75(12): 1173-1175, 1978, Swedish.

In order to investigate the effect of asphalt exposure on the respiratory system, 194 road workers and 37 roof layers were examined with reference to spirometric values and subjective symptoms of bronchitis. They were compared with a control group matched by age and smoking habits that had not been exposed to gases, smoke, or dust to any large extent. The spirometric values, forced vital capacity, and forced expiratory volume in 1 sec did not indicate any significant reduction of lung function due to exposure to asphalt fumes. Smokers of both the asphalt worker group and the control group showed a tendency to higher SR-values than nonsmokers. A simple questionnaire revealed a tendency to increased relative risk for bronchitis symptoms with increased exposure to asphalt. This relative risk is statistically significant only within the group that had been exposed to asphalt for more than 8 years. The relative risk for light symptoms after long exposure is 7 ($p=0.35$), based on 40 asphalt workers compared with their controls. The corresponding figure for severe bronchitis symptoms is 6 ($p=0.031$), based on the same number of individuals. The cases of bronchitis that are found within the group that has had long exposure to asphalt are almost exclusively found among the smokers and do suggest that tobacco smoking and exposure to asphalt are factors that potentiate the effect of each other. The increase of subjective bronchitis symptoms seems to be dose-dependent.

80–1188. Sorbello, A.; Giudice, J. C.; Komansky, H. J.; Gordon, R.; Kaufman, J. L. **Evaluation of Small Airways Dysfunction in Asymptomatic Cigarette Smokers: Comparison of Six Tests.** *Journal of the American Osteopathic Association* 79: 371-374, February 1980.

A prospective study was performed on 21 asymptomatic chronic cigarette smokers (who had smoked at least one pack of cigarettes per day for more than 5 years) to compare six tests in the evaluation of small airways function. Preliminary results suggest that the flow rate after 75 percent of vital capacity has been exhaled ($Vmax_{75}$) is the most sensitive of these indicators of small airways dysfunction in cigarette smokers. The test is effort-independent and easily measured from the flow-volume curve. The $Vmax_{75}$ may represent an excellent mass screening device for studies of chronic cigarette smokers who are at risk of developing disease of the small airways. (Auth. Abs. Mod.)

80–1189. Sterk, P. J.; Quanjer, P. H.; van der Maas, L.L.J.; Wise, M. E.; van der Lende, R. **The Validity of the Single-Breath Nitrogen Determination of Residual Volume.** *Bulletin Europeen de Physiopathologie Respiratoire* 16(2): 195-213, 1980.

The validity of the single-breath nitrogen determination of residual volume (RVsb) was tested by comparison with a concurrently applied method employing forced rebreathing in a closed circuit (RVmb). The study comprised 117 conscripts, 17 patients with severe airways obstruction, and 849 men and women studied in an epidemiological survey. In all groups RVsb was systematically smaller than RVmb, but the underestimation is neither related to smoking habits (mean 4.0 pack years, range 0.1-18.0) nor to respiratory symptoms. In order to detect the influence of other variables, the residuals to the double logarithmic regression were investigated with multiple regression analyses. These residuals were not related to height in any group of conscripts, but they were weakly negatively correlated to age in groups 1 and 2 (asymptomatic nonsmokers and smokers, respectively) ($p<0.05$) and to the slope of the alveolar plateau in group 1 ($p<0.05$) and in group 4 (respiratory symptoms regardless of smoking habit) ($p<0.001$). In the epidemiological surveys, the coefficient b (allowing for the standard error of b) is also less than one in all groups but in contrast to the conscripts it appears lowest for the three groups of symptomatic men and nonsmoking symptomatic women. No trend in b values with regard to smoking habits was observed. However, in asymptomatic men the underestimation of RVsb at 2 l is significantly greater ($p<0.05$) in the smokers than in the nonsmokers. In symptomatic subjects this underestimation is even greater; in women the differences between smoking and nonsmoking groups are less clear. Repeated measurements in the conscripts showed that within subjects the biological variability was 7.7 percent for RVmb and 7.3 percent for RVsb; the variability due to random errors was 3.3 and 2.4 percent, respectively. It is concluded that the single-breath method cannot be used interchangeably with other methods and is less suited for studying subjects with airways obstruction, on account of significant underestimations of RV. However, either the forced rebreathing method, or the single-breath result corrected for uneven ventilation (for which equations are given), is a useful alternative under such circumstances. (Auth. Abs. Mod.)

80–1190. White, J. R.; Froeb, H. F. **Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke.** *New England Journal of Medicine* 302(13): 720-723, March 27, 1980.

The effect of long-term passive smoking (involuntary inhalation of tobacco smoke by nonsmokers) and long-term voluntary smoking on specific indexes of pulmonary function was evaluated in 2,100 middle-aged subjects. Regardless of sex, nonsmokers chronically exposed to tobacco smoke had a lower forced mid-expiratory flow rate (FEF 25-75 percent) and forced end-expiratory flow rate (FEF 75-85 percent) than nonsmokers not exposed ($p<0.005$). In addition, values in passive smokers were not significantly different from those in light smokers and smokers who did not inhale ($p<0.005$). An examination of the extent to which smoke exposure is related to graded abnormality revealed that nonsmokers in smoke-free working environments have the highest scores on the spirometric tests; passive smokers, smokers who do not inhale, and light smokers score similarly and significantly lower; and heavy smokers score the lowest ($p<0.005$). It is concluded that chronic exposure to tobacco smoke in the work environment is deleterious to the nonsmoker and significantly reduces small-airways function. (Auth. Abs. Mod.)

80–1191. Windsor, R. A.; Green, L. W.; Roseman, J. M. **Health Promotion and Maintenance for Patients With Chronic Obstructive Pulmonary Disease: A Review.** *Journal of Chronic Diseases* 32(11/12): 5-12, 1979.

A review of the literature revealed that the health of patients with chronic obstructive pulmonary disease (COPD) can be promoted by providing more organized and tailored sets of educational experiences. Compliance with medication-taking regimens can be improved by working with the patient to clarify specific expectations, responsibilities, and procedures. The establishment of a therapeutic alliance facilitates patient involvement in assuming responsibility for his or her self-care. A significant proportion of patients with COPD who smoke represent a potentially receptive audience for advice and instruction on smoking cessation by the physician and other health care providers. A number of educational-behavior strategies, in particular patient contracts, have been found effective in improving patient behavior. These strategies are: (1) smoke less harmful brands (low tar and nicotine), different brands, or use nicotine or water filter; (2) smoke only half a cigarette and smoke only 2-3 min after craving; (3) keep a daily record of smoking behavior, identifying time, place, and reason for smoking; (4) smoke once every hour, 2 hours, 4 hours, or set maximum number for that day; (5) smoke only at specific times and places, or only before or after certain activities; (6) smoke a proportion less each day but agree on a specific data of cessation, i.e., 40, 30, 20, 10, and stop; (7) smoke regularly but agree to stop on a specified date, usually within 10-14 days, or agree to stop immediately; and (8) write up a contract for at least 3 months with physician and patient, and have it witnessed by wife or husband. (Auth. Abs. Mod.)

See also, **80–1129, 80–1155, 80–1156**

CARDIOVASCULAR DISEASES

80–1192. Braeuer, R.; Otto, C. **Die Kupierung pektanginoser Schmerzen mit inhaliertem Isosorbiddinitrat. Auswertung einer Fragebogenaktion. [Anginal Pain Arrested by Inhalation of Isosorbide Dinitrate. Evaluation of a Questionnaire.]** *Schweizerische Rundschau fuer Medizin* 68(4): 117-123, January 23, 1979, German.

Results from an evaluation of 285 questionnaires concerning the therapeutic efficiency of isosorbide dinitrate inhalation (Isoket spray) in patients suffering from coronary heart disease are reported. The questionnaires were returned by 97 Swiss physicians, most of whom were specialists in internal medicine. The patient-related data were evaluated according to age, smoking habit, weight, blood pressure, medical history, electrocardiogram, angina pectoris incidence, basic coronary heart disease therapy, and drugs used to arrest angina. Among the 260 patients, 27 percent were smokers and 73 percent were nonsmokers. Average age of the smokers was 59.7 years and of the nonsmokers, 66.5 years, a significant difference of 6.8 years. There were 90 patients with previous heart infarct; 31 percent of these were still smoking. Isoket spray was used by 269 patients with the effect felt predominately within 15 to 30 sec after two discharges of the spray. Side effects were noted in 26 percent of the patients. The data from 97 physician questionnaires showed that 88 percent of the patients and 89 percent of the doctors were satisfied with the treatment, and 66 percent of the doctors found that Isoket spray acted faster than nitroglycerine capsules.

80–1193. Bruppacher, R.; Staehelin, H. B.; Ritzel, G. **Entwicklung von Risikofaktoren fur kardiovaskulare Krankheiten im Laufe der Adoleszenz. [Development of Risk Factors for Cardiovascular Diseases During Adolescence.]** *Sozial- und Praeventivmedizin* 24(4): 234-235, August 1979, German.

The development of risk factors for cardiovascular diseases was examined in a study of 228 boys and 219 girls at age 14 1/2 and 4 years later. During that period, the average weight of the boys increased from 55.8 to 69.4 kg, and that of the girls from 52.9 to 57 kg. Diastolic blood pressure increased more in boys than in girls (from 68.2 to 76.1 mmHg and from 68.0 to 71.0 mmHg, respectively). Cholesterol, high density lipoprotein cholesterol, and triglyceride blood levels remained approximately the same. As to cigarette smoking, there were 31 percent regular smokers and 10 percent occasional smokers among both boys and girls at age 18 1/2.

80–1194. Byington, R.; Dyer, A. R.; Garside, D.; Liu, K.; Moss, D.; Stamler, J.; Tsong, Y. **Recent Trends of Major Coronary Risk Factors and CHD Mortality in the United States and Other Industrialized Countries.** In: Havlik, R. J.; Feinleib, M.; Thom, T.; Krames, B.; Sharrett, A. R.; Garrison, R. (Editors). Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality, Bethesda, October 24-25, 1978. NIH Publication No. 79-1610, May 1979, pp. 340-380.

Data from a number of studies, including the Pooling Project, show that decreases in the prevalence of three major lifestyle-dependent risk factors for coronary disease (CHD)—blood pressure, serum cholesterol, and cigarette smoking—have made an important contribution to the recent decline in CHD mortality rates in the United States. Serum cholesterol reduction is estimated to account for only 4 percent of the decline, blood pressure control for 30 percent, and reduced cigarette smoking for 65 percent. Data from international studies also show significant correlations between trends of lifestyle (such as dietary lipid and cigarette consumption) and trends of CHD mortality.

80–1195. Chen, M. K. **The Effects of Smoking and Exercise on Pulse Rate: The Metropolitan Life Company Experience.** Papers Presented at the U.S.P.H.S. Commissioned Officers Association, Phoenix, Arizona, April 16-19, 1979, 24 pp.

Data on smoking habit and physical activity were collected for 764 male employees of the Metropolitan Life Insurance Company over a 6-year period (1969-1975) in order to assess their effect on cardiovascular fitness. Resting pulse rate was recorded in participants who were then given a series of standardized exercises that consisted of jogging, rowing, bicycling, and situps for 90 sec each. Immediately after the exercises, the employee's pulse rate was again recorded, and subsequently at 1-min and 2-min intervals. Analyses of the data show that the pulse rates of physically active, nonsmoking subjects slowed toward normal much faster than those of inactive, smoking subjects. This phenomenon reflects the greater capacity of the cardiovascular system of the nonsmoking and physically active employees to cope with the stress of physical activity. Deficiencies in the data were discussed. (Auth. Abs. Mod.)

80–1196. Da Silva, A.; Widmer, L. K.; Ziegler, H. W.; Nissen, C.; Schweizer, W. **The Basle Longitudinal Study: Report on the Relation of Initial Glucose Level to Baseline ECG Abnormalities, Peripheral Artery Disease, and Subsequent Mortality.** *Journal of Chronic Diseases* 32(11/12): 797-803, 1979.

Glucose determinations were made as part of repeat examinations among 1,499 middle-aged employed men in Basel, Switzerland. Both prevalence of electrocardiogram (ECG) abnormalities and frequency of peripheral artery disease were analyzed to investigate possible association with initial glucose level. No consistent association was seen between baseline glucose and resting ECG endpoints, either in univariate or multivariate analyses. However, in multivariate (though not univariate) analysis, abnormalities on the postexercise ECGs were positively associated with elevated glucose. The number of cigarettes smoked was significantly related to major ECG abnormalities ($p<0.05$) and occlusive artery disease ($p<0.01$). Unexpectedly, peripheral artery disease was negatively associated with glucose elevation. In mortality analyses, too few deaths from coronary disease occurred in 5 years to permit any conclusions on long-term risk of hyperglycemia. The data available, however, did not show a positive association of mortality with glucose level. (Auth. Abs. Mod.)

80–1197. Daniels, L. M.; Kochar, M. S.; Itskovitz, H. D.; Hoffman, W. K.; Ettle, S. N. **Hypertension Control on an Urban University Campus.** *Journal of the American College Health Association* 27(5): 234-238, April 1979.

The University of Wisconsin/Milwaukee Student Health Services and the Milwaukee Blood Pressure Program have developed and implemented a campus hypertension detection and treatment program. Through publicity, education, and a variety of episodic and ongoing detection efforts, approximately 6,600 individuals were screened on campus. Elevated blood pressure levels were found in 17 percent of the subjects; of these, 106 students elected to be treated at the campus health center. The health center operation includes complete evaluation and treatment of hypertension, cardiovascular risk factor evaluation and education (including information about smoking and alcohol consumption), and modification programs. Of the hypertensive patients, 46 percent were symptomatic; 49 percent were diagnosed labile, 42 percent essential, and 3 percent secondary (oral contraceptive induced) hypertensives. Treatment of 75 percent was by lifestyle modifications; 23 percent received diuretic therapy, and 2 percent propranolol. Followup (range 3-15 months) has revealed that normalization of blood pressure was achieved in 49 percent. Substantial reductions also occurred in smoking and alcohol consumption. Between initial and followup evaluations, the percentage of nonsmokers or occasional smokers rose from 79 to 90, the percentage of <1 pack/day smokers fell from 13 to 9, and the percentage of 1-2 packs/day smokers fell from 7 to 1. Notable changes in weight control were also documented. (Auth. Abs. Mod.)

80–1198. Davies, D.M.; Smith, D. J. **Electrocardiographic Changes in Healthy Men During Continuous Low-Level Carbon Monoxide Exposure.** *Environmental Research* 21(1): 197-206, February 1980.

In separate experiments, six groups of young, healthy, nonsmoking human subjects lived in a closed-environment exposure chamber for 18 days, during the middle 8 of which they were exposed continuously to 50, 15, or 0 ppm of carbon monoxide (CO) in air. About half the subjects were tobacco smokers, but all were required to give up smoking 3 days before their CO experiment began, and no smoking or alcohol drinking was permitted during the experiments. Standard 12-lead electrocardiograms (ECG) were recorded from each subject during the control, exposure, and recovery periods. P-wave changes were clearly seen in 6 of 15 subjects at 50 ppm and in 3 of 15 at 15 ppm, but in none of 14 at 0 ppm; in addition, one subject showed marked S-T changes at 15 ppm. Review of a pilot study at 75 ppm CO revealed significant ECG changes in 7 of the 10 subjects. Mean (and range) midday carboxyhemoglobin levels in the control period were 0.5 (0.1-1.9) percent for the nonsmokers and 5.1 (2.0-8.2) percent for the smokers, and in the exposure period, 10.9 (8.4-12.1) percent for the nonsmokers and 14.9 (11.0-17.4) percent for the smokers. For seven subjects there was clear evidence of P-wave, S-T segment, or T-wave changes, and in one case supraventricular extrasystoles, in response to CO exposure. (Auth. Abs. Mod.)

80–1199. de Gennes, J. L.; Turpin, G.; Baulac, L. **Accidents Cardiovasculaires de la Contraception par Oestroprogestatifs et Anomalies des Lipides Circulants. [Cardiovascular Accidents From Oral Contraceptives and Circulating Lipid Anomalies.]** *Annales d'Endocrinologie* 38(6): 447-448, 1977, French.

Fifty-six major cardiovascular accidents are analyzed in women using oral contraceptives and suffering from familial hyperlipidemia. At the time of an accident, cholesterol and triglyceride levels were determined in 29 women out of 35 and were found clearly abnormal in 18 cases, that is, they exceeded the maximum levels found in a control group of 100 normal women who used Stediril (50 μg ethinyl estradiol + 0.5 mg norgestrel) over a 1-year period. Thus, the incidence of hyperlipidemia was 62 percent in women who had an accident vs. 2.5 percent in the general population of the same age. A total of 21 out of 35 women were hyperlipidemic. Among other risk factors, tobacco smoking (over 20 cigarettes/day) was found in four cases only. Three types of cardiovascular accidents were observed. Only in three out of eight coronary accidents in hyperlipidemic women was smoking found to be an associated risk factor. In conclusion, the metabolic factor of lipid abnormality, mostly of familial origin, prevails over all other risk factors, particularly hypertension, tobacco smoking, and vascular disorders.

80–1200. Fuller, J. H.; McCartney, P.; Jarrett, R. J.; Keen, H.; Rose, G.; Shipley, M. J.; Hamilton, P.J.S. **Hyperglycaemia and Coronary Heart Disease. The Whitehall Study.** *Journal of Chronic Diseases* 32(11/12): 721-728, 1979.

In a 5-year followup of 18,403 male London civil servants, aged 40-64 years, coronary heart disease (CHD) mortality has been examined in relation to baseline levels of blood glucose concentration measured 2 hours after a 50 g oral glucose load. When deciles of the blood sugar distribution were considered, no trend of age-adjusted 5-year CHD mortality rates was apparent. There was also no significant trend in the prevalence of Minnesota-Coded electrocardiogram (ECG) abnormalities with deciles of blood sugar. However, for individuals with blood sugar levels above the 98th centile (106 mg/dl for ages 40-59 years; 110 mg/dl for ages 40-64 years) there was approximately a doubling of CHD mortality, this increase being independent of age and blood pressure. The proportions of current smokers did not differ for subjects above and below the 98th centile. The prevalence of ECG abnormalities (Whitehall Criteria) was also increased above the 98th centile of blood sugar. In a multiple logistic analysis of ECG abnormalities and six variables (i.e., blood sugar, age, systolic blood pressure, weight/height, cholesterol, and smoking), smoking was significantly ($p<0.05$) associated with definite myocardial infarction abnormalities in 71 cases of ages 40-59 and 109 cases of ages 40-64. In another multiple logistic analysis smoking was also significantly ($p<0.001$) associated with 5-year mortality caused by cardiovascular and coronary diseases for ages 40-64. (Auth. Abs. Mod.)

80–1201. Greig, M.; Pemberton, J.; Hay, I.; MacKenzie, G. **A Prospective Study of the Development of Coronary Heart Disease in a Group of 1202 Middle-Aged Men.** *Journal of Epidemiology and Community Health* 34(1): 23-30, March 1980.

A prospective study of the development of coronary heart disease (CHD) in men aged 45-54 was conducted in six group practices in Belfast, Northern Ireland. Of the 1,343 men examined once by clinical, electrocardiographic, biochemical, and anthropometric methods, 1,202 free from CHD entered the prospective study. All 1,202 men were followed for at least 5 years, and the average annual incidence of CHD during this period was estimated as 1.73 percent (104 men developed CHD). Of the 28 measurements made at initial examination, four—S-T depression, total lifetime tobacco consumption, diastolic blood pressure, and height—were investigated in more detail. Using multidimensional contingency table methods, the probability of developing CHD was found to depend, in rank order of importance, on S-T depression, total lifetime tobacco consumption, and diastolic blood pressure. The risk of development was maximal (0.3732, four times the "random" risk) in men with S-T depression, greater than average total lifetime tobacco consumption, and a diastolic pressure $\geq$100 mm Hg. (Auth. Abs. Mod.)

80–1202. Gsell, O. **Hypertonie durch Rauchen oder Kaffeetrinken? [Hypertension From Smoking or Coffee Drinking?]** *Medizinische Klinik* 75(6): 8, March 14, 1980, German.

There is no evidence at present that smoking causes or maintains hypertension. On the average, blood pressure in smokers is somewhat lower than in nonsmokers. However, in the case of chronic hypertension, it is recommended that patients abstain from smoking because nicotine affects coronary and peripheral vessels and hence, indirectly affects blood circulation.

80–1203. Haynes, S. G.; Feinleib, M.; Kannel, W. B. **The Relationship of Psychosocial Factors to Coronary Heart Disease in the Framingham Study. III. Eight-Year Incidence of Coronary Heart Disease.** *American Journal of Epidemiology* 111(1): 37-58, January 1980.

An extensive psychosocial questionnaire was administered to 1,674 coronary-free individuals participating in the Framingham Heart Study between 1965 and 1967. The respondents were followed for the development of coronary heart disease (CHD) over an 8-year period. Type A women developed twice as much CHD and three times as much angina as Type B women. Among men aged 45-64 years, Type A behavior was associated with a twofold risk of angina, myocardial infarction, and CHD in general, as compared to Type B behavior. The association was found only among white-collar workers and was also independent of the standard coronary risk factors and other psychosocial scales. Serum cholesterol was only associated with CHD among white-collar workers, and cigarette smoking was only associated with CHD among blue-collar workers. This prospective study suggests that Type A behavior and suppressed hostility may be involved in the pathogenesis of CHD in both men and women. (Auth. Abs. Mod.)

80–1204. Hickey, N.; Mulcahy, R.; Daly, L.; Bourke, G.; Moriarty, J. **The Relationship Between Blood Glucose and Prevalence of Coronary Heart Disease: A Study in the Republic of Ireland.** *Journal of Chronic Diseases* 32(11/12): 767-772, 1979.

Volunteers in the mass screening program conducted in communities throughout the Republic of Ireland between 1969 and 1972 were invited for a repeat examination in 1974, during which serum glucose was measured 1 hour after a 50-g oral load. For the 3,357 40- to 54-year-old men seen at that time, univariate analysis of glucose level and prevalence of electrocardiographic (ECG) abnormalities indicative of coronary heart disease did not indicate a linear relationship. With multivariate analysis, considering also age, systolic blood pressure, relative weight, serum cholesterol, and cigarette smoking, no independent association was seen between glucose level and frequency of ECG abnormalities. However, there was a significant relationship between cigarette smoking and serum glucose ($p<0.01$) and between cigarette smoking and ECG abnormalities. (Auth. Abs. Mod.)

80–1205. Hoffmann, R. G.; Blumlein, S. L.; Anderson, A. J.; Barboriak, J. J.; Walker, J. A.; Rimm, A. A. **The Probability of Surviving Coronary Bypass Surgery. Five-Year Results From 1,718 Patients.** *Journal of the American Medical Association* 243(13): 1341-1344, April 4, 1980.

This study of 1,718 men with bypass surgery from 1970 to 1974 in Milwaukee has indicated that the following factors are significantly associated with 5-year survival: left ventricular end-diastolic pressure (LVEDP), plasma cholesterol level, coronary occlusion score, age, and left ventricular function (LVF). Diastolic blood pressure, triglycerides, height, weight, diabetes, and smoking were not significantly associated with 5-year survival. The 31 men with a previous stroke had a high risk (5-year survival was 32 percent). To serve as a guide to patient risk, a logistic model is used to predict 5-year survival. These probabilities are tabulated for all combinations of LVEDP, age, LVF, occlusion score, and plasma cholesterol level. (Auth. Abs. Mod.)

80–1206. Koch, A.; Hoffmann, K.; Steck, W.; Horsch, A.; Hengen, N.; Moerl, H.; Harenberg, J.; Spohr, U.; Weber, E. **Acute Cardiovascular Reactions After Cigarette Smoking.** *Atherosclerosis* 35(1): 67-75, January 1980.

The effect of cigarette smoking on the cardiovascular system was determined as follows: two cigarettes of relatively high (1.54 mg) and very low (0.08 mg) nicotine content were smoked and compared to sham smoking. After inhalation under standardized conditions there was a fivefold increase ($p<0.01$) of the plasma nicotine levels and a subsequent exponential decrease. Two hours after smoking the levels were still elevated. After two low-nicotine cigarettes, there was a significant ($p<0.05$) small short-term increase. The changes of the pulse rate were directly related to the nicotine levels ($p<0.01$) and the pulse pressure transit time from the heart to the calf, and the digital blood flow was indirectly related to them. Systolic blood pressure decreased until 45 min after smoking two high-nicotine cigarettes, at which time there was a significant increase ($p<0.01$) in blood pressure. The regulation of these parameters is exactly related to the nicotine levels probably through the release of catecholamines. The cardiovascular reactions after smoking may indicate the additional myocardial work load after cigarettes of different nicotine content. (Auth. Abs. Mod.)

80–1207. Kleinman, J. C.; Feldman, J. J.; Monk, M. A. **Trends in Smoking and Ischemic Heart Disease Mortality.** In: Havlik, R. J.; Feinleib, M.; Thom, T.; Krames, B.; Sharrett, A. R.; Garrison, R. (Editors). Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality. Bethesda, October 24-25, 1978, NIH Publication No. 79-1610, May 1979, pp. 195-217.

The possible impact of decreased smoking on ischemic heart disease (IHD) mortality is considered here. Published data from four studies (i.e., Veterans Administration, American Cancer Society (ACS), British physicians, Pooling Project) were used to estimate dose—response curves which were fitted to the observed data from each study. Goodness-of-fit statistics and smoothed death rates were generated; the latter were then applied to the distributions of the population by amount smoked in 1965 and 1976, to obtain "expected" IHD death rates. The linear dose—response model fit the VA study data best. This model provided an expected reduction of 8 percent, or 34 percent of the observed reduction. The logarithmic model was the best fitting in two cases, ACS and British physicians. The estimated risks from these studies were similar: ACS results showed a 9.3 percent decline (40 percent of observed), and results for British physicians suggested a 9.8 percent decline (42 percent of observed). The exponential model provided the best fit to Pooling Project data and resulted in an expected decrease of only 3.2 percent (14 percent of the observed). The data from these four studies were consistent with a wide variety of estimates for the potential impact of smoking (from 14 to 42 percent of the observed mortality decline). The limitations of the type of analysis presented include no consideration of lag times or type of cigarette smoked. These problems are discussed.

80–1208. Kolonel, L. N.; Hirohata, T.; Chappell, B. V.; Viola, F. V.; Harris, D. E. **Cancer Mortality in a Cohort of Naval Shipyard Workers in Hawaii: Early Findings.** *Journal of the National Cancer Institute* 64(4): 739-743, April 1980.

A retrospective cohort study was performed in Hawaii among 4,779 male shipyard workers exposed to asbestos and 2,757 similar male workers without known asbestos exposure. Observed deaths from cancer and other causes in the two groups were compared with expected deaths on the basis of the general population of Hawaii by the use of a modified life-table method of analysis. A risk ratio for lung cancer of 1.7 was found for the exposed group after 20 or more years of followup. No increased risk for lung cancer was seen in the nonexposed group. These findings could not be attributed to differences in smoking habits in the two shipyard groups or between the shipyard groups and the general population—58.8 percent of the general population, 63.8 percent of the exposed, and 62.5 percent of the nonexposed shipyard workers had ever smoked, and the lifetime use among smokers in the three groups was very similar. Because the maximum duration of followup for this analysis was 24 years, greater risks for lung cancer may be seen in the exposed group when the followup period is extended. (Auth. Abs. Mod.)

80–1209. Kuller, L. H.; LaPorte, R. E.; Weinberg, G. B. **The Decline in Ischemic Heart Disease Mortality: Environmental and Social Variables.** In: Havlik, R. J.; Feinleib, M.; Thom, T.; Krames, B.; Sharrett, A. R.; Garrison, R. (Editors). Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality, Bethesda, October 24-25, 1978. NIH Publication No. 79-1610, May 1979, pp. 312-339.

Studies on the relationship of environmental and social factors to the decrease in arteriosclerotic heart disease (ASHD) are reviewed. Cigarette smoking is a major source of environmental pollutants, such as carbon monoxide (CO), an important risk factor for heart attack. The levels of CO in cigarette smokers are far higher than from environmental pollutants. In studies relating social factors to ASHD there appeared to be a positive relationship between cigarette smoking and ASHD mortality, whereas a negative relationship was found for total alcohol consumption.

80–1210. Kurt, T. L.; Rabinowitz, B. O.; Huber, G. L.; Abelmann, W. H. **Hemodynamic Effects of Cigarette Smoking. (Letters).** *Circulation* 61(2): 460, February 1980.

In response to a recent study of systolic time intervals in healthy nonsmokers exposed to carbon monoxide (*Circulation* 60(4); 752-760, October 1979), a double-blind study of nine healthy, nonsmoking males exposed to carbon monoxide alone vs. filtered air, in a closed ventilation system is reported. The results suggest that carbon monoxide, while being inotropic immediately after exposure (2-3 min) by proportionately increasing left ventricular ejection time, reverses this at a later interval (19-22 min). This is interpreted as immediate compensation followed by decompensation. In their reply, the authors of the original article state that the concentrations of carbon monoxide and the conditions of that study are different from those encountered during their study. See also 79-1478.

80–1211. National Center for Health Statistics. **Chartbook for the Conference on the Decline in Coronary Heart Disease Mortality.** U.S. Department of Health, Education, and Welfare, Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, August 1978, 46 pp.

To assess mortality statistics and information about the declining trends in ischemic heart disease, the National Center for Health Statistics has published a chartbook which focuses attention on pertinent mortality trends. The chartbook also provides tabular data and graphic presentation of trends in ischemic heart disease. Information on the nature and sources of data, and the population bases for the rates are also provided.

80–1212. Prineas, R. J.; Jacobs, D. R., Jr.; Crow, R. S.; Blackburn, H. **Coffee, Tea and VPB.** *Journal of Chronic Diseases* 33(2): 67-72, 1980.

Anecdotes have long suggested that caffeine-containing beverages and other personal habits and excesses trigger palpitations and cardiac extrasystoles. A systematic population survey of 7,311 men, aged 37-57 years and free of any history of heart disease or diabetes was undertaken to examine the association of ventricular premature beats (VPB) with food and drink, cigarette smoking, and sleep habits. VPB were detected among 3.6 percent of nonsmokers, 3.2 percent of 1 to 15 cigarettes/day smokers, 3.6 percent of 16 to 20 cigarettes/day smokers, 4.1 percent of 21 to 29 cigarettes/day smokers, and 4.4 percent of ≥ 30 cigarettes/day smokers. The consistent upward trend of smoking and VPB is not, however, statistically significant. Blood pressure and serum cholesterol were also measured and a 2-min rest electrocardiogram recorded at a screening station. In multiple regression analysis, a significant relationship with prevalence of VPB was found for age and for the average daily number of cups of tea or coffee consumed, adjusting simultaneously for the influence of other factors. Heavy coffee consumption (≥ 9 cups/day) was associated with more than twice the likelihood of VPB being present at any age. (Auth. Abs. Mod.)

80–1213. Reunanen, A.; Pyorala, K.; Aromaa, A.; Maatela, J.; Knekt, P. **Glucose Tolerance and Coronary Heart Disease in Middle-Aged Finnish Men: Social Insurance Institution's Coronary Heart Disease Study.** *Journal of Chronic Diseases* 32(11/12): 747-758, 1979.

The relationship of plasma glucose 1 hour after an oral glucose load to the prevalence of symptoms and electrocardiogram (ECG) signs of coronary heart disease and to 4-year mortality was investigated in 3,351 men aged 40-59 years from the Finnish Social Insurance Institution's Coronary Heart Disease Study. The 1-hour post-load plasma glucose appeared to have no relationship to symptoms of coronary heart disease but showed a linear and partly independent relationship to ischemic ECG abnormalities. There were 19.9 percent never-smokers, 26.2 percent ex-smokers, and 53.9 percent smokers. The prevalence of smokers decreased and the prevalence of nonsmokers increased with the post-load plasma glucose level. During a 4-year followup, the 1-hour post-load plasma glucose level had no relationship to total mortality or mortality from cardiovascular diseases or coronary heart disease. (Auth. Abs. Mod.)

80–1214. Robitaille, N.-M.; Christen, A.; Dagenais, G. R.; Lupien, P. J.; Rochon, J. **Prevalence des Facteurs de Risque de la Maladie Coronarienne Dans la Region de Quebec. [Prevalance of Coronary Risk Factors in the Quebec Region.]** *Clinical and Investigative Medicine* 2(1): 13-16, 1979, French.

Hypercholesterolemia, hypertension, and excessive cigarette smoking were assessed in a French-Canadian population to determine whether their prevalence rates differ from those of other North American or French populations. In a random sample of 4,830 35- to 64-year-old men, living in the suburbs of Quebec City, hypercholesterolemia (≥ 240 mg/dl) was observed in 10.1 percent, arterial hypertension ($\geq 160/95$ mm Hg) in 21.3 percent, and excessive smoking (>20 cigarettes/day) in 39.2 percent of subjects. These results differ from surveys done in the United States of America and in France. A univariate analysis reveals no correlation between cholesterol and educational status whereas smoking and hypertension are negatively related to the number of years of education. (Auth. Abs.)

80–1215. Rosenberg, L.; Hennekens, C. H.; Rosner, B.; Belanger, C.; Rothman, K. J.; Speizer, F. E. **Oral Contraceptive Use in Relation to Nonfatal Myocardial Infarction.** *American Journal of Epidemiology* 111(1): 59-66, January 1980.

The relation of oral contraceptive (OC) use to the risk of hospitalization for myocardial infarction (MI) was evaluated among 121,964 U.S. nurses who responded to a mail questionnaire. There were 156 women who reported having been hospitalized for MI before the menopause, and 23 (15 percent) were OC users at the time of the MI. Of 3,120 controls matched to the cases for menopausal status at the time of the MI and for age, 304 (10 percent) were using OCs at the time of the MI. The apparent increase in the risk of MI for current OC users was not explained by cigarette smoking, hypertension, elevated cholesterol, or other identified risk factors for MI. It is estimated that OC use increased MI risk 1.8-fold overall, 2.8-fold among nonsmokers without other risk factors, 5.0-fold for current cigarette smokers with no other risk factors, and 8.9-fold for hypertensive smokers. The increase in risk attributable to the combined effect of current OC use, cigarette smoking, and hypertension was considerably greater than what would be predicted from the sum of the separate effects of these factors. (Auth. Abs. Mod.)

80–1216. Sakuma, K.; Hashimoto, T.; Maeda, Y.; Kusano, S.; Jitsukawa, H.; Ueda, A.; Morioka, M.; Kurihara, T.; Nagashima, A.; Hosoda, Y.; Kondo, K.; Fukuda, Y.; Ohtake, H. **Report on Hyperglycemia in Middle-Aged Male Japanese National Railways Workers.** *Journal of Chronic Diseases* 32(11/12): 779-786, 1979.

In a population study of railway employees in Japan, baseline examination was performed on 4,847 men aged 40, 45, and 50 in 1975. In order to study the significance of asymptomatic hyperglycemia for cardiovascular disease, analysis focused on men with no prior diagnosis of diabetes. An assessment of the frequency of electrocardiographic (ECG) abnormalities at various levels of glucose was made both excluding and including the large number of men on antihypertensive medication. Prevalence of definite myocardial infarct in this population was too low to permit any conclusion about possible association with asymptomatic hyperglycemia. However, all other ECG abnormalities indicative of coronary heart disease were more frequent at higher glucose levels. The association was seen in univariate as well as multivariate analysis, both when treated hypertensives were included and excluded. Among the 3,322 current smokers, those in the upper glucose levels had the highest rates of ECG abnormalities, but such clustering was either not present or less consistent for those who never smoked (913 men) or who quit smoking (434 men). (Auth. Abs. Mod.)

80–1217. Satiani, B. **A Practical Approach to Management of Chronic Arterial Insufficiency of Lower Extremities.** *Ohio State Medical Journal* 76(3): 165, 167-169, March 1980.

Intermittent claudication usually is easily identified by a comprehensive history and physical evaluation and objective confirmation with noninvasive vascular laboratory examination. Arteriography is necessary only when surgery is indicated. Dietary control, cessation of smoking, and a well-regulated exercise program are important in nonsurgical management. Cessation of smoking is strongly recommended since the occlusion rate of implanted prosthetic grafts and thus the amputation rate is much higher in smokers than in nonsmokers. Where surgical reconstruction is required, the results are excellent. Newer modalities in Doppler testing have made the diagnosis, qualitative assessment, and followup of patients with claudication much easier. (Auth. Abs. Mod.)

80–1218. Shephard, R. J.; Cox, M.; West, C. **Some Factors Influencing Serum Lipid Levels in a Working Population.** *Atherosclerosis* 35(3): 287-300, March 1980.

Lifestyle variables (perceived fitness, excess body mass, percent fat, lean mass, predicted maximum oxygen intake, cigarette consumption, alcohol consumption, use of contraceptive medication) have been related to the lipid profile (serum triglycerides, total cholesterol, high density lipoprotein (HDL) and low density lipoprotein (LDL) cholesterol) in a sizable volunteer population of Toronto officeworkers. Much of the fitness effect is related to body fat, which is positively correlated with triglycerides and (in the men only) with total and LDL cholesterol, but is negatively related to HDL cholesterol. However, maximum oxygen intake per unit of body mass shows a small independent association with triglycerides, total cholesterol (women only) and LDL cholesterol. Total cholesterol readings are unrelated to smoking habits, but triglyceride readings are significantly elevated in the smokers ($0.02>p>0.01$). The increase is more marked in cigarette than in pipe or cigar smokers, and within the cigarette smokers the increase of triglyceride levels is apparently dose-related ($p<0.001$). Smoking is also associated with low HDL cholesterol and (in women only) with high LDL cholesterol levels. Alcohol consumption is independently related to high values for total and LDL cholesterol, but is also related to high HDL cholesterol readings. (Auth. Abs. Mod.)

80–1219. Stenhouse, N. S.; Murphy, B. P.; Welborn, T. A. **Busselton Population Study: Risk Associated with Asymptomatic Hyperglycemia.** *Journal of Chronic Diseases* 32(11/12): 693-698, 1979.

The possible relation of asymptomatic hyperglycemia to prevalence of coronary heart disease was examined in the Busselton survey of a rural community in Western Australia. Among 649 men, age 40-59, the rate of electrocardiographic abnormalities in the highest quintile of post-load plasma glucose was, in univariate analysis, twice that observed in the lowest glucose quintile. In an 11-year mortality followup, similar univariate analysis showed no association of baseline glucose value and subsequent coronary or cardiovascular death. When such other factors as age, blood pressure, serum cholesterol, relative weight, and cigarette smoking (approximately 50 percent of the men were cigarette smokers, with most of them smoking ≥ 15 cigarettes/day) were considered in multivariate analysis, no association between initial glucose level and mortality was seen. The data do not give evidence that asymptomatic hyperglycemia was a significant risk factor for mortality in this population. However, caution is required in drawing conclusions, because of the relatively small number of coronary events. (Auth. Abs. Mod.)

80–1220. Tuomilehto, J.; Puska, P.; Virtamo, J.; Salonen, J.; Koskela, K. **The Levels of the Major Risk Indicators of Cardiovascular Diseases in Eastern Finland Prior to a Community-Based Intervention Programme (The North Karelia Project).** *Acta Cardiologica* 34(6): 359-374, 1979.

The design, implementation, and major results of a study for measuring the prevalent coronary heart disease (CHD) risk indicators in Eastern Finland are presented. The study formed the baseline survey of the North Karelia Project and was conducted in 1972 for a representative random sample of the population aged 25-59 in the county of North Karelia and a matched reference area. Altogether 11,992 persons were examined with a participation rate of 93 percent in North Karelia and 90 percent in the reference area. The prevalence rates of the "hard" CHD risk indicators (blood pressure, cholesterol, smoking, and triglycerides) are presented and reference is made to the prevalence rates of some other "softer" risk indicators. In general, the level of the "hard" risk indicators is high and this is especially true for the younger males. About half of the men and one-eighth of the women in the two areas were current smokers. Of the men, 4 percent had stopped recently. Percentages of nonsmokers decreased among men and increased among women toward the older age groups. On the other hand, the percentage of ex-smokers increased with age among males, so that the percentage of male current smokers was about the same in all age groups. Among women, smoking was clearly more common in towns than in the countryside; men did not differ in this respect. About 80 percent of the male and 90 percent of the female smokers smoked cigarettes. Some of the men smoked pipes (11 percent) or cigars (2 percent). The mean cigarette consumption of the men was 18, and of the women, 10 cigarettes/day. There were no major differences in the average daily consumption according to different age groups. Heavy cigarette consumption ($\geq$25/day) was observed in 20 percent of the male and in 2 percent of the female population. (Auth. Abs. Mod.)

80–1221. Williams, R. R.; Lyon, J. L.; Brockert, J. E.; Maness, A. T. **Decline in Coronary Mortality Rates: Utah vs. United States.** In: Havlik, R. J.; Feinleib, M.; Thom, T.; Krames, B.; Sharrett, A. R.; Garrison, R. (Editors). Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality, Bethesda, October 24-25, 1978. NIH Publication No. 79-1610, May 1979, pp. 48-57.

Mortality rates in Utah and the United States were examined to help explain the 20 percent decline in coronary heart disease (CHD) death rates during the period 1968-1976. Some observations suggest as much as 28 percent of the decline could be artifactually related to shifts to competing lethal diseases or recent increases in the accuracy of diagnosis of other conditions previously confused with CHD before the availability of improved diagnostic tests. The observed period of the CHD decline was also concurrent with increasing application of improved methods for treating lethal arrhythmias and congestive heart failure associated with CHD. Many health professionals hope that increasing interest in modifying CHD risk factors will result in preventing or delaying CHD deaths. Recent trends in cessation of cigarette smoking could be responsible for some decrease in CHD deaths in men. However, it would not explain the large decline in the largely nonsmoking population of Utah, nor in younger U.S. women who showed the largest percentage decline in CHD in a period when their smoking rates increased. The recent decline in CHD mortality rates likely reflects the influence of several of these factors. (Auth. Abs.)

PREGNANCY AND INFANT HEALTH

80–1222. American College of Obstetrics and Gynecology. **Cigarette Smoking and Pregnancy.** American College of Obstetrics and Gynecology, Technical Bulletin No. 53, September 1979, 4 pp.

Fetal, maternal, and various pathopharmacological effects of smoking during pregnancy are discussed. Placental weights are not significantly different between smokers and nonsmokers, but the fetal/placental weight ratio is significantly reduced. Offspring of mothers who smoked during pregnancy have been observed to have significantly lower IQ scores and increased incidence of reading disorders than matched offspring of nonsmokers. The incidence of minimal brain dysfunction syndrome (hyperactivity) is also reported as increased in the offspring of mothers who smoke. The risk of spontaneous abortion and of fetal or neonatal death is increased. The increase of perinatal mortality is directly related to the level of maternal smoking ranging from a 20 percent increase in women who smoke less than one package per day to a 35 percent increase in women who smoke more than one package. Abruptio placentae, a complication that, when severe, carries a 10 percent maternal mortality rate, occurs significantly more often in smokers than in nonsmokers. The risk of developing abruption is increased by 24 percent in moderate smokers and by 68 percent in heavy smokers. The increased risk of this complication in smokers may be related to its transient hypertensive effect or to specific arteriolar degeneration of uterine vessels observed in smokers. Placenta previa also occurs more frequently among smokers. The incidence of placenta previae is increased by 25 percent in moderate smokers and by 92 percent in heavy smokers.

80–1223. Finnegan, L. P. **In Utero Opiate Dependence and Sudden Infant Death Syndrome.** *Clinics in Perinatology* 6(1): 163-180, March 1979.

For many years, an increased incidence of sudden infant death syndrome has been seen in infants born to alcoholic and drug-addicted mothers. The relationship of passive cigarette smoking to sudden infant death syndrome also has been studied. Smoking habits of 56 families who lost babies to sudden infant death syndrome were compared with those of 86 control families. A higher proportion of mothers whose infants died with sudden infant death syndrome smoked both during pregnancy (61 vs. 42 percent), and after their babies were born (59 vs. 37 percent). These mothers also smoked a significantly greater number of cigarettes than did controls. In another study it was found that cigarette smoking caused a reduction in the incidence of fetal breathing movements in normal and abnormal pregnancies. The size of the reduction varied: it was greatest in small-for-date pregnancies and pregnancies complicated by fetal distress in labor, and was least in preeclamptic pregnancies. The fall in the amount of fetal breathing movements was significantly related to the rise in maternal plasma nicotine after smoking, but was unrelated to the rise in carboxyhemoglobin.

80–1224. Joffe, J. M. **Influence of Drug Exposure of the Father on Perinatal Outcome.** *Clinics in Perinatology* 6(1): 21-36, March 1979.

Animal and human studies of paternal drug exposure and perinatal outcome are discussed in this review. Among the topics considered are the effects of lead, ethanol, methadone, anesthetic gases, and smoking. In a survey of operating room personnel it was found that the rates of congenital abnormalities in children whose fathers were exposed to operating room anesthetics were higher than in children of unexposed fathers. In a study of parental smoking and low birthweight infants, it was found that if the father smokes there is no significant increase in low birthweight infants, as compared to a statistically significant increase if both parents smoke. Further, the highest incidence of neonatal mortality in low birthweight infants was found in offspring of nonsmoking mothers and smoking fathers. Other factors besides smoking habit, however, must also be considered in smokers and nonsmokers, such as coffee and alcohol consumption.

80–1225. Pelkonen, O.; Karki, N. T.; Koivisto, M.; Tuimala, R.; Kauppila, A. **Maternal Cigarette Smoking, Placental Aryl Hydrocarbon Hydroxylase and Neonatal Size.** *Toxicology Letters* 3(6): 331-335, June 1979.

Aryl hydrocarbon hydroxylase (AHH) activity was determined in placental samples from smokers and nonsmokers. When smokers and nonsmokers were analyzed as whole groups, the offspring of smoking mothers were about 240 g lighter and about 7 mm shorter at birth than those of nonsmoking mothers. The offspring from pregnancies in which placental AHH was elevated weighed about 400 g less and were over 1 cm shorter than those from nonsmoking mothers, whereas those from AHH-negative pregnancies were of the same size as those from nonsmoking mothers. The offspring of mothers who had stopped smoking after the first trimester of pregnancy were of the same size as those of nonsmokers. Differences between the offspring of mothers with prescribed drug intake—indicating pregnancy-related disorders which may affect birthweight—and with no drug intake were considerable within the groups of smokers. It is suggested that placental AHH be used as a measure of fetal exposure to maternal cigarette smoking. (Auth. Abs. Mod.)

OTHER DISEASES AND CONDITIONS

80–1226. Amindzhanov, S. A.; Achilova, A. B.; Rybin, I. S. **Kureniye Sigaret; Funktsii (Sekretsiia i Motorika) Zheludka pri Yazvennoi Bolezni Dvenadtsatipersnoi Kishki i Nekotorykh Drugikh Zabolevaniiakh Organov Pishchevareniia. [Cigarette Smoking and Stomach Functions (Secretion and Motility) in the Presence of Duodenal Ulcer and Other Digestive Organ Diseases.]** *Terapevticheskii Arkhiv* 52(2): 36-39, 1980, Russian.

Secretory and motor gastric functions have been tested in 53 male and 15 female patients, aged 21-65 years, with duodenal ulcer (47 patients) and other digestive organ diseases (21 patients), such as chronic gastritis, gastric erosion, chronic cholecystitis, and chronic hepatitis. In the patients with duodenal ulcer, all indices of gastric secretion increased after smoking two filter cigarettes: the average gastric juice content increased from 48.3 to 102 ml/hour, the total acid output/hour increased from 97.5 to 240.8 mg, the hydrogen ion secretion rate increased from 1.13 to 4.45 mg ion/l, the pepsin content increased from 2.4 to 5.5 mg percent, and the gastromucoproteid content increased from 18 to 43.8 mg percent. All these increases were statistically significant. The effect of cigarette smoking on gastric secretion was also significant in the patients with other digestive organ diseases. Passive smoking also increased the indices of gastric secretion in nearly all patients. The gastric juice content and the total acid output/hour increased in 10 out of 12 patients, the hydrogen ion secretion rate increased in 9, and pepsin and gastromucoproteid contents increased in 7 out of 12 patients. Electrogastrograms of the patients with duodenal ulcer show that the average rhythmic oscillation amplitude increased from 0.18 mV on an empty stomach to 0.35 mV after smoking two cigarettes, the rhythmic oscillation rate increased from 1.85 to 2.5 oscillations/min, and the energy ratio of gastric motility increased from 0.48 to 0.85. In seven patients with duodenal ulcer, cigarette smoking stimulated gastric juice secretion to the same or greater extent as a histamine injection. Preliminary data indicate that tobacco smoking may damage the stomach mucosa, aggravate the clinical process of duodenal ulcer, and cause development of reflexes.

80–1227. Angersbach, D.; Knauth, P.; Loskant, H.; Karvonen, M. J.; Undeutsch, K.; Rutenfranz, J. **A Retrospec-**

tive Cohort Study Comparing Complaints and Diseases in Day and Shift Workers. *International Archives of Occupational and Environmental Health* 45(2): 127-140, February 1980.

A retrospective cohort study was carried out in a chemical plant. The sick leave records of 370 shift workers in a 12-hour shift rotation and of 270 day workers with comparable work were analyzed from 1966 to 1977. The incidence of sickness in general did not differ between permanent shift workers and day workers, but was highest in shift workers who transferred later to day work mostly for medical reasons. Furthermore, on the average, shift workers were sick for longer periods than day workers. Significantly more shift workers than day workers consulted the occupational health services about gastrointestinal complaints than day workers. Shift workers had more frequent gastrointestinal diseases than day workers and more severe ones such as peptic ulcers. Gastrointestinal diseases were more frequent in the following groups of shift workers: Young (21 to 25 years) and unmarried subjects, heavy ($>$10 cigarettes/day) smokers as compared to nonsmokers ($p<0.1$), and subjects with a past history of gastrointestinal diseases. No differences between shift workers and day workers could be detected concerning cardiovascular diseases including myocardial infarction. Psychosomatic disorders were seldom diagnosed and did not differ between shift and day workers. Surprisingly, skeletal diseases including the sequelae of accidents and injuries occurred more often in the shift workers' cohort; the possible cause of this was a higher incidence of moonlighting in shift workers or other activities besides work. (Auth. Abs. Mod.)

80–1228. Drew, J. H. **Biliary Reflux: Postoperative and Idiopathic.** *Medical Journal of Australia* 2(3): 125, 128, August 11, 1979.

The clinical features of biliary reflux and its treatment (both conservative and surgical) are discussed and illustrative cases described. Biliary reflux has been found to result from cigarette smoking (if inhaled). Treatment which is directed toward keeping bile out of the stomach includes advising patients to reduce or stop smoking.

80–1229. Ducimetiere, P.; Kritsikis, S.; Richard, J.-L.; Pequignot, G. **Variation de Poids des Fumeurs d'Age Moyen Apres Modifications de Leur Consommation de Tabac. [Changes in Body Weight of Middle-Aged Smokers After Modification of Their Tobacco Consumption.]** *Revue d'Epidemiologie et de Sante Publique* 26(2): 195-198, 1978, French.

Changes in smoking habit and mean body weight within a 24-month period were determined in two groups of subjects with a high risk of ischemic heart disease and in a representative random sample of 133 subjects aged 38-45 years. One of the high risk groups (intervention) was repeatedly advised about smoking and nutrition. The control high risk group was not advised. Originally, 95 percent of the subjects in both high risk groups had smoked an average of 20 g tobacco/day. Only 53 percent of the representative group smoked an average of 14 g tobacco/day. Within the 2-year period, 30 percent of the intervention group subjects reduced their smoking by a minimum 5 g/day as compared to 16 percent in the control group, and 9 percent in the representative sample. Variations among the three groups were insignificant in the proportion of subjects who stopped smoking. The mean body weight decreased by 1.8 kg in the intervention group, while it remained constant in the control group or increased slightly in the representative group. Body weight decreased significantly (1.5-2.4 kg) regardless of smoking habit in the intervention group, while it increased by over 2 kg in the subjects who stopped smoking in the two other groups.

80–1230. Kaufman, D. W.; Slone, D.; Rosenberg, L.; Miettinen, O. S.; Shapiro, S. **Cigarette Smoking and Age at Natural Menopause.** *American Journal of Public Health* 70(4): 420-422, April 1980.

In a cohort of 656 naturally postmenopausal women who were interviewed at age 60-69 years, and who had reached their menopause between the ages of 35 and 59 years, the mean age at menopause declined with increasing number of cigarettes smoked. Mean age at menopause was 49.4 years among women who had never smoked, 49.2 among ex-smokers, 48.0 among women who smoked 1-14 cigarettes/day, and 47.6 among women who smoked $\geq$15 cigarettes/day. The differences between never-smokers and each of the categories of current smokers were statistically significant ($p<0.02$). However, among the current smokers the differences by smoking level were not significant. The relationship was not attributable to the onset of menopause inducing women to take up smoking. (Auth. Abs. Mod.)

80–1231. Koepsell, T. D.; Weiss, N. S.; Thompson, D. J.; Martin, D. P. **Prevalence of Prior Hysterectomy in the Seattle-Tacoma Area.** *American Journal of Public Health* 70(1): 40-47, January 1980.

The frequency of hysterectomy among women in the general population is of interest because it affects the population at risk for uterine diseases and because the procedure itself carries significant personal and socioeconomic consequences. Factors related to the occurrence of hysterectomy were studied by interviewing a representative sample of women aged 35-74 ($n=1,087$) in two urban Washington counties during 1976-1977. One-third of the women studied had had a hysterectomy. Later birth cohorts were at higher risk. The age-adjusted prevalence of prior hysterectomy was negatively associated with education and age at first childbirth; it was positively associated with parity, history of irregular menses, and history of a variety of other health conditions. Contrary to expectation, income was negatively associated with hysterectomy rates in one county and showed no association in the other. No significant difference was found between cigarette smokers and nonsmokers with regard to hysterectomy frequency. It is concluded that (1) despite economic predictions based on the discretionary nature of the procedure, hysterectomies are not necessarily more common among high-income women; (2) age at first childbirth may be a more important risk factor for uterine disease than previously thought; and (3) estimates of hysterectomy frequency based on clinic populations may be misleading. (Auth. Abs. Mod.)

80–1232. Madsbad, S.; McNair, P.; Christensen, M. S.; Christiansen, C.; Faber, O. K.; Binder, C.; Transbol, I. **Influence of Smoking on Insulin Requirement and Metabolic Status in Diabetes Mellitus.** *Diabetes Care* 3(1): 41-43, January-February 1980.

This study was performed in order to examine the influence of tobacco smoking on carbohydrate and lipid metabolism and microangiopathy in diabetic patients with normal serum creatinine. Among 163 adult insulin-treated patients, 114 smoked daily (smokers). Compared with nonsmokers, smokers had on the average a 15-20 percent higher insulin requirement ($p<0.001$) and serum triglyceride concentration ($p<0.05$), increasing to a 30 percent rise in heavy smokers ($p<0.01$). The degree of retinopathy was equal in the two groups, as was the average creatinine clearance (99 ± 2 (mean $\pm$ 1 SEM) vs. 101 ± 4 ml/min in smokers compared with nonsmokers). Smokers and nonsmokers were comparable regarding sex ratio, age at diabetic onset, duration of diabetes, residual beta-cell function, fasting hyperglycemia, and glycosuria. Evidently, tobacco smoking represents a strain on both carbohydrate and lipid metabolism in insulin-treated diabetes mellitus. (Auth. Abs.)

80–1233. Sudan, B.J.L.; Sterboul, J. **Sensibilisation Tabagique. Le Tabac: Un Allergene, La Nicotine: Un Haptene. Diagnostic par le Test de Degranulation des Basophiles. [Tobacco Sensitization. Tobacco: An Allergen, Nicotine: A Hapten. The Basophil Degranulation Diagnostic Test.]** *Nouvelle Presse Medicale* 8(43): 3563, November 5, 1979, French.

A case of familial atopy to tobacco smoke is described, the symptoms of which were sudden facial dermatitis, desquamation, and Quincke's edema. The diagnosis of atopic dermatitis was based on family history of allergy, regular occurrence of dermatitis crises after tobacco smoke exposure, positive skin test, and specific hyposensitization with allergenic extracts of tobacco leaves. All the patients with the same dermatitis had a positive test of in vitro degranulation of the basophils in the presence of tobacco allergen and nicotine hapten bound to human serum albumin. The lesions of the described dermatologic syndrome are exactly the same as those of seborrheic dermatitis. The syndrome is induced by nicotine hapten from tobacco smoke via degranulation of the tissue mastocytes.

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–1234. Botvin, G. J.; Eng, A. **A Comprehensive School-Based Smoking Prevention Program.** *Journal of School Health* 50(4): 209-213, April 1980.

Eighth, ninth, and tenth graders ($n=281$) from two schools in suburban New York participated in a study to test the efficacy of a 10-session comprehensive-psychosocial smoking prevention program focusing on self-improvement and development of basic life skills. Schools were randomly assigned to experimental and control conditions. All students were given a pretest, posttest, and 3-month followup which assessed smoking behavior as well as knowledge and personality variables. There were significantly fewer new smokers in the experimental school than in the control school at both the initial posttest ($p<0.01$) and the 3-month followup ($p<0.05$) along with significantly greater changes on some of the knowledge and personality variables. Overall, these results support the use of a smoking prevention strategy that focuses on the main psychosocial factors promoting the onset of cigarette smoking by teaching students basic coping skills. (Auth. Abs. Mod.)

80–1235. Buhl, F. **Le Tabagisme en France: Bilan d'une Premiere Campagne Nationale d'Information. [Tobacco Smoking in France: Results of the First Nationwide Information Campaign.]** *Revue Internationale d'Education Pour la Sante* 20(3): 174-181, 1977, French.

An antismoking campaign conducted in France from October to November 1976 was designed to disseminate a large volume of information among the entire population, particularly adolescents and pregnant women. Several opinion surveys after the campaign indicated that 88 to 90 percent of the adults, 75 percent of the adolescents aged 10-17 years, and 91 percent of the young mothers in the delivery rooms were aware of the campaign. The adult TV watchers (78 percent) and the high school students (84 percent) questioned were in favor of the campaign. After the campaign, 5 percent more adults and 7 percent more adolescents thought that smoking more than one pack of cigarettes/day was a health hazard. Also, 4 percent more adults and 8 percent more adult smokers indicated that smoking was a hazard to the health of passive smokers. Among the pregnant women, 6 percent more indicated that smoking was very dangerous to the unborn children. Three percent fewer adults and 1 percent fewer adolescents were smoking after the campaign. Among the smokers, 15 percent of the adults and 16 percent of the adolescents reduced their daily tobacco consumption. The campaign impact was confirmed when it was found that 4.2 percent fewer cigarettes were sold during the last trimester of 1976 than during the corresponding trimester of 1975. A complete text of the French law on smoking control is appended.

80–1236. Cohen, F. L. **Survey of Smoking in Institutions That Educate Health Professionals.** *Public Health Reports* 94(6): 544-552, November-December 1979.

This study was designed to determine current smoking practices and policies in various U.S. institutions in which health professionals are being educated. The survey showed that differences in smoking status among the administrators of different program types were statistically significant ($p<0.001$). Dental, nursing, and medical program administrators accounted for the highest percentage of current cigarette smokers, while program administrators in public health, health education, and elementary education accounted for the lowest percentage. The highest percentage of former cigarette smokers was found among respondents from public health and health education programs. Schools with larger student bodies were more likely to have written smoking policies and departments that sponsored some type of antismoking program ($p<0.02$). Departments of health education were far more likely to sponsor such programs than any other group. The majority of the antismoking programs were aimed at students and faculty; only half were geared to the general public. The smoking status of the program administrators was the chief influence on their perceptions of the role of health professional faculty and students in the restriction of smoking, in public education about smoking, and in other areas. (Auth. Abs. Mod.)

80–1237. Elkind, A. K. **Smoking Among the Staff of a Medical School.** *Medical Education* 13(3): 163-171, 1979.

A postal questionnaire sent to the staff of Manchester Medical School ($n=483$, response 86 percent) examined their smoking behavior and views about smoking in relation to their students. Of the staff, 12 percent were regular cigarette smokers and 45 percent ex-smokers. As for other forms of tobacco, 11 percent of the staff members smoked a pipe and another 11 percent were cigar smokers. Half of the under 35 age group were nonsmokers compared to only one-fifth of those over 50. Long-term health risks and expense were the main reasons for trying to give up cigarettes, whereas the need to set an example, in particular to students, was not stressed. A minority (19 percent) smoked when teaching, but fewer smoked when with students than with their colleagues. A quarter of the staff allowed students to smoke during teaching sessions; fewer than half thought students should be persuaded not to smoke; and four out of ten considered that staff smoking does not deter students from giving up smoking. Degree of contact with students and qualifications had some influence on these views, but age was more important. Smokers were no more likely to permit smoking during teaching but were less likely to value persuasion or to think staff smoking deters students from giving up smoking. (Auth. Abs. Mod.)

80–1238. Hendtlass, J. **Survey of Alcoholism Treatment Services in Victoria. Relationship of Alcoholics' Drug Use to Their Use of Medical Facilities.** *Medical Journal of Australia* 2(12): 635-636, December 15, 1979.

A survey of 370 individuals treated at 9 Victorian alcoholism treatment centers showed that men and women alcoholics did not differ significantly in indices of their lifetime consumption of alcohol or tobacco per kilogram of body weight, despite considerable differences in their present use of these drugs. Men smoked more cigarettes and reported heavier alcohol use than women ($p<0.05$ for both). Physical disability was associated with extended tobacco consumption both in men and in women ($p<0.01$ and $p<0.05$, respectively). While heavy use of health services was not associated with present smoking habits, women's use of health services was positively correlated with tobacco lifetime consumption ($p<0.05$). (Auth. Abs. Mod.)

80–1239. Krasnegor, N. A. (Editor). **Cigarette Smoking as a Dependence Process.** National Institute on Drug Abuse Research Monograph Series, No. 23, DHEW Publication No. (ADM) 79-800, January 1979, 200 pp.

This monograph includes papers presented at a symposium held in June 1978, sponsored by the National Institute on Drug Abuse and the Committee on Substance Abuse and Habitual Behavior of the National Research Council. The object of the meeting was to review current knowledge on the psychosocial, behavioral, and psychobiological factors which characterize the dependence process associated with cigarette smoking and make cessation difficult. Among the topics discussed were (1) smoking as an addictive disorder; (2) cigarette smoking as a precursor of illicit drug use; (3) regulation, withdrawal, and nicotine addiction; and (4) the tobacco withdrawal syndrome. A brief agenda for future research on smoking is also provided.

80–1240. Lavrova, I. G.; Malaia, E. V.; Benevolenskii, A. V.; Shatalova, A. P. **Problema Rasprostranennosti Kurenila Sredi Studentov Medikov. [The Problem of Smoking Incidence Among Medical Students.]** *Sovetskaya Meditsina* 11: 144-147, November 1978, Russian.

Results are reported of a smoking survey of 803 5th and 6th year students at 7 Soviet medical institutes. Analysis of the questionnaires revealed that 50 to 85 percent of the men and 2 to 40 percent of the women were smokers. Only 20-30 percent of the male students smoked before enrolling in an institute. Female students smoked ≤ 10 cigarettes/day. Of the male smokers, 90 percent smoked <20 cigarettes/day. Smoking incidence was the same for 6th year students living in a dormitory as for those living in private dwellings. However, the incidence in 1st year students living in a dormitory was 20 times higher than that in students living in private dwellings. There were more smokers among the students who lived with other smokers. The majority of the students started smoking because of curiosity or were influenced by friends. A majority also considered smoking a bad habit, and 10-15 percent of the students at a Moscow institute admitted that smoking is unhealthy. A large majority of the students smoked in the presence of close friends, and over one-third of the male students smoked in the presence of children or sick persons, although they admitted that passive smoking was hazardous to health. Over one-half of all students believed smoking to be compatible with sports. The percentage of male students who wanted to quit smoking varied from 50 to 88, that of female students varied from 42 to 65. However, the relapse rate was 80 to 100 percent among all students after a short period of abstinence. Health deterioration motivated 38 to 100 percent of the male students and 25 to 75 percent of the female students to quit smoking.

80–1241. Leathar, D. S.; Davies, J. K. **Smoking Among Student Nurses.** *World Smoking and Health* 4(3): 22-24, Autumn 1979.

In a 1977 survey by the U.K. Department of Health and Social Security of the levels of smoking among several professional groups, it was found that 48 percent of hospital nurses smoked, more than any other group. The Scottish Health Education Unit carried out research to learn why. Indepth interviews were conducted with 93 nurses to determine the problem areas and how to approach them. The subjects were general, psychiatric, and pediatric nurses, from beginners to senior administrators. The crucial time for smoking among nurses appeared to be the first 18 months of training when many began to smoke and many others increased their smoking. The psychological pressures, work responsibilities and problems, and outside friendship groups help initiate and maintain a student nurse's smoking habits. (Auth. Abs. Mod.)

80–1242. Malaia, E. V. **O Bor'be s Kureniem Sredi Studencheskoi Molodezhi. [Control of Smoking Among the Student Youth.]** *Sovetskoe Zdravookhranenie* (5): 32-34, 1979, Russian.

A survey was made of smoking habits in 1,880 senior students of 13 medical, technical, pedagogical, and theatrical institutes. Analysis of the survey results showed that 20-40 percent of the male and 0-4 percent of the female first-year students smoked. In their senior years, up to 90 percent of the men and 60 percent of the women smoked. Among the students, 67 percent of the men and 85 percent of the women believed that smoking was compatible with sports. Most of the men smoked ≥ 20 cigarettes/day while most of the women smoked 10 cigarettes/day. Up to 64 percent of the male smokers and 54 percent of the female smokers in their senior years wanted to stop smoking because of deteriorating health, but resumed smoking after a short period of abstinence. Development of a wide-range program of medical and social propaganda is recommended for control of smoking among students.

80–1243. Malkin, S. A.; Allen, D. L. **Differential Characteristics of Adolescent Smokers and Non-Smokers.** *Journal of Family Practice* 10(3): 437-440, March 1980.

This survey concerning adolescent smoking behavior includes an 82 percent sample of the entire 8th and 11th grades (229 students) of a rural school in the United States. A relatively high incidence of smoking (31 percent) is noted when compared with national statistics. No differences between smokers and nonsmokers were found with respect to knowledge of smoking effects, athletic self-perception, or exposure to the smoking behavior of teachers, physicians, dentists, or clergy. Significant differences ($p<0.001$) were found with respect to exposure to the smoking behavior of parents, siblings, and peers. A significant association between smoking males and depression was noted ($p<0.02$). There was no significant correlation between smoking behavior and depression for females. Efforts are in process to decrease peer smoking exposure by altering school policy. It is predicted that success in decreasing opportunities for exposure will result in a lower incidence of smoking in this population. (Auth. Abs. Mod.)

80–1244. Mueller, R.; Cahannes, M.; Tecklenburg, U.; Weiss, W. **Der Einfluss des schulischen Milieus auf das Rauch- und Trinkverhalten von Schulern. [Effect of the School Environment on Smoking and Drinking Habits of Students.]** *Sozial- und Praeventivmedizin* 23(4): 263, August 1978, German.

Students selected from the 6th, 7th, and 8th grades from all regions of Switzerland were asked to fill out a questionnaire about their smoking and drinking habits. The preliminary results are based on the answers of 2,491 students from the German-speaking region of Switzerland. About 86 percent of the boys first tried smoking at age 12 compared to 43 percent of the girls; the respective percentages at age 16 were 90 and 76. Comparable findings were noted with respect to alcohol consumption. The findings confirm the hypothesis that adolescent smoking and drinking increase with growing symbolic participation in adult values. Adolescents also tend to consume tobacco and alcohol as tension releasers, a conclusion based on findings that show a correlation between school adjustment and frequency of tobacco or alcohol consumption.

80–1245. Rimpela, M.; Kantanen, M.; Kokko, S.; Vuori, H. **Laakarien Terveyskayttaytyminen. [A Study of the Health Behavior of Physicians.]** *Suomen Laakarilehti* 31(34): 2639-2648, December 1, 1976, Finnish.

In a Finnish study, 1,644 male physicians were compared with 594 male engineers with regard to the effect of training and profession on smoking habits. The percentage of daily smokers was 31 percent in physicians and 26 percent in engineers, but this difference was compensated for by a higher average daily consumption of tobacco among engineers. For the under 40 age group, 11 percent of the physicians and 18 percent of the engineers smoked more than 24 g tobacco/day. For the over 40 age group the percentages were 20 and 34, respectively. Of the smoking physicians, 55 smoked >14 cigarettes/day as compared to 67 percent of the smoking engineers. The tobacco consumed as cigarettes was lower (64 percent) for physicians than for engineers (72 percent) or the total male population over age 15 (88 percent). The consumption of cigars and cigarillos together was inverted for these three groups (17 vs. 12 vs. 1 percent, respectively). Pipe tobacco consumption was 19 vs. 16 vs. 11 percent, respectively. Of the physicians, 24 percent, and of the engineers, 23 percent had tried to quit smoking during the past year. In both groups, one-third of the smokers had started to smoke before college age. However, more physicians than engineers had started to smoke in college. There were few significant differences with regard to tobacco political actions. Prohibition of the sales of tobacco in hospitals was supported by 56 percent of the physicians and 43 percent of the engineers. The prohibition of tobacco advertisement was supported by 90 percent of the physicians, 78 percent of the engineers, and 83 percent of the male population 25-49 years of age. The tobacco prices should increase at least as much as the general cost of living according to 76 percent of the physicians, 74 percent of the engineers, and 57 percent of the total male population aged 25-49.

80–1246. Russell, M.A.H.; Sutton, S. R.; Feyerabend, C.; Saloojee, Y. **Smokers' Response to Shortened Cigarettes: Dose Reduction Without Dilution of Tobacco Smoke.** *Clinical Pharmacology and Therapeutics* 27(2): 210-218, February 1980.

This study was designed to examine the response of smokers to shortening their usual brand of cigarettes. The shortening reduces the dose of smoke available from each cigarette without affecting concentration and therefore differs from dose reduction by dilution, which occurs when smokers switch to cigarettes with lower tar and nicotine deliveries. Measures of smoking behavior (e.g., cigarette consumption, puff rate), mouth-level nicotine intake (calculated from butt content), and intake to the lungs (plasma nicotine and carboxyhemoglobin) were made in 10 smokers (9 females and 1 male) after 48 hours ad libitum smoking of full, three-quarter, and half-length cigarettes in a Latin square design. Mouth-level smoke intake was maintained on shortened cigarettes due to a combination of two types of compensatory maneuver: (1) by increasing the intensity of puffing and thereby extracting proportionately more of the smoke available from each cigarette and (2) by smoking more cigarettes. The amount of smoke inhaled, on the other hand, was only partially maintained (58 percent compensation). This was achieved by increase in cigarette consumption alone. There was no evidence of any compensatory increase in the amount of smoke inhaled from each cigarette. Increase in consumption was thus the only maneuver that contributed to maintaining smoke intake at lung level; mouth-level intake was regulated by increasing intake per cigarette as well as consumption. (Auth. Abs.)

80–1247. Wilson, R. W.; Wingender, M. K.; Redican, K. J.; Hettler, W. **Effects of the Health Hazard Appraisal Inventory on Practices of College Students.** *Health Education* 11(1): 28-30, January-February 1980.

The purpose of this study was to determine the effects of the Health Hazard Appraisal (HHA) inventory on the health practices of college students. A random sample of 170 undergraduate students was selected from a student population of 22,000. Six weeks after inventory completion, the 89 responding subjects (52.7 percent) were invited to attend an information session where 27 received the computer analysis printout which graphically illustrated three life expectancy predictions and comparisons, specific I hazards, probable causes of death, and suggestions for health improvement. The remaining 62 subjects became controls. After a discussion period, the students were given a booklet containing information on negative lifestyles and their effects on longevity. One month later a followup was conducted and baseline HHA data were examined. Of three behavioral variables (i.e., smoking, drinking, seatbelts), only smoking was significantly different in the experimental and control groups ($p=0.005$). Of those reporting smoking at the beginning of the study (5 experimental, 10 control), 6 (controls) were lost in followup. Of those smokers reached in followup, 4 had cut down or quit (2 experimental, 2 control).

See also, **80–1163**

80–1248. Conway, J. B. **A Handbook of Self-Control Procedures for Cigarette Smokers.** American Psychological Association, Journal Supplement Abstract Service Manuscript No. 1021, Washington, D.C., 1975, 43 pp.

This handbook is designed to provide behaviorally based self-control strategies for smokers. Versions of the handbook have been tested in individual and group treatments and have been used in combination with a variety of aversive conditioning procedures. An introduction explores general notions of behavioral self-control of smoking. Chapter 1 examines the smoker's motivations for wanting to quit, establishes some ultimate aversive consequences of continued smoking, and suggests several cognitive-behavioral self-control techniques. Chapter 2 outlines a variety of strategies for limiting conditions under which cigarettes are smoked and bringing under control the stimuli associated with smoking. Chapter 3 details the basic procedures of goal setting, self-contracting, and contingency self-management. Chapter 4 reviews self-control procedures for the maintenance of nonsmoking, and suggests activities to replace smoking, especially self-relaxation exercises. (Auth. Abs.)

80–1249. Danaher, B. G. **Smoking Cessation Programs in Occupational Settings.** *Public Health Reports* 95(2): 149-157, March-April 1980.

For reasons of health and economics, the business community is displaying a growing interest in providing smoking cessation programs for employees. An examination of the current research on smoking cessation methods revealed a number of promising directions that smoking cessation programs can take, for example, aversive smoking approaches combined with self-control strategies. A review of current smoking cessation programs in occupational settings revealed some emphasis on physician counseling, but a relatively greater emphasis on use of consultants (especially in proprietary programs) or of contingency programs to encourage nonsmoking. The smoking cessation programs in businesses can move in a number of innovative directions, including (1) increased use of inhouse programs with a variety of smoking cessation strategies; (2) greater emphasis on the training of program participants in nonsmoking behavioral skills, combined with contingency or incentive programs for smoking control; (3) vastly improved research methods, including complete followup assessments of program participants and chemical tests to validate their self-reported abstinence; (4) greater concern about the need for empirically tested procedures for recruitment of participants for the programs; and (5) expanded interchange among behavioral scientists (especially behavioral psychologists), health professionals in occupational health and medicine, union and employee groups, and management. (Auth. Abs.)

80–1250. Jaloustre, H. S.; Ruasse, J.-P. **Lutte Contre le Tabagisme. [Smoking Control.]** *Concours Medical* 99(33): 4832, September 10, 1977, French.

In answer to a reader's questions about (1) a brand of antismoking pills and (2) acupuncture efficiency in smoking withdrawal treatment, the pills' composition, the principle of treatment, and primary and secondary effects are given, as is an opinion on acupuncture. The pills' primary effects are comparable to those of nicotine but without accompanying toxicity, addiction, or side-effects. In addition, the pills decrease nervousness and anxiety, and improve metabolism. Acupuncture is often recommended as cases of its successful application are known.

80–1251. LeCron, L. M. **How to Stop Smoking Through Self-Hypnosis.** North Hollywood, California, Wilshire Book Company, 1979, 190 pp.

In the first few chapters, reasons for smoking (oral, tension, addiction) are discussed as are advantages for cutting down or quitting (health, saving money, accomplishment). It is pointed out that nicotine is a poisonous addictive drug that is not totally removed from the body until about a week after quitting smoking. A general discussion of the benefits and uses of hypnosis ensues. It is claimed that the more intelligent subject is the better subject. Other types are said to be hard to hypnotize because of poor concentration and inability to follow suggestions. It is stated that one may learn self-hypnosis after first being hypnotized by a certified professional. A daily program for the addict or heavy smoker is given. This 3-week program includes means such as gum, lozenges, tranquilizers, lobeline sulfate, mouthwash, distraction, breathing exercises, visual imagery, and self-hypnosis.

80–1252. Miller, G. H.; Rodway, J. S. **The Cummins Engine Company Stop Smoking Program.** *Journal of the Indiana State Medical Association* 73(4): 233, April 1980.

An SOS Stop Smoking Clinic for employees was recently inaugurated by the Cummins Engine Company. The program consists of a 1-year course and uses the "reverse peer pressure system," which involves the assistance of friends and the immediate family of the smoker. In addition, the "buddy system" is used to assist participants. Group counseling and individual counseling are also provided. The following results for 33 participants were reported after 4.5 months of the clinic's existence: 23 persons (70 percent) had stopped smoking, 6 (18 percent) were still trying to stop, and 4 (12 percent) were again smokers.

80–1253. Pene, C.; Kelledjian, A. J.; Klein, M. **A Propos d'une Tentative d'Arret Tabagique par Auriculotherapie en Clientele O.R.L. Resultats Apres un An. [Concerning an Attempt at Smoking Withdrawal by Auricular Therapy of Otorhinolaryngology Patients. Results After One Year.]** *Medecine d'Afrique Noire* 24(12): 853-854, December 1977, French.

Two hundred healthy subjects who have been smoking and inhaling cigarettes for more than 5 years were treated by application of surgical agraffes to two specific points on the pavilion of the ear for 2 to 3 days, and then by transfixing the helix base with a wire and leaving the wire in place for 1 month. The operation was repeated 1 week later in case smoking withdrawal was not complete. A complete withdrawal was noted in 62 percent of the subjects and a very substantial reduction of smoking in the rest immediately after application of the wire. The percentage of complete withdrawals decreased to 50, 34, and 24 after 1 week, 3 months, and 1 year, respectively. Twenty subjects treated by repeated wire stimulations every 8 days relapsed after 1 year to their original consumption. It is advisable to supplement this treatment with simple psychotherapy, frequent absorption of fruit juices (vitamin C), respiratory exercises, and, if necessary, light sedatives and a diet.

80–1254. Poupy, J.-L. **Traitement Antitabagique par Acupuncture. [Acupuncture as Smoking Withdrawal Therapy.]** *Gazette Medicale de France* 84(27): 2897-2900, September 16, 1977, French.

Short-term results are reported of the use of acupuncture in the smoking withdrawal treatment of 350 patients who had smoked an average of 30 to 35 cigarettes/day. Treatment consisted of inserting needles in appropriate cephalic points for 30 min each time. After 8 days, the treatment was repeated. Prior to the second treatment, 49 percent of the treated patients quit smoking completely, 37 percent reduced their smoking by over 80 percent, and 14 percent reduced their smoking by less than 80 percent. Five weeks after the second treatment, 77 percent of the patients acknowledged complete smoking withdrawal. Seventy percent of the patients who quit smoking did not feel any urge to smoke, 48 percent had some trouble in suppressing the desire to smoke, and 32 percent expressed revulsion to smoke. The side effects of withdrawal were increasing nervousness (60 percent), insomnia (37 percent), bulimia (47 percent), modification of food taste (27 percent), and increase of olfactory perception (40 percent). The treatment causes a revulsion to one's own smoking but not to the smoking of others. Some smokers merely reduced their smoking, e.g., from 40 to 5 cigarettes/day.

80–1255. Puska, P.; Koskela, K.; McAlister, A.; Pallonen, U.; Vartiainen, E.; Homan, K. **A Comprehensive Television Smoking Cessation Programme in Finland.** *International Journal of Health Education* 22(4, Supplement): 1-29, October-December 1979.

A comprehensive smoking cessation program was presented on Finnish television in the spring of 1978. It included general information to the public and was supported by health services and voluntary health organizations. The actual television course was based on the method developed and tested in the North Karelia project. The program consisted of seven sessions of approximately 45 minutes each and involved the participation of a studio group of 10 smokers. A nationwide evaluation of the program showed (1) more than half the population saw at least one session of the program and 7 percent saw at least four sessions; (2) the program was mainly seen at home and just as much by smokers as by nonsmokers; (3) of all the smokers in the country, 10 percent followed the program closely, over 2 percent stopped smoking, and 1 percent succeeded for a period of at least 6 months; (4) in spite of this success, the impact on national smoking rates was limited. However, where intensified field activity accompanied the program, the impact was doubled.

TOBACCO ADDITIVES

80–1256. Kovats, E.; Demole, E.; Ohloff, G.; Stoll, M. **Cycloaliphatic Unsaturated Ketones as Odor and Taste Modifying Agents in Tobacco Products.** United States Patent No. 4,187,863, February 12, 1980, 30 pp.

New cycloaliphatic ketones are being used as flavoring and taste modifying agents in tobacco products, food, beverages, animal feed, and pharmaceutical products. The compounds can be used in proportions ranging from 0.1 to 10 ppm. Depending on the products to which the compounds are added, fruity, herb-like, wine-like, floral, or berry notes will develop.

80–1257. Triest, F. J. **Smokers' Flavor Concepts Evolving as Additives' Importance Increases.** *Tobacco International* 181(17): 20-21, August 24, 1979.

Flavoring additives are ameliorants or intensifiers to the basic cigarette flavor. Ameliorants include varieties of vanilla, tonka, deertongue, caramel, maple, rum, prune, fig, raisin, and St. John's bread. These flavorings are used to smoothen or to mildly sweeten the smoke and to blend the various natural smoke flavor ingredients. They are also masking agents for off-notes contributed by inferior grades of leaf used in the blend. This category of ameliorants includes menthol. The demand for flavor intensifiers was created by the filtered cigarette. Natural tobacco extractives best restore the flavor loss due to dilution of the smoke stream through aeration. High filtration cigarettes are flavored in several stages. The restoration of individual flavor ingredients lost during filtration is accomplished through the addition of aroma chemicals to the cigarette. Vanillins, coumarins, formates, and valerates, which have been used for years for this purpose, have recently been joined by aroma chemicals originally designed for food products.

See also, **80–1080**

TOBACCO MANUFACTURING AND PROCESSING

80–1258. Atanasov, B. **Khimicheski Sustav i Pushatelni Svoistva na Napravliavano Sushen Orientalski Tiutiun. [Chemical Composition and Smoking Characteristics of Artificially Dried Oriental Tobacco.]** *Bulgarski Tiutiun* 22(3): 21-28, 1977, Bulgarian.

Oriental tobacco leaves of the Kharmanliyska Basma 163 and the Pazardzhik 17 varieties were processed by three different artificial drying and curing techniques: conventional flue curing at 50°C and 65 percent moisture for 35-60 hours (I), high-temperature drying (90-100°C) for 50-70 min (II), and drying at above cited conditions combined with curing at 42-45°C and 65 percent moisture for 24 hours (III). Chemical analysis of the leaves processed by these techniques revealed a significant difference in composition, except for protein content, compared to that of the sun-dried uncured leaves. The nicotine content (in percent of dry tobacco) changed, depending on sampling, from 0.75, 0.55, 0.39, or 0.42, to 0.68, 0.65, 0.52, or 0.26 after treatment I; to 0.68, 0.52, 0.49, or 0.23 after treatment II; and to 0.71, 0.52, 0.55, or 0.26 after treatment III, respectively. The tobacco samples processed by technique II were found to possess the best smoking quality with respect to taste and flavor. Resin content (in percent of dry tobacco) decreased from 7.13 in the control sample to 6.34, 6.57, and 6.43 after treatments I, II, and III, respectively. Simultaneously, resene ester content decreased and resin acids increased with a positive effect on flavor. After a 6-month storage, insoluble protein increased significantly in artificially dried leaves. Also, the differences between the three methods with respect to various amino acid contents of the processed tobacco leaves decreased to some extent after a 6-month storage. However, the content of many amino acids remained high in leaves treated at a high temperature. Smoking characteristics of tobacco can be significantly improved by storage after drying and curing.

80–1259. Buisson, J. **Smokable Tobacco Fiber-Material.** United States Patent No. 4,196,739, April 8, 1980, 4 pp.

A smokable material is described which is formed from coherent spheroidal aggregates of entangled tobacco fibers without any preferential directions. The new material is obtained particularly well from some elements of the tobacco plant, but can be obtained from similar elements of other plants which are smokable. Among the advantages the new smoking material offers as compared to known materials are: (1) it is more economical since it uses elements from the tobacco plant with little commercial value (leaf veins, ribs, and stalks); and (2) the material presents a much reduced risk for the smoker's health: notably, the tar content of the smoke from such articles is particularly low.

80–1260. Cremaschi, D. **Il Tenore di Nicotina nei Prodotti Curati e Fermentati di Tabacco Nostrano del Brenta. [Nicotine Content in Cured and Fermented Leaves of Nostrano del Brenta Tobacco.]** Stazione Sperimentale Tabacco Annuari 1, 1973-1974, pp. 129-143, Italian.

The nicotine level in tobacco leaves varies according to several agronomic and technological factors. For the purpose of reducing the nicotine content, a study of the relationship between the number of leaves per plant, fermentation technology, and alkaloid content was undertaken. Leaves of nontopped and topped Nostrano del Brenta tobacco plants at 6, 8, 10, and 12 leaves were utilized. Nostrano del Brenta is a traditional Italian tobacco which is topped in order to obtain particular characteristics, but its utilization by foreign manufacturers is difficult due to its high nicotine content. Yield, leaf surface, and filling value were determined; in addition, the cured leaves were submitted to bulk fermentation with four and six turnings. Results of 2 years indicated no statistically significant differences in yield between 12, 10, and 8 leaves per plant. Nicotine content (expressed as percent) was as follows: 2.50 for cured leaves of plants topped to 6 leaves, 0.96 for nontopped plants, and 1.73 and 1.96 for plants topped to 12 and 10 leaves, respectively. Comparing cured leaves and those fermented five and seven times, a statistically significant nicotine decrease (approximately 11 percent) was observed only after seven fermentations. The most effective topping was at 10 useful leaves, utilizing some superior leaves to the detriment of ground leaves. A greater number of fermentation processes tended to lower the nicotine content of the leaves. (Auth. Abs.)

80–1261. De Jong, D. W.; Lam, J. J., Jr. **Application of Homogenized Leaf Curing to Protein Recovery and to the**

Alteration of Leaf Chemistry for Production of Less Hazardous Tobacco. *Tobacco Research* 5(1): 1-27, June 1979.

A method is described for processing freshly harvested tobacco leaves that involves crushing the leaves and curing by subsequent incubation in slurry form followed by drying. The system, termed homogenized leaf curing (HLC), provides opportunities for protein removal as a byproduct as well as for changing the chemical composition of the tobacco. Protein was recovered from the slurry by separation of solid and liquid phases with pressure filtration, followed by successive precipitation of colloidal chlorophyll and soluble protein fractions with heat. Deproteinized juice, chlorophyll, and fibers were recombined to make the final dried tobacco product. Several alternative HLC production strategies including once-over and whole plant harvest are discussed in terms of altered tobacco chemistry. Extractable protein levels in tobacco leaves attain a maximum of 16 percent by dry weight in fully expanded midstalk leaves. Some differences among varieties were noted with respect to rate of protein decline during senescence and to the purity of protein in the precipitates. Sugar, starch, total nitrogen, and total alkaloid concentrations were reduced as a result of deproteinization. A reduction in both lipid- and water-soluble components was observed with a corresponding increase in the proportions of cell wall- associated material (including pectic substances as well as lignin and celluloses). The HLC process offers promise for dealing with the smoking and health issue by furnishing a means for decreasing chemical components known to be toxic or carcinogenic while at the same time yielding a useful foodstuff-leaf protein. (Auth. Abs. Mod.)

80–1262. Deletang, J.; Albo, J.-P. **Mise au Point d'une Methode de Fabrication a Partir de Brins Courts de Tabac, de Cigarettes Destinees a la Determination des Taux de Condensats de Fumee. [Method of Cigarette Manufacturing, Using Short Tobacco Shreds to Determine Smoke Condensate Rates.]** *Annales du Tabac* 13(2): 170-180, 1976, French.

A simple, efficient method is described for manufacturing cigarettes from short tobacco shreds using small (about 100 g) tobacco samples, and for determining dry smoke concentrate rates. The dimensions (i.e., 0.4-0.8 mm) of tobacco shreds were selected so that cigarette and cigarette smoke condensate weight were comparable to those obtainable with cigarettes manufactured with cut tobacco. The comparative smoking tests show that the anhydrous condensate yields obtained by the above method are 2 to 3 mg higher than those obtained with the commercially produced cigarettes.

80–1263. Gopalam, A.; Gopalachari, N. C. **Biochemical Changes in Leaf Pigments and Chemical Constituents During Flue-Curing of Tobacco.** *Tobacco Research* 5(2): 117-124, December 1979.

Changes in leaf pigments and other chemical constituents were studied in the two varieties Kanakaprabha and CTRI Special during different stages of curing, i.e., beginning of curing, end of yellowing, end of color fixing, and end of curing in the bottom, middle, and top primings. There was little variation in the concentration of chlorophylls *a* and *b*, carotenes and xanthophylls between the two varieties at the different stages of curing. From the beginning of curing to the end of yellowing, the depletion of all the pigments took place at a faster rate than at later curing stages. The content of starch, total sugars, and reducing sugars was higher in the leaves of Kanakaprabha than those of CTRI Special, whereas sucrose showed a reverse trend. A 20-fold increase in the concentration of sucrose was observed at the termination of the curing process. Total nitrogen and protein nitrogen varied little among the varieties. Nicotine in Kanakaprabha leaves was significantly higher than that of CTRI Special (0.64 vs. 0.31 g per 100 g of initial green lamina, respectively; $p<0.05$). Both of the nitrogen fractions decreased during the curing process. Kanakaprabha leaves had higher amounts of chlorogenic acid (13.55 mg/g dry weight) and rutin (10.70 mg/g dry weight) than those of CTRI Special. Rutin decreased steadily during curing in both the varieties, whereas chlorogenic acid remained constant during curing. (Auth. Abs. Mod.)

80–1264. Maeda, K.; Kondo, F.; Tokida, A.; Noguchi, K. **Fuiruta—Tabako no Hinshitsu ni Oyobosu Makigami to Chippu no Kaiko Koka (dai shi ho) Gasu Joki Sochu Shuyo Seibun no Teigen ni Tsuite. [Effect of the Degree of Perforation of Cigarette Paper and Tipping Paper on the Performance of Filter Cigarettes. (IV). On the Reduction of the Main Vapor Components in Cigarette Smoke.]** Scientific Papers of the Central Research Institute, Japan Tobacco and Salt Public Corporation No. 120, 1978, pp. 51-60, Japanese.

The effects of some parameters on the reduction of acetaldehyde, isoprene, and acetone delivery were investigated, and the results are shown as follows: (1) as the air permeability of cigarette paper increases, the reduction of the main vapor components in cigarette smoke increases linearly, (2) the ventilation rate through cigarette paper is more effective in the reduction of vapor components than in the reduction of nicotine and tar, (3) as the ventilation rate through tipping increases, the reduction of vapor components increases linearly, (4) the ventilation rate through tipping is more effective than through cigarette paper on the reduction of vapor components, and (5) the effect of the combination of perforated cigarette paper and ventilated tipping paper on the reduction of vapor components can be calculated. Perforations on the cigarette paper and tipping paper tended to reduce cigarette smoke constituents in the following order: acetaldehyde $>$ isoprene $\geq$ acetone $>$ total particulate matter $\geq$ tar $>$ nicotine. (Auth. Abs. Mod.)

80–1265. Takemoto, N.; Sakurai, T.; Mashiko, K.; Tomari, H. **Method of Processing Smoking Composition.** United States Patent No. 4,186,756, February 5, 1980, 8 pp.

A method of processing a smoking composition to improve its water resistance is described. The smoking composition can be reconstituted tobacco or a tobacco substitute, and be composed of natural tobacco material and/or organic or inorganic material(s), which are combustible as raw materials, and of sodium carboxymethyl cellulose (CMC-Na) as binder. The composition is heated at 110-240°C, preferably at 150-200°C, for 5-150 min, so as to reduce the moisture content to 1 percent and below. The heat-processed smoking composition exhibits a superior water resistance compared to the smoking composition not heat-processed, while the former exhibits equal physical properties except water resistance and organoleptic properties compared to the latter. (Auth. Abs. Mod.)

80–1266. Tobacco International. **New Method of Leaf Protection Through Insect Growth Regulation.** *Tobacco International* 182(4): 18, 21, February 22, 1980.

The first insect growth regulator to be approved for application to stored tobacco, KABAT Tobacco Protector, was recently registered by the U.S. Environmental Protection Agency. It protects stored tobacco by preventing adult emergence of cigarette beetles and tobacco moths. Unlike conventional pesticides that kill larvae or adults, KABAT controls insect populations by eliminating future generations. The chemical is sprayed onto tobacco after moisture content is adjusted and just before compaction into storage containers. Applied at 10 ppm, adult emergence is prevented for more than 24 months. Since 22 months is the average holding period, the insect growth regulator offers a single treatment approach to protecting stored tobacco. Although KABAT is new to tobacco, the active ingredient, methoprene, has been used commercially since 1974. Radiochemical experiments show that in burning cigarettes, methoprene is largely distilled unchanged into smoke. In simulated smoking tests, approximately 50 percent of the radioactivity appeared in sidestream smoke, 40 percent in mainstream smoke, and 10 percent in the cigarette butt. Of the radioactivity in mainstream smoke, more than 98 percent is unchanged methoprene. In all tests, including exposure of rats to smoke, methoprene showed a wide margin of safety. It did not cause any harmful effects at exaggerated levels of 375 and 10 mg/kg/day administered in the diet of rats and mice, respectively, for their entire life span. By comparison, it is estimated that a heavy smoker (2 packs/day) will be exposed only to 0.00143 mg/kg methoprene/day.

See also, **80–1077**

TOBACCO ECONOMICS

80–1267. American Automatic Merchandiser. **Tobacco Sales and Margins Strengthen.** *American Automatic Merchandiser* 21(12): 23-24, December 1979.

The American Automatic Merchandiser survey among cigarette vendors revealed that surging vend prices are outpacing rising product costs to brighten the current cigarette vending picture. The average price for all cigarettes vended went up to 72.7 cents per pack while the average product cost was 49.2 cents per pack. This product cost is 4.7 cents per pack higher than the previous year average, resulting in an average gross margin of 23.5 cents per pack compared with a margin of 22.1 cents per pack in the previous year. The weekly average sale per machine is 62.30 dollars compared to the previous year average of 57.95 dollars. On a unit pack basis, the weekly sales volume per machine is also up from 84 to 88 packs per machine per week. To maintain pack sales averages, vendors are using demographic cigarette machine stocking methods which monitor consumer preference. Proliferation of cigarette brands or styles has causes a substantial increase from 37 to 46 different brands or styles being vended. Fourteen of these 46 are low-tar cigarettes, and the 21.1 percent of total vended cigarette sales provided by low-tar brands represents the fastest growing category in vendor inventory.

80–1268. Chase, S. **The Birth of a European Cigar.** *Tobacco International* 181(23): 17, 45, 47, November 16, 1979.

The Agio factory in Holland, established in 1904, concentrated on producing cigars for the home market until 1973, when labor costs forced Agio to open a factory in Malta. Production costs analysis enabled Agio to make a cigar the same size as the average German or French whiff at a cheaper price, or a larger cigar at the same price. Further, the outside wrapper could be added by hand to give a high quality image. The name Meharis, registered internationally, and a new pack design helped launch the new cigar in Holland, Germany, and France in 1976. First year sales were 30 percent above target (9 million cigars) and are expected to reach 43 million this year.

80–1269. Hill, D. **Dutch Cigar Sales Decline at Home but Gain Significantly Abroad.** *Tobacco International* 181(23): 12-13, November 16, 1979.

Cigar consumption in the Netherlands fell slightly from 812 million cigars and 272 million cigarillos in 1977 to 784 million cigars and 268 million cigarillos in 1978. A slight decline also took place in total production: from 1,086 million cigars and

1,238 million cigarillos in 1977 to 1,078 million and 1,209 million, respectively, in 1978. Meanwhile, the Netherlands continues its success as an exporter of cigars, selling 2,315 million cigars abroad in 1975, compared to 2,592 million in 1978.

80-1270. Kiger, H. C. **Flue-Cured Exports: Foreign Markets Needed.** *Tobacco Reporter* 107(3): 36-37, March 1980.

Since it appears likely that there will be further declines in the use of U.S. flue-cured tobacco for domestic consumption, any future increase in its production will depend upon the expansion of foreign sales. The United States has not shared in the increased world market for flue-cured tobacco because of its high price and the decline in quality. The problems faced by the U.S. tobacco industry in the sale of flue-cured tobacco in its three major markets (the United Kingdom, West Germany, and Japan) and the outlook for exports during the 1980's are discussed.

80-1271. Maxwell, J. C., Jr. **How the Brands Ranked. Maxwell International Estimates.** *World Tobacco* (65): 67-68, 70-71, 73-74, July 1979.

The market shares of some leading cigarette brands are tabulated for 1976-1978 by country for 36 countries as varied as Argentina, Egypt, West Germany, Israel, Mexico, Norway, Poland, United States, and Zaire. Marlboros led in the United States in 1978 with 16.6 percent of the total market, followed by Winston with 13.9 percent of the total market. In Poland, Sport and Klubowe captured 57.0 and 26.1 percent of the market in 1978. In 1976, the corresponding percentages were 61.8 and 23.7. Cleopatra KS captured 80.8 percent of the market in Egypt in 1978. In Greece in 1978, Karelias Filter, No. 1 Filter, and No. 22 Filter captured 26.3, 15.2, and 10.2 percent of the market, respectively.

80-1272. Tobacco International. **French Growers Are Looking to Virginia.** *Tobacco International* 182(2): 14, January 25, 1980.

Despite a drop in the planted area in 1979, French production of tobacco should amount to 52,200 metric tons (mainly light dark tobacco). Burley tobacco was not planted in 1979. However, new Virginia varieties are being tested in trial plots in the different tobacco-producing regions. Under the pressures of strong promotion for the "blond" tobacco made by Anglo-American companies, a large share of the market has shifted from dark to blond tobacco. French producers want to be able to supply this market with locally grown tobacco. However, commercial production of Virginia tobacco is not likely to begin for at least 2 years, and no large changes in tobacco production are expected in 1980.

80-1273. Tobacco International. **Strong Market Keeps Chewing Very Much Alive at RJR.** *Tobacco International* 182(6): 26, March 21, 1980.

While R. J. Reynolds is best known as one of the largest cigarette manufacturers in the United States, it remains very active in the production and marketing of chewing tobacco for which demand has been increasing. The total chewing tobacco market has shown an annual growth rate of 4.1 percent since 1974, with sales amounting to 102.1 million lbs in 1978. The increase in the popularity of chewing tobacco has been attributed to many factors, including more leisure time, increased outdoor activity, and advertising which promotes chewing as "an individual thing." The typical user is found to be older, somewhat rural, and very traditional in his outlook.

80-1274. Tobacco Reporter. **Spain's Tobacco: A Growing Interest.** *Tobacco Reporter* 107(2): 46, February 1980.

Spain's tobacco production for 1979 is currently estimated at 38,084 metric tons, nearly 28 percent above the revised 1978 output and the largest crop on record since Spain began to produce tobacco in the early 1920's. The record high leaf output in 1979 is attributed to a substantial increase in acreage and to higher yields for most types of tobacco, the result of most favorable growing conditions. The Spanish Government continues to encourage the production of flue-cured, Burley, and cigar leaf, mainly flue-cured, through expanded acreage allotments, increased support prices, and certain forms of assistance, such as free seed and subsidized fertilizer. The government is showing a growing interest in producing larger quantities of tobacco to partially substitute imported leaf. In September 1979, the Ministry of Agriculture submitted a 5-year tobacco expansion program which calls for raising tobacco production to 44,150 metric tons by 1985.

80-1275. Tobacco Tax Council. **The Tax Burden on Tobacco. Historical Compilation.** Tobacco Tax Council, Inc., Volume 14, Richmond, Virginia, 1979, 178 pp.

The history of tobacco taxes from the years of the Civil War to the present is presented in this booklet, the 1979 issue of a report published annually by the Tobacco Tax Council. Prior to the Civil War, tobacco was taxed in various ways, but with the advent of the war, the taxation took the form of excise taxes which have continued to the present. While revenues from cigarettes accounted for less than one-tenth of 1 percent of Federal tobacco excises in 1865, at the close of World War I they were more than 50 percent, a figure which rose to 90 percent at the end of World War II. Today the revenue from cigarettes accounts for more than 98 percent of the total Federal tobacco tax excises, yielding more than 2.3 billion dollars annually. In addition to the Federal Government, all the States and 365 local governments imposed cigarette taxes by 1979, bringing the total tax take to almost 6.2 billion dollars annually. These taxes represent about five times the gross receipts of the tobacco farmer and have doubled the price of cigarettes for consumers. The report is presented in the form of tables divided into two sections: (1) composite figures on Federal and State excise taxes on cigarettes and other tobacco products, and (2) data for each State on the history and current status of the cigarette tax.

80–1276. World Tobacco. **Leaf Exports Pause as Cigarette Trade Booms.** *World Tobacco* (66): 81-83, October 1979.

The export outlook for U.S. flue-cured and Burley leaf tobacco is good despite fears that the current hostility toward the tobacco industry at home may eventually affect sales abroad. Two reasons for optimism are the weakness of the dollar and the changes in European Economic Community (EEC) tariffs which formerly tax-penalized superior U.S. flue-cured and Burley tobacco grades in order to protect Italian-grown fine cigar wrapper. There are reasons for pessimism, however. One is concern over the impact of Zimbabwe-Rhodesian tobacco production on U.S. markets. Another is a residue of discrimination against U.S. leaf tobacco in some EEC countries in the cigarette tax formula. Finished product exports are, however, doing very well. Cigarette and cigar exports are 13 and 18 percent higher in volume than 1978 levels. It is thought that both the American image and the flavor and aroma of U.S. cigarette brands are responsible for international consumption.

GENERAL

80–1277. Abdullaev, R. A. **Pervichnaia Profilaktika v Klinike Vnutrennikh Boleznei. [Primary Prophylaxis in the Clinic for Internal Diseases.]** *Terapevticheskii Arkhiv* 51(1): 9-13, 1979, Russian.

In a general discussion of disease prevention, an extremely important role is attributed to smoking and alcohol abuse in the development of cardiovascular, lung, digestive tract, and central nervous system diseases. The known disease symptoms in smokers are discussed. There are several tens of millions of smokers in the U.S.S.R., and per capita consumption of tobacco has been increasing. Before adulthood, 60 percent of men and 20 percent of women start smoking. Schoolteachers, university professors, parents, and medical personnel should be drawn into the campaign against smoking. Mass media must also be used to a greater extent.

80–1278. American Medical Association. **AMA 1979 Interim Meeting Report. Board of Trustees Report J. No Smoking on Commercial Aircraft (Appendix H).** *Journal of the Tennessee Medical Association* 73(4): 274-276, April 1980.

In January 1979, the Civil Aeronautics Board (CAB) amended its rules on smoking aboard aircraft to require special segregation of cigar and pipe smokers, a no-smoking area for each class of service, and enough seats in no-smoking areas for all who wish to be seated there. The American Medical Association Board of Trustees is recommending new measures to the CAB and Federal Aviation Agency (1) to determine the levels of all cigarette smoke contaminants in smoking and no-smoking areas in commercial aircraft in order to establish the air quality under present and proposed practices, and (2) to ban cigar and pipe smoking on all commercial aircraft, cigarette smoking on flights of 1 hour or less and on planes of fewer than 60 passengers, and to establish special partitions between smoking and no-smoking areas.

80–1279. Barth, W.; Gibel, W.; Krannich, W.; Landmann, H.; Paun, D.; Schwarz, W. **Gesundheit und Rauchen. Informations- und Argumentationmaterial zur Gesundheitserziehung. [Health and Smoking. Information and Discussion Material for Health Education.]** Institute for Health Education, German Democratic Republic, Series 1, No. 3, 1979, 134 pp. German.

This card file publication on smoking and health is designed to support dissemination of knowledge relevant to health education in order to promote social activities regarding nonsmoking. A scientifically based attitude toward smoking and toward reducing smoking-induced health damage must be formed to control cigarette consumption which increased greatly in the German Democratic Republic in the last decade, particularly among the youth. The increase amounted to 47 percent from 1960 to 1977. The negative effect of smoking on health is illustrated by an increase in new cases of malignant bronchi and lung tumors, from 2,488 in men and 326 in women in 1953, to 6,661 and 946, respectively, in 1975. It is estimated that in the German Democratic Republic, over 27,000 men die annually from smoking-related diseases. The higher disease incidence and higher mortality in smokers as compared with nonsmokers have a negative effect on the national economy. Men and women who smoked >5 cigarettes/day have lost 20 and 25 percent more working hours than nonsmokers because of sickness. Smoking control methods, including legal measures, are outlined and new measures are recommended. Health education of children and youth, and methods for smoking withdrawal are mentioned. The file is updated to include data as recent as 1977.

80–1280. Bellanca, J. J. **Asbestos-Related Diseases.** *U.S. Navy Medicine* 70(5): 18-19, May 1979.

Asbestosis, mesothelioma, and pulmonary carcinoma are three asbestos-related diseases. The latter's prevention in workers is most effective with cessation of cigarette smoking and avoidance of airborne asbestos exposure. However, dire warnings about the dangers of smoking and asbestos are seldom effective in getting people to stop smoking. Support and "how to" suggestions lead to more successful interventions. Health organization materials to help people stop smoking are also useful.

80–1281. Blackwell, M. J.; French, J. G.; Stein, H. P. **Adverse Health Effects of Smoking and the Occupational Environment.** *American Industrial Hygiene Association Journal* 40(7): A38, A40, A43, A45-A47, July 1979.

There is increasing evidence of adverse health effects due to the combined actions of tobacco use and exposure to chemical and physical agents in the workplace. The National Institute for Occupational Safety and Health recommends that the use of and/or carrying of tobacco products into the workplace be curtailed in situations where employees may be exposed to physical or chemical substances (e.g., asbestos, chlorine, plastics) which can interact with tobacco products. Additionally, curtailment of the use of tobacco products in the workplace should be accompanied by simultaneous control of worker exposure to physical and chemical agents. These recommendations are based on evidence which indicates that smoking can act in combination with hazardous agents to produce or increase the severity of a wide range of adverse health effects. (Auth. Abs. Mod.)

80–1282. Farm Index. **U.S. Tobacco Policy: Burning at Both Ends?** *Farm Index* 18(10): 10-12, November 1979.

While the Department of Health, Education, and Welfare (HEW) urges smokers to quit, the Agriculture Department (USDA) administers a tobacco price support and production control program for growers. The seeming conflict is dismissed by USDA experts who maintain that the price support program does not encourage tobacco use nor would its elimination greatly affect smoking except to possibly increase consumption by decreasing the price of cigarettes. The experts contend that as long as tobacco use is legal, the price support program makes economic sense. It limits production, enables the farmer to get higher prices, and otherwise contributes to the American economy through taxation, employment, and exports.

80–1283. Gart, J. J.; Schneiderman, M. A.; Gori, G. B. **"Low Risk" Cigarettes: The Debate Continues. (Letters).** *Science* 204(4394): 688, 690-692, May 18, 1979.

These letters pertain to the ongoing controversy over the less hazardous cigarette (*Journal of the American Medical Association* 240(12): 1255-1259, September 15, 1978). The authors reiterate a previously stated position on both the erroneous statistical foundation and the misleading table of "critical levels" for low tar and nicotine cigarettes. The primary focus of this letter is on the statistical methodology used to calculate relative risks. Several pertinent examples are given. A reply from the author of the original article follows. See also 79-0369.

80–1284. Hospitals. **Cancer Society Cites 9 Hospitals for Smoking Clinics.** *Hospitals* 53(5): 19-20, March 1, 1979.

Special citations have been awarded to nine U.S. hospitals by the American Cancer Society for their leadership in developing hospital-based smoking cessation programs. The hospitals had participated in a demonstration project which resulted in a guide to aid hospitals in the development of quit-smoking clinics.

80–1285. Journal of the National Medical Association. **Smoking and the Workplace—A National Survey.** *Journal of the National Medical Association* 72(5): 527-528, May 1980.

A recent nationwide survey of 3,000 U.S. companies conducted under the auspices of the National Interagency Council on Smoking and Health revealed that approximately 15 percent of U.S. businesses have programs to encourage and assist their employees to quit smoking. A significant number of the responding companies have policies that restrict or prohibit smoking in the workplace. In addition, one-third of the respondents indicated an interest in developing or expanding smoking and health programs for their employees.

80–1286. Kornegay, H. R. **Chances for Tobacco in the Coming Decade.** *Tobacco International* 182(6): 36, 39, 43, March 21, 1980.

Despite the temporary ebb in flue-cured tobacco exports in 1979, the industry outlook is positive and optimistic. One reason is the worldwide popularity of American tobacco. Another is that despite the mounting antismoking barrage, the number of smokers from 1972 to 1978 rose approximately 7 percent, from 49 to 52 million smokers. During this period, individual consumption rose 1 percent. The antismoking message is also apparently without influence in Congress where some 20 antismoking bills have not moved beyond the introduction stage. Several leading antagonists are no longer in Congress. Most important, however, is that the indictment of tobacco as a cancer-causing agent is based on data manipulation to reach conclusions not supported by all of the data. The suggestion is made that the President could help the economy and reduce the budget deficit by cutting the 52 million dollars devoted to smoking and health programs as the bulk of these funds go toward antismoking propaganda and agitation.

80-1287. Lancet. **Unforeseen Attack on Tobacco Industry.** *Lancet* 1(8131): 1415, June 30, 1979.

In an address to the Fourth World Conference on Smoking and Health by the British Parliamentary Under-Secretary for Health, it was stated that the solution to many of today's medical problems would not be found in hospital research laboratories but in Parliament. Further, the costs of illness caused by smoking are not borne by the individual but by taxpayers as a whole through the National Health Service. Government was urged to use all the weapons at its disposal (e.g., advertising controls, pricing, health education, and persuasion) to reduce, and eventually to eliminate, the diseases caused by smoking. The speech delighted antismoking campaigners and gave little comfort to the tobacco industry.

80-1288. Lee, P. N. **Carbon Monoxide Yield of Cigarettes. (Letter).** *British Medical Journal* 2(6204): 1584, December 15, 1979.

In reply to a suggestion that carbon monoxide (CO) yields of individual cigarette brands be published (*British Medical Journal* 2(6192): 731, September 22, 1979), the author offers three rebuttals. First, it is stated that there is presently no evidence of harmful CO exposure effects in animals and birds fed normal diets. Second, CO yields in U.K. and U.S. cigarette brands appear to have declined. Third, the 1973 publication of cigarette tar levels did not stimulate production of lower tar level cigarettes. See also 80-0013.

80-1289. Longest, W. G., Jr. **That's Great Tobacco!—But Will It Smoke?** *Tobacco International* 181(2): 7, 9-10, January 26, 1979.

Recent changes in cigarettes (e.g., filters, ventilation, flavorants, chemical constituents) have not necessitated great changes in the basic raw material—tobacco. Dramatic changes in varieties and cultural practices are not necessary to produce a good smoking low-tar and low-nicotine cigarette. Flavorful tobaccos are important. Resources of manpower and scientific expertise must be deployed to continue to identify every component of cigarette smoke and to isolate smokability factors. While increased criticism of Federal funding of tobacco production research is expected under the present sociopolitical climate, it is realistic to expect continued public funding because the benefits derived by the public outweigh the cost of research.

80-1290. Masironi, R. **Controlling the Smoking Epidemic: Summary of a New Report by a WHO Expert Committee.** *World Smoking and Health* 4(3): 4-8, Autumn 1979.

A World Health Organization Expert Committee considers smoking an international epidemic with resultant harmful health effects (e.g., respiratory diseases, lung cancer, myocardial infarct, prenatal death) in both smokers and passive smokers. Among suggested strategies are crop substitution, sales restrictions, health warnings, smoking restrictions, and cessation.

80-1291. McGill, A. M. (Editor). **Proceedings of the National Conference on Health Promotion Programs in Occupational Settings, January 17-19, 1979.** U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, 1979, 84 pp.

In these proceedings, issues and approaches addressed at the National Conference on Health Promotion Programs in Occupational Settings are discussed. The escalating cost of health benefits has been a major incentive for industry to become involved in preventive health care. Additional benefits of such a program may include reduced absenteeism, increased productivity, and increased ability to cope with problems on and off the job. In the principal chapters of the proceedings, the background and purpose of the Conference, the seven health promotion components (including smoking cessation), and health promotion program issues are discussed. In view of the increased risk to smokers exposed to certain occupational hazards, an increasing number of companies are expressing interest in providing smoking cessation programs for employees. Current research on smoking cessation methods reveals a number of promising directions including aversive smoking approaches combined with self-control strategies. Some smoking cessation programs at the worksite emphasize physician counseling, but relatively greater interest is shown in the use of consultants or contingency programs to encourage nonsmoking.

80-1292. Miller, G. H. **The Filter Cigarette Controversy.** *Journal of the Indiana State Medical Association* 72(12): 903-905, December 1979.

While it is widely accepted that filter cigarettes are safer than nonfilter cigarettes, there are some deficiencies (e.g., wide confidence limits, narrow smoking history limits, combined smoking categories, inappropriate animal model methodologies) in the data generated by research devoted to verifying this hypothesis. Further, experimental evidence indicates that carbon monoxide in cigarette smoke is the major cause for the higher incidence of cardiovascular diseases. One study revealed that the seven most popular brands of filter cigarettes provided smokers 28 percent more carbon monoxide than the seven most popular nonfilter cigarettes. The same study reported that people smoking filter cigarettes had higher carboxyhemoglobin levels than those smoking nonfilter cigarettes. Another study showed that women smokers 35-44 years of age were contracting lung cancer at higher rates than men even though more women smoked filter cigarettes. It is stated that filter vs. nonfilter

best copy available

cigarettes are not really "safe", and that carbon monoxide must be recognized as a dangerous compound.

80-1293. Palmer, C. A. **Dentists and Smoking—Surgeon General's Report Looks at the Impact on Patients.** *Journal of the American Dental Association* 99(2): 415-416, August 1979.

Health care professionals have been perceived to have an impact on the smoking habits of the public and their patients. In one study, 20 percent of the subjects thought that dental associations should be more actively involved in smoking education efforts. However, dentists are less likely than other professionals to believe that they have responsibilities as smoking educators. Surveys in 1967 and 1970 found that about two-thirds of physicians, but only one-third of dentists, routinely asked about patients' smoking histories. Among dentists in 1967, three of four said that they warned leukoplakia patients about smoking, but only 36 percent gave this warning to patients with soft tissue lesions. The latest Surgeon General's report analyzed health professional roles in affecting the public's smoking habits and concluded that dentists and other health professionals have preceded the general public in improving their smoking habits. However, future dentists and physicians must be educated on the health hazards of smoking and their responsibilities as health educators.

80-1294. Rawbone, R. G. **Self-Titration by Cigarette Smokers. (Letter).** *British Medical Journal* 2(6192): 731-732, September 22, 1979.

Self-titration by smokers has been reported when they switch from a middle-tar to either a high-tar or a low-tar cigarette (*British Medical Journal* 2(6186): 357-360, August 11, 1979). As the respondent points out, short-term (2 week) switching studies indicate that smokers attempt to compensate when switching to a lower tar cigarette, but that on balance switching is beneficial. A study of subjects who had smoked either middle-tar or low-tar cigarettes for at least 2 years indicated, that compensation does not persist and that long-term benefits from switching to low-tar products are greater than short-term switching studies suggest. See also 79-1394.

80-1295. Richmond, J. B. **Smoking and Health.** *Public Health Reports* 94(2): [inside cover], March-April 1979.

The new "Smoking and Health. A Report of the Surgeon General" provides overwhelming evidence connecting cigarette smoking with disabling and fatal diseases. It reflects the results of 30,000 studies undertaken since the original report was issued in 1964. These studies clearly illustrate that cigarette smoking is far more dangerous to health than was believed 15 years ago. The new report summarizes current knowledge about many aspects of smoking, including health consequences, behavioral aspects, and educational and preventive efforts.

80-1296. Robillard, D. **Politique de Lutte Contre le Tabagisme. [Smoking Control Policy.]** *Canadian Medical Association Journal* 120(9): 1168-1169, May 5, 1979, French.

A document issued by the Quebec Health Planning Service of the Ministry of Social Affairs cites statistics on smoking and health and states the basic principles of an antismoking policy. Nonsmokers account for <13 percent of all deaths from bronchial and lung cancer. The risk of ischemic heart disease in the smokers aged 35 to 44 years is 6.24 times greater than in nonsmokers. Emphysema affects 99.7 percent of smokers of more than 20 cigarettes/day vs. 10 percent of nonsmokers. The greatest damage is inflicted upon the fetus of a smoking mother. Babies of mothers who smoke 1-20 and >20 cigarettes/day weigh an average 215 and 295 g, respectively, less than those of nonsmoking mothers. According to the plan of the Ministry of Social Affairs, most of the objectives of the antismoking policy should be met by January 1, 1982. Health education of the target groups of population will be funded by a 12 percent increase in cigarette pricing in addition to increases due to inflation. The average tar and nicotine contents of cigarette smoke are expected to be reduced to 5 and 3 mg, respectively, by the target date. All advertisement of tobacco products will be prohibited in Quebec starting from that date. Provincial legislation will be introduced to prohibit or restrict tobacco consumption in specified public places.

80-1297. Schmidt, F. **Zwangsrauchen am Arbeitsplatz—ein Gegengutachten. [Passive Smoking at Work—A Critical Opinion.]** *Kassenarzt* 19(34): 3060-3076, 1979, German.

A recently published legal opinion in favor of smoking at work is sharply criticized. The discussion of smoking at work almost exclusively in terms of individual freedom is found particularly objectionable, as it overlooks the importance of the public's health. German statistics of 140,000 premature deaths and 100,000 premature disability cases per year are cited to support the view that smoking should be regarded as socially detrimental behavior, or even as suicide. Data are presented to show that smoking is the single most important cause of injury at the workplace. Prohibition of smoking in all workplaces is urged based on numerous studies about the health risks of passive smoking.

80-1298. Seideman, N. **The New England Deaconess Hospital: Nearly Smoke Free.** *World Smoking and Health* 4(3): 18-21, Autumn 1979.

The New England Deaconess Hospital in Boston has established a strong antismoking policy. Patients are not allowed to smoke without their physician's written permission, staff members' and employees' smoking has been restricted to departmental lounges and one section of the cafeteria, and visitors have been permitted to smoke in only one section of the lobby. No cigarettes are sold in the hospital. A special Smoking Policy Committee oversees the program. With fewer than 5 percent of 7,200 hospitals in the United States involved in the antismoking effort, the Deaconess is creating an effective model for other hospitals.

80–1299. Stanulovic, D. **Pusenje i Zdravlje (Usmerena Diskusija). [Smoking and Health (Round-Table Discussion).]** *Medicinski Pregled* 31(9/10): 389-402, 1978, Serbo-Croatian.

Data on smoking and health, and smoking control are briefly presented by a panel of 12 from the Novi Sad Medical Faculty. The presentations are based on the results of clinical observations and epidemiologic studies within the framework of the World Health Organization. Statistical data from several studies were reported, including one of cement factory workers of whom 90.5 percent of the men and 9.3 percent of the women smoked. The study data confirmed that respiratory symptoms and chronic bronchopneumopathy incidence increased with cigarette consumption. A study conducted in Zagreb indicated an 8:1 ratio of smokers to nonsmokers with bronchial carcinoma. Nearly the same ratio (8.5:1.7) was found in the Vojvodina region. Other panel participants reported on physiologic, toxicologic, neuropsychiatric, cardiovascular, and digestive tract effects of smoking. Passive smoking during pregnancy and in children was also discussed. A preventive medicine specialist reported the existence of a consultation clinic for smoking withdrawal using the 5-day group plan of the United States.

80–1300. Tobacco International. **British Study Defends Cigarette Advertising.** *Tobacco International* 182(4): 40, February 22, 1980.

A recent study commissioned by Imperial Tobacco and Gallaher found no evidence that the level of cigarette advertising had any measurable effect on the total consumption of cigarettes in Great Britain during the 1958-1978 period. The report is being used by two British tobacco product manufacturers in their efforts to show that banning cigarette advertising would not be an effective way of reducing cigarette consumption. The current 3-year agreement on advertising between manufacturers and the government will expire shortly and a new agreement is under negotiation.

80–1301. Tobacco Reporter. **U.K. Tobacco: A Tough Year Ahead.** *Tobacco Reporter* 107(2): 42-43, February 1980.

In 1980, it will be increasingly difficult for the tobacco industry in the United Kingdom. The Department of Health and Social Security will be asking Buckingham Palace to withdraw its royal warrants from tobacco companies. A warrant allows various cigarette (e.g., Benson and Hedges) manufacturers to emboss their packs with both the Royal Crest and the legend, "By appointment to Her Majesty the Queen." The Department will also seek to widen the voluntary agreement between the government and the tobacco industry on regulating advertising. There are also reports that a pack of cigarettes will go up 11-22 cents as part of indirect taxation in order to finance promised tax cuts.

80–1302. Turner, J.A.M.; Sillett, P. W.; McNicol, M. W. **The Inhaling Habits of Pipe and Cigar Smokers.** *World Smoking and Health* 4(3): 35-37, Autumn 1979.

Seven pipe and cigar smokers' (aged 30-61) inhaling habits were studied. All subjects had stopped cigarette smoking 4-18 years prior to the study. A second group of seven primary pipe and cigar smokers (persons who started smoking cigars or pipes and have never smoked cigarettes) was also studied. Results indicate primary pipe and cigar smokers do not inhale or absorb significant quantities of nicotine, and all the carboxyhemoglobin (COHb) levels in this group before, during, and after smoking were within the range for nonsmokers. The small rises in COHb and plasma nicotine are similar to those found in passive smokers. The results also showed that secondary pipe and cigar smokers (persons who initially smoked cigarettes) do not lose their inhaling habit, even after having stopped smoking cigarettes for up to 18 years, and that they continue to inhale pipe and cigar smoke, absorbing large amounts of carbon monoxide and nicotine. (Auth. Abs. Mod.)

80–1303. U.S. Department of Health, Education, and Welfare. **Smoking and Health. An Annotated Bibliography: School Edition.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, DHEW Publication No. (NIH) 79-1908, March 1979, 41 pp.

This school edition is a selected, annotated compilation of 125 items for use in the primary and secondary school classroom. Its purpose is to provide educators with sources of teaching aids for smoking education. The aids encompass pamphlets, films, games, and other educational materials.

80–1304. U.S. Department of Health, Education, and Welfare. **Healthy People. The Surgeon General's Report on Health Promotion and Disease Prevention. Background Papers.** U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health and Surgeon General, DHEW (PHS) Publication No. 79-55071A, 1979, 492 pp.

This collection of papers was prepared in response to a request from the Office of the U.S. Surgeon General for a review of the state-of-the-art in health promotion and disease prevention in the United States. Among the topics discussed are prevention of cancer and cardiovascular diseases; infant and child health; health promotion for the elderly; improving the quality of the work environment; health education; and tobacco, alcohol, and drug abuse. The factors and processes that may be involved in the onset of smoking are analyzed, and some promising preventive strategies are suggested. The importance of cessation of smoking in reducing the incidence of cancer and cardiovascular diseases is emphasized.

80–1305. U.S. Department of Health, Education, and Welfare. **Healthy People. The Surgeon General's Report on Health Promotion and Disease Prevention.** U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health and Surgeon General. DHEW (PHS) Publication No. 79-55071, 1979, 188 pp.

This document is the first Surgeon General's Report on Health Promotion and Disease Prevention. It represents an emerging consensus among scientists and the health community that the Nation's health strategy must be dramatically recast to emphasize the prevention of disease. The thesis of the report is that further improvements in the health of the American people can and will be achieved—not alone through increased medical care and greater health expenditures—but through a renewed national commitment to efforts designed to prevent disease and to promote health. Among the topics discussed are risks to good health, health goals for various age groups, and actions for health services, protection, and promotion. Cigarette smoking is identified as the largest single preventable cause of illness and premature death in the United States. Tobacco is associated with an estimated 320,000 premature deaths a year; another 10 million Americans suffer from chronic diseases caused by smoking. The problem of educating the public about the risk of smoking and smoking cessation is discussed.

80–1306. United States Tobacco Journal. **FTC Wants Antismoking Ads and Rotating Caution Notices.** *United States Tobacco Journal* 208(1): 1, 17, January 10, 1980.

The Federal Trade Commission (FTC) wants Congress to appropriate money for a mass media public information campaign "on the health and other consequences of smoking." The agency also wants Congress to require rotating health warnings on cigarette packages and in cigarette advertising such as are used in Sweden. Both recommendations were included in the FTC's report on smoking, which it is required to file each year with Congress. Though no amount of funds was requested for the antismoking ads and commercials, the very suggestion of such a campaign resulted in considerable protests from cigarette distributors.

80–1307. Wennen, E. **The Smoke Screen in Africa.** *World Smoking and Health* 4(3): 9-13, 34, Autumn 1979.

Early signs of a future epidemic of smoking in the Third World are reflected in the annual cigarette production figures from developing countries. Only 11 of 45 Third World countries had imposed restrictions on cigarette marketing by 1974. To counter the tobacco advertising explosion in the developing countries, two recommendations are made: (1) exposure of the collaboration of the tobacco industry with governments, and (2) counteradvertising campaigns financed by 10 percent of tobacco tax revenues.

80–1308. WHO Chronicle. **Smoking: Health and Socioeconomic Implications.** *WHO Chronicle* 32(12): 486, December 1978.

A meeting of the WHO Expert Committee on Smoking Control in October 1978 considered new evidence of the harm done by smoking, particularly to women and children. With the introduction of control measures in industrialized countries, tobacco companies are making an effort to conquer new markets among women and in the developing countries. The Expert Committee proposed a number of control measures, including legislation, higher taxation, international cooperation, and health education of the community.

80–1309. Wiley, J. A.; Camacho, T. C. **Life-Style and Future Health: Evidence From the Alameda County Study.** *Preventive Medicine* 9(1): 1-21, January 1980.

Certain aspects of daily lifestyle were predictive of future health status among survivors in a 9-year longitudinal study. In a sample of 3,892 white adults under the age of 70, cigarette smoking, alcohol consumption, physical exercise, hours of sleep/night, and weight in relation to height are significantly associated with overall health outcomes 9 years later, controlling for initial level of health. As expected, those who were nonsmokers in 1965 have higher adjusted health scores than those who were smokers at that time. In addition, the health scores vary according to quantity smoked in 1965. The average score of nonsmokers (i.e., former smokers and persons who have never smoked) is 0.21; average scores of smokers range from 0.10 (≤ 0.5 packs/day) to -0.41 (≥ 1.5 packs/day). The strongest association between smoking and health status occurs in the male sample. Among women in our sample, there are some inconsistencies in the relationship of smoking to consequent health and the relationship is not statistically significant; it is, however, generally in the same direction as that observed in the male sample. Also, the higher scores of former light-to-moderate smokers indicate greater improvement relative to initial level of health rather than a higher absolute level of health in 1974. (Auth. Abs. Mod.)

80–1310. Williams, R. **De Sanitate Urbanorum.** *British Medical Journal* 2(6205): 1617-1620, 1622, December 22-29, 1979.

The development of mechanical transportation in the last century has detrimentally affected public health in the cities, particularly that of cyclists. Carbon monoxide and lead contaminate the air which is breathed. Two packs (40 cigarettes)/day may produce a carbon monoxide saturation in blood of 14 percent; however, levels in nonsmoking taxi drivers, motorists, or stationary traffic policemen at busy intersections range from 0.8 to 3.0 percent. The author, himself a cyclist, had carboxyhemoglobin measurements made after a 10-mile trip and the level was 0.5 percent. His blood lead levels (0.8 μmol/l) were considered average for a central London cyclist of 2,600 miles/year. The bicycle is extolled as a clean, efficient mode of transportation.

80-1311. World Medicine. **Watching the BBC Can Cause Cancer.** *World Medicine* 15(6): 3, December 15, 1979.

The British Broadcasting Corporation (BBC) is criticized for permitting indirect advertising of cigarettes by televising sports events sponsored by cigarette manufacturers. It is claimed that the corporation is hypocritical as it will not show more European football matches because too many foreign teams have advertisements on their shirts. The editorial maintains that given the medical evidence on smoking, the BBC, through its telecasting of tobacco company sponsored sports events, is an accessory to murder.

80-1312. Yeargin, B. **The Anti-Smoking Campaign Failed, But Did Some Damage.** *Tobacco International* 182(6): 47-49, March 21, 1980.

The antismoking efforts of former Secretary Califano and his Department of Health, Education, and Welfare between 1978 and 1980 are discussed and their adverse impact on the tobacco industry described. From 1978 to 1979, there was a 41 percent increase in the total number of tobacco-related bills filed on a State-by-State basis, including a sizable increase in smoking restriction legislation, and a 70 percent increase in the number of bills filed in categories other than smoking restriction (taxation, etc.). A total of 135 legislative actions were reported on the local level across the country in 1979. There have also been increasing efforts to ban free samples of cigarettes. No decrease in the volume of antitobacco bills is expected for 1980. The tobacco industry's position of allowing consumers to make up their own minds about the pleasures they seek and to pursue their own personal happiness (as long as it is legal) is stressed.

AGRICULTURE

80–1313. Aycock, M. K., Jr.; McKee, C. G. **Response of Maryland Tobacco Cultivars to Various Rates of Nitrogen Fertilization.** *Tobacco International* 181(19): 67-71, September 21, 1979.

A 3-year study was conducted to determine the response of Maryland tobacco to increased nitrogen rates, and to determine if presently grown cultivars exhibited a differential response to varying applications of nitrogen. Four nitrogen rates (89.6, 112.0, 134.4, and 156.8 kg/hectare (ha)) and six cultivars (Md. 10, Md. 59, Md. 609, Md. 64, Md. 872, and Md. 201) were used. In general, as nitrogen rates increased, there was an increase in yield, total alkaloids, and total nitrogen; and a decrease in duration of burn and weather fleck ratings. Varying nitrogen rates had no effect on price, quality index, days to flower, or filling capacity. The nitrogen rate x cultivar interaction was not significant for any of the nine traits studied; however, a trend toward reduced yields at the high nitrogen rate was observed for Md. 872. A rapid decrease in duration of burn was observed as nitrogen rates increased from 112.0 to 134.4 kg/ha for Md. 59, Md. 609, and Md. 64. The results from this study suggest that increased nitrogen rates would be beneficial for increasing the yield of most cultivars. However, the increase in total alkaloids and total nitrogen levels and decrease in duration of burn may be considered a disadvantage with high nitrogen rates and caution should be exercised with high rates of nitrogen fertilization. (Auth. Abs.)

80–1314. Chakraborty, M. K.; Prabhu, S. R.; Suryanarayana, T. K. **Effect of Ethrel on Ripening, Curing and Physicochemical Characteristics of Flue-Cured Tobacco.** *Indian Journal of Agricultural Science* 49(7): 520-525, July 1979.

Ethrel, a newly marketed growth regulator containing 2-chloroethyl phosphonic acid as active ingredient, was applied to the upper primings of flue-cured tobacco plants (*Nicotiana tabacum* Linn.) as a fine spray 3 days after the harvest of lower primings. The spray caused the yellowing of all the remaining leaves. The use of ethrel reduced the number of primings and curing time. At higher doses of ethrel the yield was reduced, but at a mild dose of 90 mg/plant the yield was not affected, nor was there any effect on grade output. Ethrel treatment significantly increased the amount of reducing sugar in the leaf without changing other physicochemical characteristics. It did not influence the nitrogen level and topping effect. (Auth. Abs. Mod.)

80–1315. Gaines, T. P.; Stephenson, M. G.; Gooden, D. T. **Effects of Nutrient Deficiencies on Chemical Constituents of Flue-Cured Tobacco.** *Tobacco Science* 20: 95-97, 1976.

The effects of nutrient deficiencies on tobacco chemical constituents were studied using hydroponically-grown tobacco plants in the greenhouse. Healthy plants were transferred to deficient nutrient solutions when about 40 cm tall and allowed to develop to full flower maturity of the control to simulate field-grown plants developing nutrient deficiencies. The effects of treatments deficient in boron, chlorine, iron, calcium, potassium, sulfur, zinc, magnesium, phosphorus, and nitrogen on total alkaloid, nitrogen, reducing sugar, and starch contents of tobacco were evaluated. A negative correlation was found between total nitrogen (N) and reducing sugars ($p=0.02$) and between total N and starch ($p=0.07$). (Auth. Abs.)

80–1316. Patel, G. J.; Desai, M. V.; Shah, H. M.; Patel, D. J. **Effect of DD and DBCP on Seed Germination of Some Cereals and Tobacco.** *Tobacco Research* 5(2): 143-146, December 1979.

The minimum time lag necessary between application of DD and DBCP nematicides and the crop seeding for tobacco (*Nicotiana tabacum* L.) was determined. Earthen pots (20 cm top diameter) were filled with steamed soils. The nematicide DBCP (1 cc/pot) was applied in the form of Nemagon 60 percent emulsifiable concentrate along with water while the nematicide DD was applied (5 cc/pot) through a hole made in the center of the pot. Each pot was seeded with 50 mg bidi variety Anand 2. Both nematicides significantly reduced the germination when applied immediately before seeding, there being complete inhibition in the case of DD application. With time lapse from 1 to 3 weeks, the germination was improved, and it was maximum when seeds were sown 2 weeks after the application of the nematicide.

CHEMISTRY

80–1317. Battard, J.-C.; Chauvette, C. **Caracterisation Rapide des Sucres du Tabac par Chromatographie Liquide Haute Performance. [Rapid Analysis of Tobacco Sugars by High Pressure Liquid Chromatography.]** *Annales du Tabac. Section 1* (16): 6-12, 1978, French.

A modified high pressure liquid chromatographic method is introduced for separation and identification of different tobacco sugars. Sugars are extracted with water, the extract is evaporated in vacuum, the dry residue is then redissolved in water, and the solution is analyzed, using a chromatem-type chromatograph. An auxiliary active carbon column preceded the partition column to protect the latter from deposition of vegetative pigments. The partition column was filled with silica grafted with an amino radical (lichrosorb-NH_2). The mobile phase consisted of a 80/20 acetonitrile/water mixture. Preliminary testing revealed that the optimum flow rate was 1.60 ml/min at 0.60 bar pressure corresponding to a 2.0 partition coefficient for a fructose-glucose solution. The method was applied to an extract of Oriental tobacco to separate fructose, glucose, and maltose. These sugars were also identified in chromatograms of 18 Oriental and Virginia tobacco brands with 10.5-14.2 and 6.3-21.2 percent, respectively, of total reducing sugars. Sucrose was identified only when tobacco contained at least 1 percent sucrose. This was possible with only three Virginia brands which contained 1.8-4.9 percent sucrose. This chromatographic method is faster than most commonly used methods of sugar analysis and can be easily adapted to routine analysis. However, quantitative determination of sugars in tobacco is not yet possible.

80–1318. Behr, D.; Wahlberg, I.; Nishida, T.; Enzell, C. R. **(3*S*,5*R*,8*S*,9ξ)-5,8-Epoxy-6-Megastigmene-3,9-Diol and (3*S*,5*R*,6*R*,7*E*,9ξ)-3,6-Epoxy-7-Megastigmene-5,9-Diol. Two New Nor-Carotenoids of Greek Tobacco.** *Acta Chemica Scandinavica. Series B* 33(10): 701-704, 1979.

Two new C_{13} nor-carotenoids were isolated from Greek tobacco and shown to be (3*S*,5*R*,8*S*,9ξ)-5,8-epoxy-6-megastigmene-3,9-diol and (3*S*,5*R*,6*R*,7*E*,9ξ)-3,6-epoxy-7-megastigmene-5,9-diol by chemical and spectroscopic methods. (-)-Loliolide, (+)-isololiolide, and (3*S*,5*R*,6*S*,7*E*,9ξ)-5,6-epoxy-7-megastigmene-3,9-diol were also obtained. The biogenesis of these compounds is discussed. (Auth. Abs.)

80–1319. Chamberlain, W. J.; Snook, M. E.; Baker, J. L.; Chortyk, O. T. **Gel Permeation Chromatography of Oxygenated Components of Cigarette Smoke Condensate.** *Analytica Chimica Acta* 111(1): 235-241, 1979.

Oxygenated neutral constituents of a tumor-inhibiting fraction of cigarette smoke condensate were separated from interfering phenolic compounds by a combination of silicic acid and gel chromatography. Silicic acid adsorption and Bio-Beads gel chromatography separated aliphatic from aromatic ketones, and gel chromatography on Sephadex LH-20 resolved aromatic ketones from phenols. Identifications were achieved by studying individual fractions by gas chromatography and gas chromatography—mass spectometry. (Auth. Abs.)

80–1320. Crosthwaite, L.; Sheen, S. J.; Burton, H. R. **Alkylating Activity in the Extract and Pyrolyzate of Tobacco Leaves Varying in Genotype and Chemical Treatment.** *Tobacco International* 181(7): 110-112, April 6, 1979.

Alkylating activity in the extract and pyrolyzate of air-cured leaves of 12 tobacco cultivars and lines was determined by a spectrophotometric method employing 4-(4′-nitrobenzyl) pyridine as the nucleophilic reagent. Pyrolysis conditions varied from 350 to 750°C under nitrogen or air atmosphere. Varietal difference in alkylating activity was statistically significant in most cases. Averaged over all varieties, alkylating activity in leaf extract was equivalent to 13.68 μg of 2-iodobutane/g dry weight. The activity was proportionally increased as pyrolysis temperature was elevated and reached a level of 47.94 μg/g. Solvent extraction of Kentucky 1R1 Reference tobaccos resulted in lowering alkylating activity as much as 68 percent. The amount of reduction is a function of solvent polarity. Enrichment of cigarette leaves with potassium chloride, potassium nitrate, glucose, and starch did not alter the activity, whereas casein hydrolysate and chlorophyllin caused a slight increase when pyrolyzed in air. Nicotine enhanced activity up to twofold in the 8 percent enriched tobacco. Results suggest that aromatic nitrogenous compounds are good precursors of alkylating compounds in the pyrolyzate. Genetic control of alkylating activity in tobacco leaf is feasible but much less effective than leaf modification in solvent treatment. (Auth. Abs.)

80–1321. Gori, G. B.; Ellis, R. L. **Reduction of Carbon Monoxide in Cigarette Smoke.** *Preventive Medicine* 8(3): 358-368, 1979.

Recent studies have shown that the mainstream carbon monoxide (CO) yield is a function of the amount of tobacco burned during puffing, the concentration of the pyrolytic precursors, the heating rate of the tobacco during puffing, and the permeability of the wrapper toward outward diffusion of CO. Modification of these parameters may lead to reduced yields of CO. Other studies have shown that the condensation or absorption of the gas on the filter or unburned tobacco column does not occur to any significant degree and is not a variable affecting the CO yield. Using available technology, cigarette yields of less than 10 mg (tar) and 10 ml (CO) have been obtained in several cigarette brands. The National Cancer Institute has prepared and analyzed approximately 94 cigarette variations in four separate studies to define characteristics of less hazardous cigarettes. In these four sets of experimental cigarettes, a standard experimental blend cigarette (SEB) was used for comparative purposes; its composition was based on the 1970 sales-weighted averages of various tobacco types including reconstituted tobacco sheet (RTS). In each experiment, the tobaccos used for preparing the SEB cigarettes were from that year's crop. Those experimental cigarettes with at least a 25 percent reduction in CO delivery, compared with the SEB delivery, on a per-puff basis, are described. Methods should continue to be developed for improving the tobacco, modifying tobacco sheet technology, and developing improved high-permeability paper; improvement of smoke-dilution devices and better fillers should also be encouraged.

80–1322. Ishiguro, S.; Sugawara, S. **Comparisons of Smoke Components from Lamina and Midrib Cigarettes of Flue-Cured Tobacco Leaves by Trimethylsilylation Method.** *Agricultural and Biological Chemistry Journal* 42(2): 407-410, 1978.

A method for direct gas chromatographic determination of tobacco smoke was developed. Tobacco smoke condensate was collected on a glass fiber filter and the components were converted into their trimethylsilyl derivatives and then subjected to glass capillary column gas chromatography. By this method, all tobacco smoke components, including unstable phenolic substances and water-soluble polyhydroxy compounds, were simultaneously determined. Compositional differences between lamina and midrib smoke of flue-cured tobacco leaves were also clarified by the method. The results indicate that there are quantitative differences in nicotine, phenols, levoglucosan, quinic acid γ-lactone, and the other major components between lamina and midrib smoke. (Auth. Abs.)

80–1323. Jenkins, R. W., Jr.; Francis, R. W.; Flachsbart, H.; Stoeber, W. **Chemical Variability of Mainstream Cigarette Smoke as a Function of Aerodynamic Particle Size.** *Journal of Aerosol Science* 10(4): 355-362, 1979.

A new method to measure whether chemical differences exist between the various particle sizes of mainstream (MS) cigarette smoke is described. This method uses the combination of radiolabeled materials and the separation of the labeled smoke aerosol by a spiral centrifuge. The measurement of specific radioactivity of each separated particle size zone was employed. The cigarettes were made up of ^{14}C-labeled Bright, Burley, and Oriental tobaccos, and smoke was generated by a conventional reverse puffing technique. The techniques are chemically independent of the amount of water or the inorganic composition of the particles. It was concluded that (1) smoke particles are chemically different, dependent on their aerodynamic sizes in the 0.1-0.5 μm diameter range; (2) particle analyses in the size range larger than 0.5 μm indicated most probably an influence of coagulation randomizing the chemical composition, and representing the overall average of total particulate mass in mainstream smoke (MS TPM); (3) in general, the distillables are depleted in the particles of sizes below 0.5 μm and there is a positive slope of the specific activities as particle sizes increase; (4) the pyrolytic products appear enriched in the small particle size range below 0.5 μm as evidenced by the increasing specific activities with decreasing size in this region; and (5) a mass balance of the contribution from each major ingredient in the reference cigarettes has been established for all particle sizes in MS smoke from 0.1 to 2 μm, and the percentage contribution to the nonvolatile portion of MS TPM from each component in this cigarette has been derived. (Auth. Abs. Mod.)

80–1324. Mokhnachev, I. G.; Sichinava, R. B.; Sirotenko, A. A. **O Perekhode Nikotina iz Tabaka v Dym. [Transition of Nicotine From Tobacco to Smoke.]** *Izvestiia Vysshikh Uchebnykh Zavedenii Pishchevaia Tekhnologiia* (1): 71-73, 1979, Russian.

Tobacco and tobacco smoke condensate samples for five tobacco varieties were analyzed for nicotine, protein, and carbohydrate contents. During pyrolysis, the nicotine content of the various smoke condensates varied between 33.3 and 47.8 percent for 181 American, 27.8 and 53.2 percent for 155 Samsun, 33.5 and 48.5 percent for 1,519 Ostrolist, 41.6 and 47.4 percent for 83 Peremozhets, and 33.3 and 43.5 percent for 219 Trapezond tobacco varieties. A series of pyrolysis experiments with synthetic samples of nicotine-free tobacco impregnated with neutral nicotine salts of 19 acids were carried out under the same conditions as in the experiments with natural tobacco. Analytical data show that the nature of nicotine salt is a significant factor of nicotine transition into smoke. A significant part of nicotine combined to a strong inorganic acid is decomposed by pyrolysis, while about one-third of nicotine combined to organic acids is carried over into smoke condensate. In the presence of excess acid, nicotine transition is significantly lowered. Only 31 percent of free nicotine is transferred to tobacco smoke from a nicotine-enriched tobacco because of rapid oxidation of most of the free nicotine.

80–1325. Nagaraj, G.; Chakraborty, M. K. **Lipophilic Constituents of *Natu* Tobacco: II. Phenols.** *Tobacco Research* 5(2): 147-148, December 1979.

Natu tobacco was extracted with hexane, methanol, and diethyl ether. Phenols were separated by steam distillation under nitrogen, saturated with sodium chloride, and extracted into diethyl ether. Individual phenols were separated and identified by thin-layer chromotography. The phenols identified were resorcinol, catechol, *p*-cresol, phenol, *m*-cresol, quaiacol, eugenol, *p*-methoxy phenol, and *m*-methoxy phenol. It was found that phenol, eugenol, quaiacol, and *p*-methoxy phenol occur in higher quantities than the other phenols. Quaiacol and eugenol along with resorcinol, *p*-cresol, and *m*-methoxy phenol are responsible for the aromatic flavor of Natu tobacco.

80–1326. Narasimha Rao, C. V.; Chakraborty, M. K. **Phytochemicals From Tobacco Wastes. 1. Survey of Raw Materials.** *Tobacco Research* 4(1): 52-58, June 1978.

Different kinds of tobacco waste materials obtainable in India have been evaluated for their nicotine, solanesol, and malic-citric acid content in order to select the appropriate raw material for chemurgic development. Analytical data show that flue-cured tobacco wastes have maximum solanesol content, bidi wastes have maximum nicotine content, and Natu type excels all others in organic acid content. Factory wastes are relatively poor raw material for the recovery of organic chemicals. An integrated approach of recovering these different phytochemicals from tobacco wastes has been proposed and work in progress has been highlighted. (Auth. Abs.)

80–1327. Pflugmacher, J.; Ebing, W. **Methode zur Bestimmung von Chlorkohlenwasserstoff-Insecticiden in Tabak mit Hilfe der Co-Sweep-Distillation. [Co-Sweep-Distillation Cleanup of Tobacco for Determination of Chlorinated Hydrocarbon Pesticides.]** *Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung* 169(5): 343-345, 1979, German.

The Co-sweep distillation (CSD) technique, commonly used to determine chlorinated insecticides in foodstuffs, was applied to the determination of chlorinated pesticide residues in tobacco. However, the CSD cleanup coupled with purification of the CSD eluate in a chromatographic column resulted in low recovery rates of certain pesticides, notably those of the DDT group. The low recovery rates were attributed to a reaction of the pesticide residues with tobacco components during the CSD process. The method was modified by adding a preliminary cleanup step. The modified CSD procedure consisted of extracting a tobacco sample with a petroleum ether-ethyl acetate mixture, purifying an aliquot of the extract through a florisil microcolumn, then processing the concentrated eluate in a CSD apparatus, and, finally, purifying the CSD eluate through a celite/sulfuric acid chromatographic column. Using this procedure, the recovery rate of added pesticides at a level of 0.05-0.3 mg/kg varied from 77.3 to 99.6 percent. The recovery rates determined by the modified CSD technique were found to be in good agreement with the values determined in the two tobacco samples by a commonly used method. The new method makes possible a quick (20 min) low-cost analysis of tobacco for chlorinated pesticides.

80–1328. Reeves, R. G.; Woodham, D. W.; Ganyard, M. C.; Bond, C. A. **Preliminary Monitoring of Agricultural Pesticides in a Cooperative Tobacco Pest Management Project in North Carolina, 1971—First-Year Study.** *Pesticides Monitoring Journal* 11(2): 99-106, September 1977.

A preliminary tobacco pest management pesticide monitoring project was initiated in two counties of North Carolina in 1971 to develop an ecologically, economically, and socially acceptable system for protecting tobacco from damaging insect and disease pests. Another purpose of the study was to monitor pesticide residues in biotic and abiotic environmental components (including tobacco leaves, fish, turtles, frogs, and soils). Results from the first year's study, including sampling procedures, sample preparation, and analysis and residue data, are presented in this paper. No evidence was produced that pesticide use in these counties is causing significant residue accumulation in the environment. (Auth. Abs. Mod.)

80–1329. Rosa, N. **Pyrolysis-Gas Chromatographic Estimation of Tobacco Alkaloids and Neophytadiene.** *Journal of Chromatography* 171: 419-423, 1979.

A pyrolysis-gas chromatographic (PGC) procedure is described that permits quantitative and qualitative evaluation of alkaloids (e.g., nicotine, nornicotine, neophytadiene) in tobacco. Alkaloid analysis by steam distillation and PGC were compared. In the steam distillation method, alkaloids were analyzed on a Varian 3700 gas chromatograph. Pyrolysis studies were conducted with a Victoreen pyrolyzer. Standard alkaloids were redistilled under vacuum for nicotine, or isolated from a sample of tobacco for nornicotine. Separation was accomplished by collecting the appropriate peaks from the Varian 3700 using thermal conductivity detectors. Standard tobaccos and samples were pyrolyzed in a sequence of 10 standards spaced among samples. The areas obtained for the standards were subjected to linear regression analysis to establish a regression formula used to estimate values of nicotine and nornicotine from the samples. Results of quantitative analysis by PGC were similar to those obtained by solvent extraction followed by gas chromatography (correlation coefficients 0.983 and 0.948, respectively). Samples with high nornicotine, low nicotine, or low total alkaloid were readily detected. The steam distillation procedure overestimated nornicotine. PGC offers a more accurate and rapid means of estimating alkaloids in tobacco than does steam distillation, and samples can be analyzed without extraction.

80–1330. Sakuma, H.; Ohsumi, T.; Sugawara, S. **Ether-Soluble Neutral Portion of Cellulose Cigarette Smoke Condensate.** *Agricultural and Biological Chemistry Journal* 43(12): 2619-2621, 1979.

The ether-soluble neutral fraction of cellulose cigarette smoke was investigated in this study. After extraction and purification over a silicic acid column, the yield had a strong and almond/cherry-like odor and was approximately 12 percent of the smoke condensate. The 93 components (53 oxygenated and 40 hydrocarbon compounds) of this fraction were identified by gas chromatography and gas chromatography-mass spectrometry. Among the 53 oxygenated compounds, there were 6 aliphatic compounds, 5 ketones, and 1 aldehyde. The other 47 compounds were ring compounds structurally classified into 10 furans, 10 benzenes, 7 lactones, 6 benzofurans, 6 cyclopentenones, 5 indanones, 2 naphthalenes, and 1 cyclopentanone. Of the 93 compounds, 63 were identified as cellulose pyrolysates for the first time in this study. Coumarin, one of the 63, has a sweet, pleasant odor, and is often added to tobacco compositions for enrichment of smoke flavor and taste.

80–1331. Sakuma, H.; Sugawara, S. **Composition of the Water-Soluble Portion of Cellulose Cigarette Smoke Condensate.** *Agricultural and Biological Chemistry Journal* 43(7): 1585-1589, 1979.

Water-soluble portion of the smoke condensate of cellulose cigarette was studied and 42 compounds (including 10 lactones, 6 cyclopentanones (or cyclopentenones), and 5 pyrans) were either positively or tentatively identified. Of these 42 compounds, 12, such as succinaldehyde and 5-hydroxy-1-pentenone were identified for the first time in cellulose pyrolyzate. Semiquantitative estimation of some of the major components in water-soluble portion revealed that 1,6-anhydroglucopyranose (levoglucosan) was the most abundant, followed by 2-hydroxy-3-methyl-2-cyclopentenone (cyclotene), acetol, and 2-hydroxy-5-pentanolide. Glycolaldehyde,which seems to be more abundant than cyclotene on the chromatogram, could not be estimated because of peak tailing. Of these water-soluble components, succinaldehyde has a very pungent and irritating effect and seems to be a cause of the pungent and irritating odor of cellulose cigarette smoke. (Auth. Abs.)

80–1332. Schmeltz, I.; Wenger, A.; Hoffmann, D.; Tso, T. C. **Use of Radioactive Tobacco Isolates for Studying the Formation of Smoke Components.** *Journal of Agricultural and Food Chemistry* 26(1): 234-239, 1978.

Tobacco plants exposed to $^{14}\mathrm{CO}_2$ during growth served as sources for radioactive phytosterols, neophytadiene, alkaloids, and fatty acids. Techniques for the isolation and identification of these compounds included solvent distribution, gel-permeation chromatography, thin-layer chromatography in conjunction with radioscanning, and gas chromatography—mass spectrometry. The radioactive isolates were incorporated into cigarettes, and their distribution patterns in the smoke were determined. A substantial portion of the labeled isolates was combusted to $^{14}\mathrm{CO}_2$, in addition to being transferred intact into the smoke tars, and degraded to volatile organic compounds. When cigarettes containing randomly labeled (^{14}C)nicotine were smoked, more than half the radioactivity was distributed into the tar of both streams, but principally in sidestream tar (32 percent). A total of about 36 percent of the radioactivity of the leaf ^{14}C-labeled phytosterols transferred into the tars of mainstream and sidestream smoke (19 percent into the former, 17 percent into the latter). Radioactivity was also distributed into the vapor phase of both smoke streams, 20 percent as CO_2 appearing predominantly in the sidestream, and 7 percent as volatile organic compounds, again predominantly in the sidestream. Neophytadiene, a major tobacco terpenoid, was distributed in the smoke as follows: in the total tar (mainstream plus sidestream)—44 percent; in predominately the sidestream, 31 percent combusted to CO_2, and 11 percent converted to volatile organics. In the case of palmitic acid, a fatty acid, 38 percent went into the tar (23 percent in mainstream, 15 percent in sidestream). Carbon dioxide accounted for 15 percent of the leaf acid, virtually all of the CO_2 appearing in the sidestream. Measurable quantities (<1 percent) were also converted to polynuclear aromatic hydrocarbons. The techniques utilized can serve as models for additional studies of this kind and lead to practical approaches for modifying smoke composition.

80–1333. Wahlberg, I.; Wallin, I.; Nordfors, K.; Nishida, T.; Enzell, C. R.; Reid, W. W. **New Labdanic Diterpenoids Isolated From Tobacco.** *Acta Chemica Scandinavica. Series B* 33(7): 541-543, 1979.

Seventeen labdanic compounds have been isolated from *Nicotiana tabacum* PB (Bergerac). Of these, eight are new tobacco constituents and seven, the (12*S*, 13*S*)-, (12*R*, 13*R*)- and (12*R*, 13*S*)-8,13-epoxy-14-labden-12-ols, 12,15-epoxy-12,14-labdadien-8-ol, the (11*E*, 13*S*)- and (11*E*, 13*R*)-11,14-labdadiene-8,13-diols and (13*E*)-15-acetoxy-13-labden-8-ol are new natural products. The isolation of six other compounds, all of which have previously been obtained as products of the reaction of (12*Z*)- and/or (12*E*)-abienol (1, 2) with singlet oxygen, strongly corroborates current views on the biogenesis of the tobacco labdanoids. (Auth. Abs.)

See also, **80–1501**

PHARMACOLOGY AND TOXICOLOGY

80–1334. Baer, D. S.; McClearn, G. E.; Wilson, J. R. **Effects of Chronic Administration of Tobacco Smoke to Mice: Behavioral and Metabolic Measures.** *Psychopharmacology* 67(2): 131-137, February 1980.

Tobacco smoke was administered to male and female mice of four inbred strains and two lines selectively bred for high activity (HA) or low activity (LA) in an open field. Administration occurred during 14 daily 10-min pretreatment sessions in a box filled with smoke from a nonfiltered cigarette (2 mg nicotine/cigarette; average density, 750 ppm carbon monoxide). When open-field activity was subsequently measured in the absence or presence of smoke (average density, 150 ppm carbon monoxide), pretreated mice had significantly lower activity scores than controls. Comparisons of open-field activity scores under smoke-present and smoke-absent conditions revealed that effects of this acute exposure were dependent upon genotype: C3H/2Ibg activity was almost tripled in the presence of smoke; DBA/2Ibg activity was increased; but HA, LA, and C57BL/6Ibg activity scores were depressed. As measured by open-field activity, development of tolerance to the effects of acute exposure to tobacco smoke after chronic pretreatment was also genotype-dependent. Significant ($p<0.001$) genotypic differences were found for nicotine remaining in liver, brain, and blood samples when mice were sacrificed 2.5 or 5 min after a weight-specific injection of radiolabeled nicotine. Tissue nicotine levels were also related to sex, time after injection, and pretreatment interactions with genotype. Strong positive correlations ($p<0.05$) were found between the measures of brain and liver levels at 5 min after injection and the behavioral measure of open-field activity under the smoke-present condition. (Auth. Abs.)

80–1335. Bell, H.; Orjasaeter, H.; Lange, H. F. **Carcinoembryonic Antigen (CEA) in Patients With Alcoholic Liver Diseases.** *Scandinavian Journal of Gastroenterology* 14(3): 273-279, 1979.

The mean plasma carcinoembryonic antigen (CEA) level was found to be significantly increased in 66 patients with alcoholic liver disease (4.4 μg/l) compared with the mean CEA level in 164 healthy blood donors (1.6 μg/l, $p<0.001$). Eighteen of the patients (27 percent) had values above our upper normal values of 5 μg/l, compared with 3 of 41 (7 percent) in a group of patients with nonalcoholic liver diseases. Of the 18 patients with CEA values values >5μg/l, 14 were smokers and 4 were nonsmokers. CEA was significantly increased in patients with complicating disorders. Much of the increase of CEA in many patients with alcoholic liver disease might be secondary to complicating disorders in the respiratory and/or gastrointestinal tracts. Other complicating diseases were found in 10 of the 14 smokers, and in 3 of the 4 nonsmokers who had plasma CEA levels >5 μg/l. The two highest plasma CEA levels (11.5 and 10.9 μg/l) were both found in smokers, the former level in a patient with moderate alcoholic liver affect and the latter level in a patient with an alcoholic fatty liver. These complicating disorders could explain why patients with alcoholic liver disease more often than other patients with benign diseases have elevated CEA. (Auth. Abs. Mod.)

80–1336. Biswas, N. M.; Patra, P. B.; Sanyal, S.; Deb, C. **Adrenogonadal Interactions in Nicotine-Induced Alteration of Steroidogenesis in Male Rat.** *Andrologia* 11(3): 227-233, May-June, 1979.

Chronic nicotine administration (10 mg/kg) inhibited steroidogenesis in rat testis. This inhibition is possibly secondary to increased adrenomedullary norepinephrine activity, since the antisteroidogenic effect of nicotine can be prevented by an alpha-adrenergic blocker like phentolamine. In vitro incubation of testicular slices with nicotine failed to elicit any appreciable effect on Δ^5-3β-hydroxysteroid dehydrogenase activity indicating lack of direct action of nicotine on testicular steroidogenesis. (Auth. Abs.)

80–1337. Boxall, R. R.; Gilpin, G. R.; Bensilum, S.; Lindsay, D. W. **Short Term Bioassay of Cigarettes Containing Cytrel Tobacco Supplement.** *Toxicology* 13(2): 175-186, 1979.

The toxicity of smoke from cigarettes containing tobacco, Cytrel tobacco supplement, or mixtures of the two materials, was investigated using six short-term test methods. Rat trachea organ culture and mouse skin thickening tests, which may provide information on carcinogenicity; a ciliatoxicity test; an alveolar macrophage viability test; a hemolysis assay; and a thiol inactivation test to provide measures of irritant and cytotoxic effects were used. In each case, a response to the smoke from cigarettes containing tobacco alone was detected, while the magnitude of this response declined as the proportion of tobacco supplement in the blend increased. The least response was found with smoke from cigarettes containing Cytrel tobacco supplement alone. (Auth. Abs.)

80–1338. Bruce, N.; Parkinson, S. **Effect of Nicotine on Uterine Blood Flow in Anesthetized Pregnant Rats.** *Biology of Reproduction* 21(1): 229-233, 1979.

A venous outflow technique was developed to examine the influence of nicotine on uterine blood flow (UBF) in anesthetized rats at days 16 or 22 of gestation; term is day 23. Nicotine was infused intravenously for 5-min periods at the rate of 1, 10, or 100μg/min which, adjusted for body weight, is about 1, 10, or 100 times the rate absorbed during cigarette smoking in humans. The results at days 16 and 22 were similar. Nicotine at the two lower infusion rates had little or only transitory effects on arterial pressure, pulse rate, and UBF. At the highest infusion rate, however, there was a pronounced rise then fall in arterial pressure, uterine vascular resistance increased, and UBF fell precipitously to about 51 percent (day 16) and 70 percent (day 22) of pretreatment values. It seems that nicotine can restrict UBF in the anesthetized pregnant rat but only at levels greatly exceeding the equivalent of those reached during cigarette smoking. (Auth. Abs.)

80–1339. Cheraskin, E.; Ringsdorf, W. M., Jr. **Tobacco Consumption and Human Hair Lead Levels. (Letter).** *Southern Medical Journal* 72(10): 1350, October 1979.

In order to examine the possible relationship of daily tobacco consumption to the concentration of lead in human hair, 58 subjects were divided into 2 age- and sex-paired groups. In the 29 smokers, the mean (±S.D.) for lead in hair was 14.9±8 ppm. In the nonsmokers, the value was 10.2±4.8 ppm. This difference was statistically significant ($p<0.005$), and on a mean basis, indicates that the lead level is approximately 50 percent higher in the smoking group.

80–1340. Chinoy, N. J.; Seethalakshmi, L. **Effects of Drugs on the Reproductive Physiology of Male Albino Rats: Part II—**

Nicotine and Morphine. *Indian Journal of Experimental Biology* 16(3): 323-325, March 1978.

Effects of morphine and nicotine on testes and epididymis revealed that these drugs reduced their androgen-dependent metabolism. Significant decreases in enzyme activity were seen for succinate dehydrogenase ($p<0.001$), alkaline phosphatase ($p<0.001$), and acid phosphatase ($p<0.001$). However, the changes induced were largely reversible by the administration of either testosterone propionate or ascorbic acid. The latter treatment was more effective in overcoming the effects of the drugs. The use of ascorbic acid in prophylactic treatment is suggested. (Auth. Abs. Mod.)

80-1341. Cumming, G.; Guyatt, A. R.; Holmes, M. A. **The Absorption of Carbon Monoxide From the Conducting Airways of the Human Lung.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 168-170.

The mechanism of carbon monoxide (CO) uptake from the conducting airways of the human lung was investigated by introducing a bolus of CO (0.3 percent concentration) into the tidal volume. Following expiration, the volume of CO recovered was measured and the absorbed volume determined. The study showed that, without breath-holding, there was no appreciable uptake of carbon monoxide in male smokers when the bolus was given in the last 120 ml of inspiration, i.e., was confined to the large airways. The results in females were similar, but much less clear. With prolonged breath-holding, however, some loss of CO may occur due to diffusion in the gaseous phase from the large airways into the gas exchanging parts of the lung.

80-1342. De Leacy, E. A.; McLeay, C. D.; Eadie, M. J.; Tyrer, J. H. **Effects of Subjects' Sex, and Intake of Tobacco, Alcohol and Oral Contraceptives on Plasma Phenytoin Levels.** *British Journal of Clinical Pharmacology* 8(1): 33-36, July 1979.

Steady state plasma phenytoin levels in 210 epileptic patients were studied by computerized analysis of covariance to determine whether the subject's sex, alcohol intake, tobacco smoking, or use of oral contraceptives influenced the relation between plasma drug level and drug dose. Sex, tobacco smoking (i.e., 0, <10, or >10 cigarettes/day), and alcohol usage had no statistically significant effect. There was a trend toward higher phenytoin levels relative to drug dose in oral contraceptive users. This finding prompted an additional study of plasma phenytoin levels in 40 oral contraceptive users and 135 age-matched nonusers. Analysis of covariance again showed higher plasma phenytoin levels relative to drug dose in users of oral contraceptives ($p=0.061$). This finding raised the possibility that the relation between plasma phenytoin level and drug dose differed between males and females who did not use oral contraceptives. However, when the relation between plasma phenytoin levels and drug dose was compared in 159 females who did not use oral contraceptives and in 101 males (both groups aged 15-70 years), no statistically significant difference was found. (Auth. Abs.)

80-1343. Drath, D. B.; Shorey, J. M.; Price, L.; Huber, G. L. **Metabolic and Functional Characteristics of Alveolar Macrophages Recovered From Rats Exposed to Marijuana Smoke.** *Infection and Immunity* 25(1): 268-272, July 1979.

Pulmonary alveolar macrophages were obtained by bronchopulmonary lavage from male rats after 30 consecutive days of in vivo exposure to marihuana or tobacco smoke. No significant differences were found between either group of experimental animals and controls in the number of cells recovered, the protein content per 10^6 cells, or the percentage of cells that adhered to plastic surfaces. The ability of macrophages to phagocytize viable bacteria was not affected by exposure to either marihuana or tobacco smoke in that both treatment groups ingested *Staphylococcus aureus* over a 60-min period as well as did control cells. Differences were found between the groups, however, with respect to cellular metabolism. Marihuana smoke inhalation caused a small decrease in the amount of oxygen consumed by macrophages during phagocytosis, as compared with control cells. This may have been reflected in the even greater decrease in superoxide formation observed during particle engulfment by these treated cells. Tobacco smoke, on the other hand, increased oxygen consumption and was without effect on superoxide release. Neither tobacco nor marihuana smoke treatment had an effect on the direct oxidation of glucose via the hexose monophosphate shunt. The results indicate that, despite several metabolic alterations in response to marihuana and tobacco smoke, alveolar macrophages were not compromised with respect to their ability to ingest a particulate challenge. (Auth. Abs.)

80-1344. Ehrly, A. M.; Schrimpf, W. J. **Der Einfluss des "akuten" Zigarettenrauchens auf die Verformbarkeit von Erythrozyten. [The Effect of "Acute" Cigarette Smoking on Erythrocyte Deformability.]** *Herz Kreislauf* 10(5): 245-246, 1978, German.

Deformability of red blood cells was measured with an 8μm-millipore-filter test system in young and elderly volunteers and in patients with vascular diseases before and after acute cigarette smoking. No change in blood filterability, i.e., red cell deformability, could be detected, indicating that a reduction in tissue blood flow cannot be attributed to impaired red cell deformability. (Auth. Abs.)

80-1345. Feron, V. J.; van den Heuvel, P. D.; Koeter, H.B.W.M.; Beems, R. B. **Significance of Particle Size of Benzo(*a*)pyrene for the Induction of Respiratory Tract Tumours in Hamsters.** *International Journal of Cancer* 25(2): 301-307, 1980.

To study the role of particle size of benzo(*a*)pyrene (BP) in the induction of respiratory tract tumors, groups of hamsters were given 52 weekly intratracheal instillations of fine (77 percent $<5.2\mu m$; 3 percent $<1.3\mu m$) or coarse (77 percent $<42\mu m$; 0 percent $<10\mu m$) BP particles suspended in a gelatin—0.9 percent NaCl solution. Both the fine and coarse particles were given at two different dose levels, i.e., 0.5 and 1.0 mg BP/instillation. Another group of hamsters received 52 weekly intratracheal instillations each consisting of 1.0 mg BP particles of a wide size range (90 percent $<50\mu m$; 3 percent $<5.0\mu m$) suspended in a 0.9 percent NaCl solution without gelatin. The retention of BP in the respiratory tract was studied in separate groups of hamsters receiving a single instillation of BP particles of the different size ranges. Coarse BP particles cleared less rapidly from the lungs and induced a much stronger tumor response in the respiratory tract than did fine BP particles. These findings demonstrated the crucial significance of particle size for the effectiveness of BP as a respiratory tract carcinogen. No appreciable differences in site of tumor formation were observed between treatment with fine and coarse BP particles, which led to the conclusion that the size of the particles did not play an important role in determining the location of the induced respiratory tract tumors. The elimination rate of the BP particles with a wide size range appeared to be in between those of the fine and the coarse particles. The tumor response found after administration of the BP particles of wide size range was very similar to that obtained with the high dose of coarse BP particles. (Auth. Abs.)

80–1346. Garrett, R.J.B.; Jackson, M. A. **Cigarette Smoke and Protein Synthesis in the Lung.** *Environmental Research* 21(2): 399-406, April 1980.

In vivo exposure of lung to cigarette smoke results in depression of protein synthesis by pulmonary tissue. Lungs from sham control rats synthesize protein at a rate of 0.97±0.16 nmole proline/mg DNA/min. Pulmonary protein synthesis during exposure to 45-60 puffs of smoke is inhibited 60-80 percent. The constituent of smoke which inhibits protein synthesis is not significantly removed by either Cambridge filters which absorb particulate matter or charcoal filters which absorb many gases. Carbon monoxide (CO) is not removed by either type of filter. To determine if a relationship does exist between the inhalation of CO and the inhibition of protein synthesis, animals were exposed to CO at a level comparable to that present in the cigarette smoke (i.e., "puffs" of 7 percent CO—93 percent N_2). The rate of protein synthesis in animals receiving 60 puffs of the CO mixture was 0.24±0.12 nmole proline/mg DNA/min ($n=8$), comparable to that of animals receiving 60 puffs of smoke. (Auth. Abs. Mod.)

80–1347. Gelbart, S. M.; Sontag, S. J. **Mutagenic Urine in Cirrhosis.** *Lancet* 1(8174): 894-896, April 26, 1980.

Multiple samples of daytime urine from 5 nonsmoking patients with cirrhosis, 12 healthy smokers, and 15 healthy nonsmokers were collected and analyzed for mutagens by *Salmonella*/microsome assay. All 12 samples collected from the 5 cirrhotic patients were mutagenic. Of the 17 samples collected from the smokers, 16 were mutagenic, while only 2 of the 24 samples from the nonsmokers were mutagenic. The discovery of mutagens in the urine of nonsmoking patients with cirrhosis is new. (Auth. Abs.)

80–1348. Gerrard, J. W.; Heiner, D. C.; Mink, J.; Meyers, A.; Dosman, J. A. **Immunoglobulin Levels in Smokers and Non-Smokers.** *Annals of Allergy* 44(5): 261-262, May 1980.

Immunoglobulin levels were compared in 90 smoking and 84 nonsmoking grain handlers and teachers. Mean IgG and IgM levels were significantly lower ($p<0.001$ and $p=0.01$, respectively), and IgE levels were significantly ($p<0.001$) higher, in smokers as a group than in nonsmokers as a group. IgA levels were not significantly lower in smokers than in nonsmokers. IgD levels were unchanged. Mean IgG levels in grain handlers and teachers were significantly ($p<0.001$) lower in smokers vs. nonsmokers for both occupations. However, IgE levels were significantly higher ($p<0.001$) in smoking vs. nonsmoking grain handlers. (Auth. Abs. Mod.)

80–1349. Giorguieff-Chesselet, M. F.; Kemel, M. L.; Wandscheer, D.; Glowinski, J. **Regulation of Dopamine Release by Presynaptic Nicotinic Receptors in Rat Striatal Slices: Effect of Nicotine in a Low Concentration.** *Life Sciences* 25(14): 1257-1261, 1979.

Rat striatal slices were continuously superfused with ^3H-tyrosine to study the effect of a low concentration of nicotine on the spontaneous release of newly synthesized ^3H-dopamine (^3H-DA). Nicotine ($10^{-6}M$) stimulated the calcium-dependent spontaneous release of ^3H-DA. This effect was prevented by nicotinic blockers such as pempidine ($10^{-5}M$) and d-tubocurarine ($5x10^{-6}M$). The stimulatory effect of nicotine and its blockade by pempidine were still observed in the presence of tetrodotoxin ($5x10^{-7}M$). This demonstrates for the first time that a low concentration of nicotine is effective in releasing DA by acting on presynaptic nicotinic receptors located on terminals of the nigro-striatal dopaminergic neurons. (Auth. Abs.)

80–1350. Goubran, E. Z.; Mikhail, M. M.; Malaty, H. A.; Mahfouz, M. **A Study of Some Histochemical Changes in the Lung, Heart and Aorta and Some Blood Enzymes Under the Influence of Experimental Exposure to Cigarette Smoke in the Rat.** *Cellular and Molecular Biology* 23: 119-131, 1978.

Adult normal male albino rats were exposed in a special apparatus to cigarette smoke, twice daily, for a period of 4 months to study serum and tissue enzymes as compared to control animals. The study of serum glutamic oxalacetic transaminase and serum lactic dehydrogenase activity showed that there was no change as a result of exposure to cigarette smoke, and resulted in the conclusion that no cellular or tissue damage took place in the heart or the lung. The study of enzymatic histochemical changes in the heart, aorta, and lungs suggested that AS-esterase was a lysosomal enzyme which increased in the phagocytic cells of the lungs as a result of smoke irritation. Acid phosphatase also increased in the phagocytic cells of the lungs as a result of smoking. The metabolic activity of the phagocytic cells of the lung increased as indicated by the enzyme succinic dehydrogenase. α-Esterase cytological localization was suggestive of being mitochondrial and practically unaffected by smoking. (Auth. Abs. Mod.)

80–1351. Guelker, H.; Brisse, B.; Brinkhoff, D. **Untersuchungen zur Wirkung von Nikotin auf Hamodynamik und Sauerstoffverbrauch des Herzens vor und nach β-Sympathikolyse und nach Gabe von Atropin. [Study of the Effect of Nicotine on Heart Hemodynamics and Oxygen Uptake Before and After β-Sympatholysis and After Administration of Atropine.]** *Verhandlungen der Deutschen Gesellschaft fuer Innere Medizin* 22: 835-839, April 26, 1979, German.

Hemodynamic parameters were measured and oxygen (O_2) uptake by the heart was calculated before and after intravenous infusion of 0.05 mg/kg nicotine over a 5-min period in 14 dogs. The dose and infusion time were comparable to nicotine uptake by a cigarette smoker. The data show that a 5-min nicotine infusion causes a significant increase in the systolic and diastolic aortic blood pressures (AoPsyst and PoPdist, respectively), the left ventricular blood pressure (LVP), the cardiac output (CO), and the cardiac contractility dp/dtmax. The average cardiac uptake of O_2 (ml/min x 100 g) increased by 19 percent after 8 min. In another experiment, intravenous infusions of atenolol were administered to eight dogs over a 20-min period to stimulate β-sympatholysis. Nicotine was then injected after a new hemodynamic steady state was established. Atenolol caused reductions in AoPsyst from 127 to 118 mm Hg, LVP from 134 to 117.9 mm Hg, CO from 1.38 to 1.17 l/min x 10 kg, dp/dtmax from 3700 to 1838 mm Hg/sec, and O_2 uptake from 7.22 to 5.52 l/100 g x min. The subsequent nicotine injection caused increases in AoPsyst to 125 mm Hg, LVP to 125.1 mm Hg, CO to 1.23 l/min x 10 kg, dp/dtmax to 2138 mm Hg/sec, and O_2 uptake to 5.96 l/100 g x min (8 percent). Heartbeat decreased significantly from 104.3 to 99.8 beats/min. In another experiment, atropine was injected intravenously to six dogs, then nicotine was administered. Nicotine caused significant increases in heartbeat (from 141 to 163 beats/min), AoPsyst (from 105 to 133 mm Hg), LVP (from 113 to 143 mm Hg), and dp/dtmax (from 3380 to 4680 mm Hg/sec). After atropine injection, O_2 uptake increased from 6.34 to 10 l/100 g x min and, after subsequent nicotine injection, it increased to 13.7 l/100 g x min. The data indicate that the positive inotropic effect of nicotine can be attributed mainly to activation of the adrenergic sympathetic system. The negative chronotropic effects of nicotine are the consequence of the reflex action upon pressoreceptors, but a direct simulation of the vagus nerve is also probable.

80–1352. Hill, P.; Marquardt, H. **Plasma and Urine Changes After Smoking Different Brands of Cigarettes.** *Clinical Pharmacology and Therapeutics* 27(5): 652-658, May 1980.

Concentrations of nicotine, cotinine, and carboxyhemoglobin (COHb) were measured in the plasma of 9 subjects (7 males, 2 females) who smoked 1 cigarette of each of 10 different brands over a period of 10 weeks and then smoked 1 brand for 2 weeks. There were higher COHb levels in smokers of low carbon monoxide yield cigarettes, whereas the rise in nicotine after one cigarette of each of the different brands was related to its nicotine content. Assay of urinary and plasma cotinine levels in smokers smoking brands with different nicotine content showed that smokers adjusted their smoking habits to maintain a constant level of cotinine. This self-titration of nicotine by the smoker may pose a health hazard even with relatively low-nicotine cigarettes. (Auth. Abs. Mod.)

80–1353. Hugod, C. **Effect of Exposure to 0.5 ppm Hydrogen Cyanide Singly or Combined With 200 ppm Carbon Monoxide and/or 5 ppm Nitric Oxide on Coronary Arteries, Aorta, Pulmonary Artery, and Lungs in the Rabbit.** *International Archives of Occupational and Environmental Health* 44(1): 13-23, 1979.

Exposure of rabbits to 0.5 ppm hydrogen cyanide (HCN) alone or in combination with 200 ppm carbon monoxide (CO) for 1 and 4 weeks or combined exposure to 0.5 ppm HCN, 200 ppm CO, and 5 ppm nitric oxide for 2 weeks did not cause morphological changes in lungs, pulmonary arteries, coronary arteries, or aorta. The need for selective toxicity studies with tobacco smoke constituents is stressed. (Auth. Abs.)

80–1354. Hutchison, D.C.S.; Desai, R.; Bellamy, D.; Baum, H. **The Induction of Lysosomal Enzyme Release From Leucocytes of Normal and Emphysematous Subjects and the Effects of Tobacco Smoke Upon Phagocytosis.** *Clinical Science* 58(5): 403-409, May 1980.

The lysosomal enzymes of circulating polymorphonuclear leukocytes contain a potent elastase; release of this enzyme within the lung is thought to be responsible for the destruction of elastic tissue in pulmonary emphysema. The release of lysosomal enzymes from blood leukocytes of normal (n = 7) and emphysematous (n = 32, 14 smokers, 18 ex-smokers) subjects during phagocytosis of particulate material was studied in vitro. Acid phosphatase and acid ribonuclease were used as markers of lysosomal enzyme release, no sufficiently sensitive assay for elastase being available. Cigarette smoke was separated into particulate and soluble fractions. In a preliminary study, the particulate fraction stimulated enzyme release; in the experiments reported here, latex particles were used to produce this effect. Approximately one-third of the total lysosomal enzyme content was released to the exterior of the cell during phagocytosis of latex particles. In this respect there was no difference between normal and emphysematous subjects. The effects of the nonparticulate soluble fraction of cigarette smoke on phagocytosis-induced enzyme release were studied. This fraction inhibited enzyme release from polymorphonuclear leukocytes of normal subjects but not from those of emphysematous patients. When the soluble cigarette smoke fraction was replaced by the respiratory inhibitor, antimycin A, a similar inhibition of enzyme release occurred. The inhibition of phagocytosis in cells of normal subjects is presumed to be due to a respiratory inhibitor such as carbon monoxide in the soluble fraction of the smoke. It is postulated that the polymorphonuclear leukocytes of emphysematous patients are adapted to hypoxic conditions so that inhibition of enzyme release does not occur. (Auth. Abs.)

80–1355. Izquierdo, I.; Graudenz, M. **Memory Facilitation by Naloxone is Due to Release of Dopaminergic and Beta-Adrenergic Systems From Tonic Inhibition.** *Psychopharmacology* 67(3): 265-268, March 1980.

The post-training administration of naloxone (0.8 mg/kg) facilitates memory consolidation of the habituation of a rearing response to a tone in rats. Amphetamine (1.0-2.5 mg/kg) or nicotine (0.2-0.5 mg/kg), and amphetamine (2.5 mg/kg) plus nicotine (0.5 mg/kg) have no effect. The higher doses of amphetamine or nicotine, however, when given together with a dose of naloxone which is ineffective alone (0.2 mg/kg), markedly enhance consolidation. Haloperidol (0.5 mg/kg), propranolol (0.5 mg/kg), and phenoxybenzamine (2.0 mg/kg) have no effect on their own; whereas tolazoline (2.0 mg/kg) impairs consolidation. The effect of naloxone (0.8 mg/kg) is antagonized by haloperidol and by propranolol, but not by phenoxybenzamine or tolazoline. The results suggest that naloxone causes memory facilitation through the release of central dopaminergic and beta-adrenergic mechanisms from a tonic inhibitory influence of endogenous opiate peptide systems. (Auth. Abs.)

80–1356. Jarvis, L. R.; Whitehead, R. **Effect of Nicotine on the Morphology of the Rat Gastric Mucosa.** *Gastroenterology* 78(6): 1488-1494, June 1980.

The effect of oral nicotine administration on the morphology of the rat gastric mucosa was studied. Cell populations, neck cell mucus volume fraction, and mucosal area and depth were measured, and differences in mean values for each group were tested for statistical significance. It was found that the equivalent of approximately 10 times the normal daily intake of nicotine achieved by human smoking is related to a significant decrease in total mucus neck cell population, neck cell mucus volume fraction and mucosal depth, and to an increase in mucosal surface area. The nicotine doses caused no significant changes in the parietal or chief cell populations, body weight, or water consumption. (Auth. Abs.)

80–1357. Jones, R. T.; Farrell, T. R., III.; Herning, R. I. **Tobacco Smoking and Nicotine Tolerance.** In: Krasnegor, N. A. (Editor). *Self-Administration of Abused Substances: Methods for Study.* National Institute on Drug Abuse Research Monograph Series, No. 20, DHEW Publication No. (ADM) 78-727, July 1978, pp. 202-208.

Preliminary information is presented on physiologic and subjective effects of repeated rapid injections of nicotine to abstinent cigarette smokers and noncigarette smokers. The nicotine dosage for smokers was 700μg and for nonsmokers 300μg. Subjects were asked to report mood and symptoms before and after each injection and were asked periodically about craving for a tobacco cigarette. Among the physiologic measurements taken continually during the experiment were heart rate as measured by electrocardiogram, skin temperature, finger tremor as measured by power spectral analysis of an accelerometer taped to the index finger, and blood pressure as measured with a cuff on the right arm. All subjects had a significant increase in heart rate after the nicotine injections ($p<.0001$). The smokers had a significant increase in heart rate after the first three nicotine injections but no significant change after the fourth. Nonsmokers had a significant heart rate increase after all four injections. Blood pressure or finger tremor did not change after nicotine injection. Seven of the eight smokers reported the nicotine injections to be rapidly followed by a pleasurable, stimulant-like sensation. All subjects experienced feelings of skin flushing and warmth, light-headedness, cool extremities, increased heart rate, and some dizziness. The effects were short-lived and disappeared 3-4 min postinjection. All subjects were able to discriminate placebo injections from nicotine injections.

80–1358. Karnik, A. S.; Coin, E. J. **Carbon Monoxide Kinetics Following Simulated Cigarette Smoking.** *Archives of Environmental Health* 35(3): 146-147, May-June 1980.

Carbon monoxide kinetics were measured in the blood (percent carboxyhemoglobin) and alveolar phase (ppm carbon monoxide) after simulated cigarette smoking. Cigarette smoking was simulated using the same amount of carbon monoxide that 2R1F cigarettes manufactured by the Tobacco Research Institute would contain. Ten boluses of air containing carbon monoxide equivalent to smoking one cigarette were inhaled by six healthy nonsmoker volunteers. Carbon monoxide in the air phase was measured by an Ecolyzer and carboxyhemoglobin was measured by a CO-Oximeter. The mean rise in alveolar carbon monoxide immediately and 20 min after inhaling the last bolus was 3.3 and 3.1 ppm, respectively ($p<0.005$). The mean rise in carboxyhemoglobin immediately and 20 min after inhalation of the last bolus was 0.8 and 0.5 percent, respectively ($p<0.005$). The changes in carboxyhemoglobin were found to be similar to changes that occur when one cigarette is actually smoked. (Auth. Abs.)

80–1359. Kawashiro, T. **Kitsuen to Shin hai Kino. 6. Ketsueki no Kokyu Kino. [Respiratory Function of Blood.]** *Japanese Journal of Thoracic Diseases* 15(9): 589-594, September 1977, Japanese.

Arterial O$_2$ tension (PaO$_2$), arterial O$_2$ content (CaO$_2$), arterial O$_2$ saturation (SaO$_2$), and arterial carboxyhemoglobin saturation (SaCO) were measured using O$_2$ electrode and gas chromatograph on asymptomatic nonsmokers, light smokers, and heavy smokers whose ages ranged between 25 and 45 years. They were studied in the supine position. The mean PaO$_2$ for light smokers (90.5 ± 5.6(S.D.) torr) and for heavy smokers (84.1 ± 8.1 torr) was lower than that for nonsmokers (94.7 ± 6.7 torr). The mean CAO$_2$ for light smokers (17.7 ± 0.9 vol percent) and for heavy smokers (17.5 ± 1.4 vol percent) was also lower than that for nonsmokers (19.3 ± 0.7 vol percent). The mean O$_2$ capacity for light smokers (18.7 ± 0.8 vol percent) and for heavy smokers (18.5 ± 1.3 vol percent) was less than that for nonsmokers (19.8 ± 1.4 vol percent). The mean SaO$_2$ for light smokers (94.9 ± 2.9 percent) and for heavy smokers (95.0 ± 3.9 percent) did not differ from that for nonsmokers (94.7 ± 4.7 percent). The results indicate that the low CaO$_2$ in smokers was associated with low O$_2$ capacity, not with low SaO$_2$. The mean SaCO for light smokers (5.0 ± 1.4 percent) and for heavy smokers (6.8 ± 2.0 percent) was higher than that for nonsmokers (0.8 ± 0.2 percent). In another series of in vitro experiments, carbon monoxide (CO) dissociation curves under various conditions were provided which permitted estimation of the arterial CO tension (PaCO) from the SaCO reported here. The mean values of the estimated PaCO in nonsmokers, light smokers, and heavy smokers were 0.004 torr, 0.025 torr, and 0.032 torr, respectively. It is well known that high CO concentration is one of the effects of smoking which would cause the blood gas findings to vary. The results indicate that in smokers 70-90 percent of the reduction in O$_2$ capacity is related to the rise in the CO concentration. High concentration of CO appears to be the major factor in lowering the O$_2$ capacity and the CaO$_2$ in smokers. (Auth. Abs.)

80–1360. Ketkar, M. B.; Haas, H.; Althoff, J. **Implantation of Cigarette Smoke Condensate in the Lungs of Syrian Golden Hamsters.** *Journal of Cancer Research and Clinical Oncology* 94(1): 111-114, 1979.

Different doses of benzo(*a*)pyrene (BaP), cigarette smoke condensate (CSC) and its nitromethane fraction (NMF), in wax pellets, were directly implanted into the lungs of Syrian golden hamsters. Pleomorphic sarcomas were induced in the BaP-treated animals. Several epithelial proliferative lesions and a few bronchiogenic adenomas were induced in CSC- and NMF-treated hamsters. A species-specific effect has been observed in this investigation. (Auth. Abs.)

80–1361. Kirpekar, S. M.; Garcia, A. G.; Prat, J. C. **Action of Nicotine on Sympathetic Nerve Terminals.** *Journal of Pharmacology and Experimental Therapeutics* 213(1): 133-138, 1980.

Interaction between 4-aminophrydine (4-AP) and nicotine on sympathetic nerve terminals was studied in the isolated cat spleen slices, labeled with (^{3}H)norepinephrine ((^{3}H)NE). Incubation of slices for 5 min at 37°C in low (50 μM) and high (2 mM) concentrations of nicotine released 0.8 ± 0.08 and 2.73 ± 0.39 percent of tissue (^{3}H)NE. Tetrodotoxin (TTX) blocked the response to low nicotine but not to high nicotine. Low nicotine did not release (^{3}H)NE in the absence of calcium. Response to high nicotine which persisted in calcium-free solution was blocked by ethylene glycol bis(β-aminoethyl ether)N,N′-tetraacetic acid. 4-AP (1 mM) not only enhanced the response to low nicotine but it effectively antagonized the suppressant effects of TTX and calcium-free solution on release induced by low nicotine. Restoration of release by 4-AP from TTX-blocked preparations occurred in the absence of calcium in the perfusion medium, but lanthanum (1 mM) blocked it. Restoration of release from spleen slices incubated in calcium-free Krebs' solution by 4-AP was blocked by lanthanum and prolonged incubation in calcium-free ethylene glycol bis(β-aminoethyl ether)N,N′-tetraacetic acid solution. It is concluded that at lower doses nicotine, by acting on nicotinic receptors, depolarizes the sympathetic nerve terminals to set off propagated action potentials which are responsible for NE release, and that 4-AP restores nicotine response in the presence of TTX or in the absence of calcium by mobilizing calcium both from extracellular and intracellular sources. (Auth. Abs.)

80–1362. Kjellstrom, T.; Nordberg, G. F. **A Kinetic Model of Cadmium Metabolism in the Human Being.** *Environmental Research* 16: 248-269. July 1978.

An eight-compartment kinetic model for the metabolism of cadmium based on animal and human tissue distribution data was formulated. The flow of cadmium between the compartments was generally assumed to follow first-order exponential functions. The 21 distribution coefficients were estimated by fitting the calculated cadmium concentrations in kidney, liver, urine, blood, and other tissues to the observed concentrations for Swedes with different smoking habits and with and without occupational cadmium exposure. It was estimated that smoking 20 cigarettes/day would add 3μg/day to the average daily cadmium intake of 16μg of a 50-year-old Swede. Calculated cadmium concentrations in the blood were 0.7 ng/g for nonsmokers and 1.6 ng/g for smokers. The calculated tissue concentrations for Japanese persons also agreed with observed concentrations. The model was used to calculate the daily cadmium intake necessary to reach critical concentration in kidney cortex. The average renal cortex concentration would reach 200μg/g at an average daily cadmium intake via food of 440μg for a European-American population and 325μg for a Japanese population. (Auth. Abs. Mod.)

80–1363. Kurogochi, Y.; Nakashima, T.; Nakanishi, Y.; Inoki, M.; Morita, N. **Kakushu Dobutsu ni Okeru Nicotine Taisha Kozo Kassei no soi ni Kausuru Kenkyu (1) Seijo Dobutsu no Taisha Kassei ni Tsuite. [Studies on Nicotine Metabolism in Various Animals (1) Strains and Species Differences of Drug Metabolizing Enzyme Activities in Male Intact Animals.]** *Journal of Nara Medical Association* 29(4/5): 677-682, October 31, 1978, Japanese.

The present study was designed to elucidate the influence of physiological factors on the metabolism of chemical compounds contained in tobacco smoke. Drug-metabolizing enzyme activities in the liver of various male animals were compared, using mice (ddY, C57BL/6J, DBA/2J, ICR, C3H/He, BALB/c, BDF_1), rats (Wistar, Sprague-Dawley, Fischer, Donryu), guinea-pigs (Hartley), rabbits (Japanese White), and mongrel dogs. These animals were given a commercial diet and tap water for a week and killed after an 18-hour fast. The liver was homogenized, centrifuged at 10,000 x g for 15 min at 0-4°C, and the supernatant analyzed for the activities of nicotine oxidase, acetylaminofluorene (AAF) hydroxylase, and benzo(*a*)pyrene (BP) hydroxylase. Nicotine oxidase activity was found, in decreasing order, in rabbit, dog, Wistar rat, and ddY mouse. Hepatic drug-metabolizing enzyme activities obtained in rats were (the highest and the lowest cases only given) as follows: nicotine oxidase—Fischer, Sprague-Dawley (85 percent of Fischer); AAF hydroxylase—Donryu, Fischer (83 percent of Donryu); and BP hydroxylase—Sprague-Dawley, Donryu (<60 percent of Sprague-Dawley). Those in mice were as follows: nicotine oxidase—C3H/He, ddY (63 percent of C3H/He); AAF hydroxylase—BALB/c, C57BL/6J (78 percent of BALB/c); and BP hydroxylase—C57BL/6J, DBA/2J (1/3 of C57BL/6J). These results indicate strain differences in drug metabolism and no conceivable correlation between the various strains as regards these analogous enzyme activities. (Auth. Abs.)

80–1364. Lellouch, J.; Claude, J. R.; Thevenin, M. **α_1-Antitrypsine et Tabac, une Etude de 1296 Hommes Sains. [Smoking and Serum α_1-Antitrypsin in 1296 Healthy Men.]** *Clinica Chimica Acta* 95(2): 337-345, 1979, French.

Serum α_1-antitrypsin (α_1-AT) was measured by radial immunodiffusion in 1,296 healthy men aged 18-50 years. Other biological criteria, including leukocyte count and α_2-globulins, were measured and the subjects were given a detailed questionnaire on their smoking habits. Results showed a very strong positive ($p<0.001$) relationship between smoking and serum α_1-AT: the heavy smokers had a serum α_1-AT 20 percent higher than the nonsmokers, and among subjects who stopped smoking, the level rapidly returned to normal. There were also close interrelationships between serum α_1-AT, smoking, leukocyte count, and α_2-globulins. A discussion of these results is presented. (Auth. Abs.)

80–1365. Lichtensteiger, W.; Richards, J. G.; Kopp, H. G. **Changes in the Distribution of Non-Neuronal Elements in Rat Median Eminence and in Anterior Pituitary Hormone Secretion After Activation of Tuberoinfundibular Dopamine Neurones by Brain Stimulation or Nicotine.** *Brain Research* 157(1): 73-88, November 17, 1978.

In order to investigate the possibility of acute functional changes in non-neuronal elements (mainly tanycytes) of the median eminence, the proportion of portal capillary surface covered by such elements was measured by quantitative electron microscopy in ovariectomized estrogen-progesterone-pretreated rats. In some of these animals, the functional state of the tuberoinfundibular dopamine (DA) neurones was assessed by histochemical microfluorimetry. Serum concentrations of luteinizing hormone (LH), growth hormone (GH), and prolactin were determined by radioimmunoassay. Two different types of treatment, i.e., systemic administration of nicotine (1 mg/kg, subcutaneously) or electrical stimulation in the medial amygdaloid nucleus, markedly reduced the percentage of capillary surface covered by non-neuronal profiles within 20 and 15 min, respectively. At the same time, the tuberoinfundibular DA system responded by an increase in cellular fluorescence intensity, reflecting neuronal activation. Medial preoptic stimulation had basically the same effect but with more variability in the change in capillary coverage by tanycytes. The action of nicotine was prevented by pretreatment with the DA receptor blocking agent, pimozide (5 mg/kg), which indicates (1) that a dopaminergic mechanism was involved in the nicotine effect and (2) that the tanycyte response was elicited by DA released from nerve terminals acting at some receptor site. Nicotine also lowered serum levels of GH ($p<0.05$) and prolactin ($p<0.01$). Pimozide antagonized only the effect on prolactin. While the reaction of DA neurones and capillary coverage by tanycytes were correlated with each other in individual rats, no statistically significant correlation was observed between tanycyte response and hormone levels, so that no conclusions can as yet be drawn as to the neuroendocrine significance of the tanycyte reaction. These results indicate that rapid changes in the proportion or portal capillary surface covered by non-neuronal profiles can be elicited by stimulation of extrahypothalamic brain areas or by activation of cholinergic mechanisms. The tanycyte response appears to be mediated at least in part by the tuberoinfundibular DA neurones. (Auth. Abs.)

80–1366. Lingjaerde, O. **Inhibition of Platelet Uptake of Serotonin in Plasma From Patients Treated With Clomipramine and Amitriptyline.** *European Journal of Clinical Pharmacology* 15(5): 335-340, June 12, 1979.

The inhibition of serotonin uptake by platelets has been measured in blood from 20 patients on amitriptyline (50-225 mg daily), 14 patients on clomipramine (25-200 mg daily), and in an untreated group of 21 depressed patients. A complete kinetic analysis was carried out in each patient. Using the increase in the kinetic parameter K_m as a measure of uptake inhibition, there was a high correlation between the daily dose and inhibition within each drug group, clomipramine being about 10 times more potent than amitriptyline. The inhibition did not vary with age, sex, duration of treatment (up to 3 years), or concomitant use of moderate doses of benzodiazepines, neuroleptics, or lithium. In the amitriptyline group, the inhibition was significantly ($p<0.05$) smaller in smokers than in nonsmokers. The kinetic parameter V_{max} was essentially unchanged in the amitriptyline group, and was markedly reduced in the clomipramine group, but without any correlation with dose. The mixed competitive—noncompetitive effect of clomipramine confirms previous in vitro findings. (Auth. Abs.)

80–1367. Lubawy, W. C.; Perrier, D. G. **The Effect of Acute Tobacco Smoke Exposure on Pulmonary Benzo(*a*)pyrene Metabolism.** *Environmental Research* 21(2): 438-445, April 1980.

The influence of whole cigarette smoke or the gas phase from smoke on the pulmonary metabolism of (^{14}C)benzo(*a*)pyrene ((^{14}C)BaP) was examined. (^{14}C)BaP disappears from plasma-perfusing isolated rabbit lungs according to first-order kinetics. When these lungs are ventilated with freshly generated whole cigarette smoke, the slope of (^{14}C)BaP disappearance curves are altered dramatically; the amount of this alteration is dependent on the dose of smoke administered. If the particulate phase is filtered out of the smoke and the resulting gas phase is administered to the lung, relatively little effect is seen. These results suggest that the acute effect of whole smoke on BaP metabolism by the isolated perfused lung is inhibition and that this effect is primarily due to the materials present in the particulate phase of smoke. (Auth. Abs.)

80–1368. Luscombe, D. K.; John, V. **Influence of Age, Cigarette Smoking and the Oral Contraceptive on Plasma Concentrations of Clomipramine.** *Postgraduate Medical Journal* 56(Supplement 1): 99-102, 1980.

The influence of age, cigarette smoking, and the oral contraceptive on the plasma profile and tolerability of clomipramine has been studied in depressed patients given the drug daily for 4 weeks. Older patients tend to have higher plasma concentrations of clomipramine than the younger and tolerate clomipramine less well; this latter is considered to be at least partly due to the increased plasma concentrations of the drug. Clearly elderly patients need a lower dose than the young if a high incidence of side effects is to be avoided. Patients smoking ≥ 15 cigarettes/day were found to tolerate daily doses of 75 mg clomipramine far better than nonsmokers. Perhaps surprisingly, this was not reflected in lower plasma clomipramine concentrations in smokers, possibly owing to the high dropout rate among nonsmokers, which biased these data. Finally, no interaction was observed in females between clomipramine and oral contraceptives, the concomitant administration of which did not influence either plasma clomipramine concentrations or its tolerability. (Auth. Abs.)

80–1369. Martin, J. C.; Martin, D. D.; Radow, B.; Day, H. E. **Life Span and Pathology in Offspring Following Nicotine and Methamphetamine Exposure.** *Experimental Aging Research* 5(6): 509-522, 1979.

Seventy-nine primiparous Sprague-Dawley rats received subcutaneous injections of either 3.0 mg/kg of pure nicotine, 5.0 mg/kg of methamphetamine hydrochloric acid, 5.0 mg/kg of saline vehicle, or received no injections during the 21-day gestational and 19-day nursing period. Male offspring were divided into three groups at weaning. The autopsy animals ($n = 80$) were sacrificed at selected periods during the lifespan, the aging animals ($n = 60$) were weighed monthly until death, and the behavioral rats ($n = 48$) were monitored on a variety of behavioral tasks throughout their lifespan. Measures reported include lifespan data, organ pathology, and tumor formation. Significant differences among the four offspring groups were found for tumor incidence, gross pathology, and age at death in the autopsy group; and weight maintenance in the aging group. (Auth. Abs.)

80–1370. Masden, R. R.; Flowers, N. C. **Extension of Experimental Infarction with Nicotine and Estimates of Infarct Size.** *American Heart Journal* 99(3): 342-348, March 1980.

The effect of acute nicotine administration on myocardial infarct size was evaluated in 19 dogs using the catheter-instilled bead technique for controlled-size myocardial infarcts. An infusion of nicotine (800μg/min) was administered to 9 dogs; 10 dogs served as controls. Body surface potential difference maps were recorded to determine the volume of infarcted muscle electrically. These volumes were compared with those calculated from morphologic assay at autopsy. By both electrical infarct volume and anatomic infarct volume assays, the volume of infarcted myocardium expressed as percentage of total heart volume was greater ($p<0.0001$) in the nine nicotine-exposed dogs than in the control dogs. This model may prove appropriate in the study of the role of tobacco smoke constitutents in the development of coronary atherosclerotic disease.

80–1371. Okano, P.; Miller, H. N.; Robinson, R. C.; Gelboin, H. V. **Comparison of Benzo(*a*)pyrene and (-)-*trans*-7,8-Dihydroxy-7,8-dihydrobenzo(*a*)pyrene Metabolism in Human Blood Monocytes and Lymphocytes.** *Cancer Research* 39(8) 3184-3193, August 1979.

The benzo(*a*)pyrene (BP) metabolite pattern formed by monocytes and lymphocytes includes three dihydrodiols, a major 7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene (7,8-diol), low or moderate levels of 4,5-dihydroxy-4,5-dihydrobenzo(*a*)pyrene, and barely detectable levels of 9,10-dihydroxy-9,10-dihydrobenzo(*a*)pyrene; at least three phenols, 3-, 7-, and 9-hydroxybenzo(*a*)pyrene; and two major separable quinones, benzo(*a*)pyrene-1,6- and 3,6-diones. BP metabolite patterns formed by benz(*a*)anthracene (BA)-induced monocytes and lymphocytes from the same individual show that each cell type has characteristic differences in the relative ratios of the BP metabolites formed. Relatively greater proportions of 7,8-diol and 9-hydroxybenzo(*a*)pyrene are synthesized by monocytes, while lymphocytes form relatively higher levels of 3-hydroxybenzo(*a*)pyrenes and the BP quinones. Thus, it appears that monocytes and lymphocytes contain different patterns of cytochrome P-450 mixed-function oxidases or metabolically related enzymes which are active in BP metabolism. The ratio of BP metabolites formed by BA-induced to that of control monocytes and lymphocytes is greater for the 7-hydroxybenzo(*a*)pyrene and 9-hydroxybenzo(*a*)pyrene and lesser for the 3-hydroxybenzo(*a*)pyrene and 7,8-diol in most samples examined. The total amounts of BP metabolites formed are quantitatively higher in BA-induced cells. These data suggest that induction by BA in these cells is selective and favors the formation of certain forms of cytochrome P-450 which change the metabolite patterns in induced cells. When (-)-*trans*-7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene is used as a substrate, both noninduced and BA-induced monocytes and lymphocytes metabolize the compound further to highly mutagenic 7,8-diol-9,10-epoxides. Analysis of the ethyl acetate-extractable tetrols and triols demonstrated that the major diol-epoxide formed is diol-epoxide 1, *r*, 7-*t*-8-dihydroxy-*t*-9, 10-oxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene. Results indicate that monocytes and lymphocytes exhibit different metabolite profiles, as do BA-induced and control cells. Induction of BA appears to favor BP over 7,8-diol metabolism, suggesting that the induction may lead to a greater ratio of detoxified to activated products relative to the ratio formed in control cells. The results also show that the amount of 7,8-diol metabolism in BA-treated cells is either similar to or only slightly greater than in control cells. This is in contrast to BP metabolism which is stimulated two- to fourfold by BA treatment. Thus, the form of cytochrome P-450 metabolizing the 7,8-diol to diol-epoxides is relatively uninducible compared to those cytochrome P-450 forms metabolizing BP. Improvements in methods are described which allow the analysis by high-pressure liquid chromatography of BP metabolism using relatively low numbers of human peripheral blood monocytes and lymphocytes. These improvements include the removal of BP by silica gel column chromatography prior to high-pressure liquid chromatography analysis and the prevention of the spontaneous oxidation of phenols to quinones during sample handling and storage by the addition of the antioxidant, vitamin E, to all the solvents. (Auth. Abs.)

80–1372. Papeschi, G.; Calsini, P.; Terreni, A.; Del Soldato, P.; Sesti, A. G. **Effect of Cigarette Smoke on the Elastic Recoil of Rat Lung and on the Physicochemical Properties of Washing Fluid.** In: Georgiev, G. A. (Editor). *Lung Lipid Metabolism. Methods of its Regulation and Alveolar Surfactant.* Proceedings of International Symposium on Lung Lipid Metabolism, Varna, Bulgaria, May 19-22, 1976, 1978, 218-225.

Two groups of 20 Wister male rats of approximately 250 g each were used to examine the effect of cigarette smoke on lung elasticity. The first group was subjected to a treatment with cigarette smoke in a cell for about 2 hours/day for 5 days. The second group was the control. Twelve animals of each group were used for the volume/pressure (V/P) curves; eight of each group were subjected to bronchopulmonary washing. The variability of the single points in the V/P curves obtained for the rat lungs of the two groups was very small both in the inflation and in the deflation sections. The slope of the inflation curve was the same both in the smoke-treated rats and in the controls. At equal volumes the pressure developed in the lungs of the smoke-treated rats is clearly greater than that of the controls. The residual volume for the smoke-treated rats is considerably smaller: this means that in these rats the elastic recoil is reduced and the elasticity is greater. The surface tension decreased in the controls more than in the smoke-treated rats. A value of 53.5±0.4 dyne/cm was obtained for the latter after 4 min, whereas at the same time a value of 51.4±0.3 dyne/cm was measured for the controls. The difference is significant and indicates either a lower concentration or a decreased surface activity of the surface-active material contained in the washing liquid.

80–1373. Pleban, P.; Ellis, K. J. **Cadmium Concentrations in Blood. (Letter).** *Science* 208(4443): 520, May 2, 1980.

In reply to an article on cadmium measurement in smokers and nonsmokers (*Science* 205(4403): 323-325, July 20, 1979), it is suggested that the whole blood cadmium concentration is a more appropriate parameter to measure than cadmium concentration in the plasma because studies on laboratory animals indicate that the major portion of the metal is contained in the erythrocytes. A significant difference ($p<0.001$) in the mean blood cadmium concentration between smokers and nonsmokers has been reported. The authors of the original article concur that the cadmium concentrations in the blood of smokers are higher than nonsmokers; however, their experiment found that plasma and urine data were not predictive of the kidney or liver cadmium burden in smokers. See also 80-1105.

80–1374. Pleho, A.; Tafro, S.; Prskalo, M.; Vatrenjak, V. **Effects of Nicotine on the Artificially Introduced Copper Content in Certain Rat Organs.** *Folia Medica (Sarajevo)* 12(1): 159-167, 1977.

Adult rats of 130 to 150 g body weight were treated with nicotine (Ni) oxalate for 8 days; subsequently, they were administered copper (Cu) sulfate intraperitoneally in a concentration of 10 mg/kg body weight. The copper content in blood, liver, kidneys, and brain was traced by spectrophotometry over three time intervals, i.e., after 1, 12, and 24 hours following copper sulfate application. Prior Ni administration in increasing doses has proved to precipitate Cu elimination from blood as well as from all three investigated organs. Precipitated elimination and consequently a lower Cu content in the organism may provide a protective effect of nicotine in Cu poisoning. The favorable effect of Ni, apart from the effect on Cu content, is apparently also due to its effect on heparin. (Auth. Abs.)

80–1375. Pop, R.; Moldovan, O.; Fodoreanu, L.; Leucuta, S. **Estimation of Diazepam Bioavailability From Tablets by the Measurement of Its Clinical Effect.** *Drugs Under Experimental and Clinical Research* 6(1): 45-52, February 1980.

The relative bioavailability of two brands of diazepam tablets (i.e., Valium and Diazepam) was estimated in 25 healthy subjects by having them perform certain psychological tests after a single oral dose of 20 mg diazepam. Quantification of the results was made by using scores established before administration of the dose, at 0.5, 1.0, 1.5, 2.0, and 3.0 hours after the dose administration. Interpretation of the data was made by analysis of variance. The bioavailability was estimated by the area under the curve of the scores vs. time. The degree of significance of the differences between the scores of each test, as well as of the total score obtained after various testing periods, was established. The statistical indexes of the linear correlation of the total score with diazepam serum concentration were satisfactory. The result showed the possibility of estimating diazepam bioavailability by measuring the clinical effect using psychological methods. A lack of sex influence on the effect, as well as a significant influence of cigarette smoking in the case of Valium, was also established. One hour after Valium administration there was a more intense effect in nonsmokers ($p<0.05$); however, after 1.5 hours, the difference was not significant. (Auth. Abs. Mod.)

80–1376. Porsius, A. J.; Van Zwieten, P. A. **The Central Actions of Nicotine on Blood Pressure and Heart Rate After Administration via the Left Vertebral Artery of Anaesthetized Cats. Distribution of Nicotine Into the Brain After Central Application.** *Arzneimittel-Forschung/Drug Research* 28(9): 1628-1631, 1978.

After injection into the vertebral artery of anesthetized cats, low doses of nicotine (range: 10^{-9}-10^{-8} mol = 0.5-5μg/kg) induced potent cardiovascular effects. A dose of 5×10^{-9} mol = 2.5μg nicotine (base)/kg reduced blood pressure by 35.8 percent and heart rate by 30.5 percent. The hypotensive action was always accompanied by a severe bradycardia. Dose—response curves for these pharmacological actions were established. It is concluded that nicotinic receptors in the medulla oblongata and pons are involved. Administration of 10^{-8} mol = 5μg (radioactive) nicotine/kg into the left vertebral artery gave rise to a specific distribution pattern. Nicotine mainly accumulated into the pontomedullary area, whereas higher brain regions (hypothalamus) contained only insignificant amounts. The experimental results suggest that the nicotine-like effect of endogenous acetylcholine might play an important physiological part in the central regulation of blood pressure and cardiac frequency. (Auth. Abs. Mod.)

80–1377. Rahimtula, A. D.; Zachariah, P. K.; O'Brien, P. J. **Differential Effects of Antioxidants, Steroids and Other Compounds on Benzo(*a*)pyrene 3-Hydroxylase Activity in Various Tissues of Rat.** *British Journal of Cancer* 40(1): 105-112, July 1979.

Antioxidants were found to inhibit the mixed-function oxidation of benzo(*a*)pyrene in several tissues of untreated and 3-methylcholanthrene-pretreated rats. The enzyme systems in the liver, kidney, and stomach were much more susceptible to inhibition than those in the lung, adrenal, colon, and small intestine. In all tissues except the stomach it was found that 3-methylcholanthrene pretreatment led to a decrease in inhibition of benzo(*a*)pyrene 3-hydroxylase activity. It is suggested that antioxidants exert their protective effect against cancer by inhibiting the formation of carcinogenic metabolites. Of the various steroids tested, only 17β-estradiol and estrone were significantly inhibitory in most tissues. Cholesterol was found to increase benzo(*a*)pyrene 3-hydroxylase activity in the gastrointestinal tract. (Auth. Abs.)

80–1378. Repace, J. L.; Lowrey, A. H. **Indoor Air Pollution, Tobacco Smoke, and Public Health.** *Science* 208: 464-472, May 2, 1980.

An experimental and theoretical investigation is made into the range and nature of the exposure of the nonsmoking public to respirable suspended particulates from cigarette smoke. A model incorporating both physical and sociological parameters is shown to be useful in understanding particulate levels from cigarette smoke in indoor environments (e.g., rooms in private residences and office buildings). Observed levels of particulates correlate with the predictions of the model. It is shown that nonsmokers are exposed to significant air pollution burdens from indoor smoking. An assessment of the public health policy implications of these burdens is presented. (Auth. Abs.)

80–1379. Robertson, G. B.; Rogers, A. W. **An Autoradiographic Search for Radioactive Particles in the Lungs of Cigarette Smokers.** *Archives of Environmental Health* 35(2): 117-122, March-April 1980.

Mucosal samples from the bifurcation of a major bronchus were examined from 23 patients undergoing diagnostic bronchoscopy. Smoking histories were recorded in detail and included age at which smoking habit began, number of cigarettes smoked/day, type of tobacco product smoked, whether the patients inhaled, current smoking status, and time interval between last cigarette smoked and the biopsy. There were six nonsmokers, nine ex-smokers, and eight smokers. Samples were autoradiographed using a technique that avoids any contact between tissue and reagents until the end of autoradiographic exposure. The autoradiographs were scanned for alpha particle tracks. No significant level of alpha activity was detected, even in samples from heavy smokers who had continued to smoke within several hours of the bronchoscopy. The lower limit of detection of alpha activity in this experiment was equivalent to about 55.5 becquerels/kg (1,500 pCi/kg) polonium-210. These results do not support the hypothesis presented by others that alpha radioactivity in particulate material of cigarette smoke contributes significantly to the association between cigarette smoking and bronchial carcinoma. (Auth. Abs. Mod.)

80–1380. Rylander, R.; Sjostrand, M.; Bergstrom, R. **Free Lung Cell Response After Combined Exposure to Cigarette Smoke and Industrial Dusts.** *Toxicology* 12(3): 211-220, 1979.

Guinea pigs were acutely exposed to different airborne dusts (manganese dioxide (MnO_2), silicon dioxide (SiO_2), aluminum oxide (Al_2O_3), and titanium dioxide (TiO_2)) and freshly generated cigarette smoke. The effect was evaluated by counting the number of free lung cells (leukocytes and macrophages) using a lavage method. An exposure to MnO_2 and smoke on the same day caused an increase in the number of leukocytes 24 hours thereafter. By increasing the time interval between the MnO_2 and smoke exposure, the effect gradually disappeared. Aluminum oxide or SiO_2 also caused an increase in the number of leukocytes, whereas TiO_2 had no effect. The pathogenesis behind the reaction and the epidemiological implications are discussed. (Auth. Abs. Mod.)

80–1381. Saunders, J. W.; Bush, L. P. **Nicotine Biosynthetic Enzyme Activities in *Nicotiana tabacum* L. Genotypes With Different Alkaloid Levels.** *Plant Physiology* 64(2): 236-240, August 1979.

Young plants of five *Nicotiana tabacum* L. genotypes were examined for activity of nicotine biosynthetic enzymes. Genotypes near isogenic except at two loci each with two alleles controlling nicotine level were used in a comparison of the four homozygous allelic combinations producing high, high-intermediate, low-intermediate, and low nicotine levels in a "Burley 21" background. Putrescine *N*-methyltransferase and quinolinic acid phosphoribosyltransferase activities in root tissue of these four genotypes were proportional to leaf nicotine level, whereas *N*-methylputrescine oxidase activity in root tissue differed in proportion and ranking. Quinolinic acid phosphoribosyltransferase activities in leaf tissue were lower than in roots, but no differences were found among the four genotypes. The homozygous recessive alleles at either locus affect levels of all three enzyme activities examined in roots. Each locus seems to be involved in regulation of nicotine metabolism, but whether directly as a regulatory locus or indirectly through the metabolic product of a structural locus is not known. No difference was observed between enzymic oxidation of putrescine and *N*-methylputrescine by leaf and root extracts of Burley 21 (a high-nicotine, low-nornicotine genotype) and a high-nornicotine cultivar, "Robinson Medium Broadleaf." Putrescine was utilized as a substrate to a greater extent than *N*-methylputrescine by leaf extracts compared with root extracts of both cultivars. It was concluded that genetic differences in levels of nicotine and nornicotine were not due to differences in enzymic oxidation of these two precursors during alkaloid biosynthesis. (Auth. Abs.)

80–1382. Schneider, R. E.; Bishop, H.; Hawkins, C. F. **Plasma Propranolol Concentrations and the Erythrocyte Sedimentation Rate.** *British Journal of Clinical Pharmacology* 8(1): 43-47, July 1979.

The plasma propranolol concentrations after a single oral dose of 40 mg were measured in 25 patients with rheumatoid arthritis and compared with those of 16 patients with Crohn's disease from a previous study. Thirteen healthy volunteers were used as controls. In both diseases some high and some low values occurred. This scatter did not correlate with any symptoms or biochemical or hematological data other than with the erythrocyte sedimentation rate (ESR). Both sets of patients were therefore separated into two groups depending on whether or not their ESRs were above or below 20 mm/1 hour. In both diseases the plasma propranolol concentrations of the patients with a raised ESR were significantly higher than the controls as well as those of the low ESR group. In rheumatoid arthritis, the plasma propranolol concentrations of the patients with a low ESR did not differ from those of the controls, but in Crohn's disease they remained significantly higher. In one patient with Crohn's disease there was a dramatic rise in propranolol concentrations during an exacerbation (ESR 91 mm/1 hour) compared with those during a remission (ESR 20 mm/1 hour). A difference in smoking habits did not seem to have been responsible for the difference in plasma propranolol concentrations. (Auth. Abs.)

80–1383. Sipal, Z.; Ahlenius, T.; Bergstrand, A.; Rodriquez, L.; Jakobsson, S. W. **Oxidative Biotransformation of Benzo(*a*)pyrene by Human Lung Microsomal Fractions Prepared From Surgical Specimens.** *Xenobiotica* 9(10): 633-645, 1979.

Microsomal fractions were prepared from 15-50g specimens of human lung tissue (mostly alveolar) obtained at surgical resections of 13 middle-aged male patients suffering from different pulmonary tumors. Marker enzyme assays indicated that the fractions contained about 25 percent of the endoplasmic reticulum of the homogenate and about 10 percent of its mitochondrial membranes. The content of cytochrome b_5 corresponded to that of rodent lung microsomes, whereas the apparent content of cytochrome P-450 was much lower. The extent of benzo(*a*)pyrene metabolism varied 13-fold between individuals in the group and was not detectable in about 40 percent of the cases. The dihydrodiols, as percent of total metabolites formed, were higher than in laboratory animals, the 7,8-dihydrodiol in most cases amounting to more than 40 percent of total dihydrodiols. The apparent rate of hydroxylation was stimulated by 1 mM 2-diethylaminoethyl 2,2-diphenylvalerate and by 1 mM 1,2-oxy-3,3,3-trichloropropane, but inhibited moderately by 0.1 mM metyrapone and extensively by 0.05 mM 7,8-benzoflavone. Ethoxyresorufin deethylation qualitatively paralleled benzo(*a*)pyrene hydroxylation among individuals. (Auth. Abs.)

80–1384. Suzuki, K.; Minei, L. J.; Johnson, E. E. **Effect of Nicotine Upon Uterine Blood Flow in the Pregnant Rhesus Monkey.** *American Journal of Obstetrics and Gynecology* 136(8): 1009-1013, April 15, 1980.

Acute effects of nicotine upon the uterine blood flow, blood pressure, maternal and fetal acid—base state, and oxygenation were determined in eight pregnant rhesus monkeys near term. Nicotine was infused intravenously to the mother in a dose of 100 μg/kg of body weight/min over 20 min. The flow rate was measured from the use of the electromagnetic flowmeter. A significant ($p<0.01$) decrease in the uterine arterial blood flow rate, as much as 38 percent of the control value, was observed during the first 15 min of the infusion while aortic pressure increased by 14 percent. The mean fetal arterial pH fell by as much as 0.100 ± 0.001 at 30 min and 0.110 ± 0.001 at 45 min following the start of the infusion ($p<0.01$) producing acidosis. A marked ($p<0.01$) fall in fetal arterial partial pressure of oxygen by 13.0 ± 1.0 mm Hg was noted at 15 min when the mean uterine blood flow fell by 38 percent, resulting in hypoxia. Considered together with previously reported data, the present investigation appears to indicate that the adverse effects of nicotine to the fetus are due to the combined effects of the reduced blood flow and the nicotine transmitted to the fetus. (Auth. Abs. Mod.)

80–1385. Travis, C. C.; Etnier, E. L.; Kirkscey, K. A. **Carcinogenic Risk of Lead-210 and Polonium-210 in Tobacco Smoke—A Selected, Annotated Bibliography.** Oak Ridge National Laboratory, Publication No. ORNL-5411, May 1978, 32 pp.

This bibliography is concerned with the possible carcinogenic risk to man from the presence of lead-210 and polonium-210 in tobacco smoke. It includes a data base on such topics as background levels of lead-210 and polonium-210 in tobacco and tobacco smoke, tobacco plant uptake of lead-210 and polonium-210 from soil, metabolic models, and dose estimates. This data base should be of interest to those concerned with assessing the health effects resulting from the emanation of radon-222 from natural and technologically enhanced sources. (Auth. Abs.)

80–1386. Ulus, I. H.; Wurtman, R. J. **Selective Response of Rat Peripheral Sympathetic Nervous System to Various Stimuli.** *Journal of Physiology* 293: 513-523, 1979.

The induction of tyrosine hydroxylase, a catecholamine-synthesizing enzyme, in sympathetic ganglia and adrenal medullae was utilized to explore the central and peripheral mechanisms through which choline, various environmental stresses, and drugs (including nicotine) that alter blood pressure or central neurotransmission affect various portions of the sympathetic nervous system. Male Sprague-Dawley rats received each treatment chronically, and enzyme activity was measured in the superior cervical, stellate, and coeliac ganglia and in the adrenal medullae. Nicotine (1.0 mg/kg, subcutaneous) and other drugs were dissolved in saline and injected daily for 4 days. A significant difference in tyrosine hydroxylase activity in all ganglia and in the adrenal medullae was found in rats receiving nicotine as compared with controls. (Auth. Abs. Mod.)

80–1387. Ved Brat, S. S.; Sisken, J. E.; Anderson, R. L. **The Effects of Nicotine on Cell Division of HeLa Cells.** *European Journal of Cell Biology* 19(3): 250-254, August 1979.

A time lapse cinemicrographic study shows that, at low concentrations, nicotine can speed up cytokinesis and, at high concentrations, prolong the duration of metaphase in HeLa cells. Based upon the known effects of the alkaloid in other systems, it appears that nicotine may stimulate microfilament activity and inhibit microtubule function by increasing the level of cytosolic calcium ions. (Auth. Abs.)

80–1388. Weber, A.; Fischer, T.; Grandjean, E. **Passive Smoking in Experimental and Field Conditions.** *Environmental Research* 20: 205-216, 1979.

In a first study, subjects were exposed for 1 hour to constant cigarette smoke concentrations corresponding to 5 or 10 ppm carbon monoxide (CO). Annoyance, subjective eye irritations, and eye blink rate increased in both conditions during the first 30 min of exposure. Respiratory frequency and heart rate variability were not altered. In a second study, the air pollution due to tobacco smoke and its irritating effects were investigated in four restaurants. The change in carbon monoxide (ΔCO) as well as the change in nitric oxide (ΔNO)—expressing the differences between indoor and outdoor concentrations—were shown to be adequate parameters for evaluating air pollution due to tobacco smoke. Subjective irritations of guests show a dose—response relation to ΔCO values ($p<0.01$). Smokers indicated eye irritations more frequently than nonsmokers ($p<0.05$). From the results of both investigations, it can be concluded that a degree of air pollution due to tobacco smoke corresponding to less than 2 ppm ΔCO can be considered as low and tolerable for healthy people. This conclusion, however, is not valid for small children, elderly people, or for individuals with impaired health, e.g., asthmatics and chronic bronchitics, whose sensitivity to tobacco smoke may be greater or different. (Auth. Abs. Mod.)

80–1389. Wilkinson, E. J.; Hause, L. L.; Sasse, E. A.; Pattillo, R. A.; Milbrath, J. R.; Lewis, J. D. **Carcinoembryonic Antigen and L-Fucose in Malignant and Benign Mammary Disease.** *American Journal of Clinical Pathology* 73(5): 669-675, May 1980.

A prospective study of 123 patients was undertaken to determine whether carcinoembryonic antigen (CEA) or L-fucose could be used as tumor markers to distinguish patients who had mammary cancer (41) from patients who had benign mammary disease (41) and from patients who did not have mammary cancer (41). The predictive value of positive test results for both L-fucose and CEA was lower for patients who smoked than for those who did not. The sensitivities of CEA and L-fucose indicators are both below 55 percent. There were frequent false-positive and false-negative results. A substantial overlap in the levels of CEA and L-fucose was found for the patients who had mammary cancer and those who had benign mammary disease. CEA and L-fucose performed similarly in distinguishing cancer from benign disease. For patients who smoked, however, the predictive value of L-fucose was only 44 percent. The effect of smoking on L-fucose levels may account for discrepancies in L-fucose data reported in previous studies of mammary cancer. Neither CEA nor L-fucose were found to be of value in screening for mammary cancer. (Auth. Abs.)

See also, **80–1505**

MORTALITY AND MORBIDITY

80–1390. Alexander, L. L. **Increased Mortality Rates Among Smokers.** *Journal of the National Medical Association* 72(5): 439-440, May 1980.

Reports of the State Mutual Life Assurance Company of America covering a 15-year period reveal that their nonsmoking policyholders survived longer, experienced fewer deaths, and received less dollar indemnification. Data from a 1979 study by the company indicate higher mortality differentials between smokers and nonsmokers from all causes of death, including mental disorders, suicides, and accidents. These differentials appear to be similar to those reported by the Surgeon General. The latter report shows that the life expectancy of a 30- to 35-year-old male, two-pack-a-day smoker is 8-9 years shorter than that of a nonsmoker. Applied to smokers of all ages, these differences reflect death rates more than twice as great as those of nonsmokers. The new data, in addition to the data from the Surgeon General's report, should provide sufficient motivation for smokers to stop.

80–1391. Athanasou, J. A. **Smoking and Absenteeism. (Letter).** *Medical Journal of Australia* 1: 234, 236, March 24, 1979.

As part of an ongoing study into the effects of smoking behavior on absenteeism, mean values for the duration of sickness absence in an industrial population are reported. The 424 subjects were in the age group 15-67 years, and all had passed a pre-employment medical examination. Current smoking habits were noted at the medical interview. There were 88 male and 131 female nonsmokers/ex-smokers, and 133 male and 72 female smokers. Duration of sickness absence was recorded over a 2-year period. The average duration of sickness absence (days/month) is higher in both male and female smokers. Although this was consistent with previous reports, the differences between groups examined were statistically significant ($p<0.01$) only for male smokers. As a positive risk factor, smoking accounts for a maximum 7 percent of the variance in absenteeism among male subjects and around 1-2 percent overall for the population studied. Even with the effects of aging excluded, this proportion remains around 1-1.5 percent for both males and females. Both medical and nonmedical factors can also be implicated in absenteeism; moreover, the synergistic action of factors such as non-narcotic analgesic use, alcohol consumption, or exposure to dust, with smoking, should not be overlooked.

80–1392. Breslow, L.; Enstrom, J. E. **Persistence of Health Habits and Their Relationship to Mortality.** *Preventive Medicine* 9(4): 469-483, July 1980.

This paper explores the relationship of seven personal health practices and subsequent mortality in the 9.5 years between an initial survey of 6,928 adults made in Alameda County, California, in 1965 and a followup survey in 1974. The seven health practices are never smoking cigarettes, regular physical activity, moderate or no use of alcohol, 7-8 hours sleep/day regularly, maintaining proper weight, eating breakfast, and not eating between meals. Over the 9-year period there was a substantial drop in cigarette smoking, more than one-fifth among men and about one-sixth among women, a trend consistent with data for the United States as a whole. The age-adjusted mortality rates for male vs. female never-smokers, ex-smokers, and current smokers were 0.091 vs. 0.70, 0.104 vs. 0.120, and 0.154 vs. 0.106, respectively. When accumulated to form a health practice score from 0 to 7, the number of health practices showed a striking inverse relationship with age-adjusted mortality rates, especially for men. Men following seven health practices had a mortality rate only 28 percent that of men following zero to three health practices. Women following seven health practices had a mortality rate 43 percent that of women following zero to three health practices. Both the health practices themselves and their relationship to mortality are shown to be reasonably stable over the 9.5-year period of followup. These results lend support to the hypothesis that good health practices and not the initial health status of the survey respondents are largely responsible for the observed mortality relationships. These and other methodological issues are explored. (Auth. Abs. Mod.)

80–1393. Cohen, B. L.; Lee, I.-S. **A Catalog of Risks.** *Health Physics* 36(6): 707-722, June 1979.

Information on risks is collected from various sources and converted into loss of life expectancy throughout life and in various age ranges. For instance, years of life expectancy lost due to amount of cigarette consumption; duration of smoking habit; and type, amount, and inhalation or noninhalation of tobacco product smoked are tabulated for both men and women, as is loss of life expectancy in years for smokers and nonsmokers of several age groups. Other risks included are radiation, accidents of various types, various diseases, overweight, alcohol, coffee, saccharin, oral contraceptives, occupation, socioeconomic factors, marital status, geography, serving in U.S. armed forces in Vietnam, catastrophic events, energy production, and technology in general. Information is also included on methods for reducing risks, risks in individual actions, "very-hazardous" activities, and priorities and perspective. Risks of natural and occupational radiation and exposure to radioactivity from the nuclear industry are compared with risks of similar or competing activities. (Auth. Abs. Mod.)

80–1394. Hammond, E. C.; Selikoff, I. J.; Seidman, H. **Asbestos Exposure, Cigarette Smoking and Death Rates.** *Annals of the New York Academy of Sciences* 330: 473-490, 1979.

The effects of cigarette smoking and exposure to asbestos dust were studied in 12,051 members of the Asbestos Insulation Workers Union. Death rates among this group were compared with data from 73,763 persons in an American Cancer Society epidemiologic study. Total death rates (from all causes, cardiovascular,noninfectious pulmonary, and asbestosis) and death rates from cancer in particular were elevated in asbestos workers as compared with controls (mortality ratios were 1.70 and 3.52, respectively). Mortality ratios for neoplasms of lung, larynx, pharynx, buccal cavity, esophagus, colon-rectum, kidney, stomach, prostate, bladder, and pancreas and, for mesothelioma, are presented. The smoking habits of asbestos workers were analyzed and mortality ratios relative to individuals who never smoked regularly were calculated. The mortality ratios for deaths from all causes were 1.98 for men who currently smoked >20 cigarettes/day, 1.89 for those who currently smoked <20 cigarettes/day, 1.29 for pipe and cigar smokers, and 1.00 for men who never smoked regularly. The mortality ratio for ex-cigarette smokers who previously smoked >20 cigarettes/day and who had given up the habit for <5 years was 2.03. For lung cancer, the mortality ratio was 10.40 for men who currently smoked >20 cigarettes/day. The asbestosis death rates of men who currently smoked >20 cigarettes/day were 2.8 times as high as the asbestosis death rate of men who never smoked regularly. It is clear that cigarette smoking greatly increases the risk of an asbestos worker dying from asbestosis. None of the men who had never smoked regularly died of cancer of the esophagus, larynx, pharynx, or buccal cavity. The expected number of deaths from these cancers was 3.6 for men who never smoked regularly. This suggests that in the absence of exposure to tobacco, exposure to asbestos dust may have little or no influence on death rates from such cancers. For men who never smoked cigarettes regularly there were eight observed deaths and eight expected deaths from pleural mesothelioma, so it appears that cigarette smoking has little or no effect upon death rates from pleural mesothelioma. A total of 276 lung cancer deaths occurred among the asbestos workers whose smoking habits were known, whereas only 6.0 were expected had their age-specific lung cancer death rates been the same as those for control group subjects who never smoked regularly. Thus, the mortality difference was 270.0 and the mortality ratio was 46.23. The mortality ratios for lung cancer for various smoking categories were 87.36 for asbestos workers who currently smoked >20 cigarettes/day; 50.82 for those who currently smoked <20 cigarettes/day; 36.56 for ex-cigarette smokers; 7.02 for pipe/cigar smokers; and 5.33 for asbestos workers who never smoked regularly. Data from the study suggest a synergistic effect between asbestos exposure and cigarette smoking.

80–1395. Rogot, E.; Murray, J. L. **Smoking and Causes of Death Among U.S. Veterans: 16 Years of Observation.** *Public Health Reports* 95(3): 213-222, May-June 1980.

In a 16-year mortality followup of some 293,000 insured U.S. veterans, specific causes of death were studied in relation to smoking status. The main results confirmed earlier findings. Mortality ratios for cigarette smokers as compared with nonsmokers were 1.73 for all causes of death, 1.58 for all cardiovascular diseases, 2.12 for all cancers, and 4.31 for all respiratory diseases. The highest ratios (those greater than 5.0) were observed for cor pulmonale, aortic aneurysm, emphysema and bronchitis, cancer of the pharynx, cancer of the esophagus, cancer of the larynx, and cancer of the lung and bronchus. The greatest excess in deaths in terms of observed numbers minus expected was found for the cardiovascular diseases, in particular for coronary heart disease. Mortality ratios for ex-cigarette smokers who had stopped smoking for reasons other than physicians' orders were much lower compared with nonsmokers than the mortality ratios for current cigarette smokers: 1.21 for all causes, 1.15 for all cardiovascular diseases, 1.39 for all cancers, and 2.08 for all respiratory diseases. For most causes of death, the mortality ratios for ex-cigarette smokers who had stopped smoking for reasons other than physicians' orders varied inversely with the number of years of cessation. For some diseases, the mortality risk for the ex-cigarette smoker returned to normal almost immediately after the cessation of smoking, whereas for others, the return to normal was more gradual. The first group included stroke and the combined category of influenza and pneumonia; the second group included cardiovascular diseases as a whole and coronary heart disease. For still other diseases, although the mortality ratio declined with the length of time smoking was discontinued, substantial excess risks remained even after 20 years of cessation. In this third group were aortic aneurysm, bronchitis and emphysema, and lung cancer—diseases with very high mortality ratios for current cigarette smokers. Parkinson's disease remained the one disease that clearly exhibited a negative association with cigarette smoking. (Auth. Abs.)

See also, **80–1397, 80–1406, 80–1437, 80–1445, 80–1456, 80–1464**

NEOPLASTIC DISEASES

80–1396. Geser, A.; Charnay, N.; Day, N. E.; de-The, G.; Ho, H. C. **Environmental Factors in the Etiology of Nasopharyngeal Carcinoma: Report on a Case-Control Study in Hong Kong.** In: de-The, G.; Ito, Y. (Editors). *Nasopharyngeal Carcinoma: Etiology and Control.* IARC Scientific Publications No. 20, Lyon, International Agency for Research on Cancer, 1978, pp. 213-229.

The purpose of the retrospective study reported here was to compare nasopharyngeal carcinoma (NPC) patients and age- and sex-matched controls in order to determine whether NPC patients had experienced anything different or had lived in an environment different from controls. Socioeconomic status, demography, dietary habits, drinking and smoking habits, health and hygiene, air pollution exposure, and weaning habits were investigated. Socioeconomic status, religious affiliation, and health were significantly different for NPC patients as compared to controls. Cigar and pipe smoking were equally frequent among NPC patients and controls, so that no association between NPC and smoking could be determined.

80–1397. Hammond, E. C.; Seidman, H. **Smoking and Cancer in the United States.** *Preventive Medicine* 9(2): 169-173, March 1980.

Based on the mortality experience of over one million Americans who have been followed in a prospective epidemiologic study since 1959, it is estimated that from 25 to 35 percent of cancer mortality in the U.S. male population and from 5 to 10 percent in the female population are mainly due to smoking of tobacco products and cigarettes. These estimates are remarkably close to those derived from other U.S. studies and similar studies in the United Kingdom and Japan. While it is clear that several factors contribute to the causation of smoking-related cancer mortality, it is unlikely that the excess in deaths would occur in the absence of tobacco usage. The contribution of smoking is hence considered as the preponderant risk, and one that could be controlled by combined actions of individuals' responses to education, legislation, and modification of cigarettes toward less hazardous characteristics. (Auth. Abs.)

80–1398. Hoover, R. N.; Strasser, P. H. **Artificial Sweeteners and Human Bladder Cancer.** *Lancet* 1(8173): 837-840, April 19, 1980.

Patients with cancer of the urinary bladder (n = 3,010) and controls (n = 5,783) drawn from the general population of 10 geographic areas of the United States were interviewed. Subjects who reported ever having used artificial sweeteners (AS), or artificially sweetened foods or beverages, showed no elevation in risk. However, positive associations between various measures of use of artificial sweeteners and risk of bladder cancer were seen in several subgroups. Inconsistencies in the data suggest that the positive associations may be due to chance, but it is noteworthy that the subgroups were those chosen, a priori, to test hypotheses derived from laboratory experiments. To search for potentiation of known bladder carcinogens, a high-risk group of male heavy smokers (>40 cigarettes/day) were examined. Within this group, consumers of diet drinks or table-top AS were at higher risk than those who never used AS. There were gradations in risk suggestive of dose—response for diet drinks, but not for table-top AS. These findings were restricted to the heavy smokers; nonsmoking men and those who smoked ≤40 cigarettes/day showed no significant trends in relative risk with increased use of either table-top AS or diet drinks. In fact, the nonsmoking males showed a nonsignificant decreasing trend with increased daily use of diet drinks. Among the female heavy smokers (>20 cigarettes/day), the heavier users of table-top AS and diet drinks (≥2 servings/day) also showed higher relative risks than those who never used AS. The trend in relative risk was significant for diet drinks. No consistent trends were seen for either form of AS among women who smoked ≤20 cigarettes/day. (Auth. Abs. Mod.)

80–1399. Infante, F.; Voirol, M.; Brahime-Reteno, O.; Raymond, L.; Hollenweger, V.; Loizeau, E. **Consommation d'Alcool, de Tabac et Alimentation Dans les Cancers du Foie et les Cirrhoses. Resultats Preliminaires. [Consumption of Alcohol, Tobacco and Food in Liver Cancers and Cirrhoses. Preliminary Results.]** *Schweizerische Medizinische Wochenschrift* 110(22): 875-876, 1980, French.

In Geneva, the consumption of food among cirrhotic patients, whether male or female, and among male patients with primary liver cancer (PLC), is virtually the same as in normals. Alcohol consumption is very high in all three groups. The heaviest drinkers are the cirrhotic males, but the highest patient/control ratio is found among cirrhotic females. Total alcohol ingestion during life appears to be higher in patients with PLC plus cirrhosis than in those with PLC without cirrhosis. Tobacco consumption by male cirrhotic and PLC patients is the same as in controls. Conversely, cirrhotic females smoke seven times as much as their standardized controls (92±62 vs. 13±20 kg, lifetime consumption). (Auth. Abs.)

80–1400. Mihatsch, M. J.; Manz, T.; Knuesli, C.; Hofer, H. O.; Rist, M.; Guetg, R.; Rutishauser, G.; Zollinger, H. U. **Phenacetinabusus III. Maligne Harnwegtumoren bei Phenacetinabusus in Basel 1963-1977. [Phenacetin Abuse III. Malignant Urinary Tract Tumors From Phenacetin Abuse in Basel During 1963-1977.]** *Schweizerische Medizinische Wochenschrift* 110(7): 255-264, 1980, German.

In the course of a 15-year retrospective study, 451 malignant tumors of the lower urinary tract were observed at autopsy or biopsy in 442 inhabitants of Basel (307 men and 135 women). Of the 442 patients, 69 were abusers of phenacetin-containing analgesics. Neoplasms of the lower urinary tract (renal pelvis, bladder, ureters, and urethra) were 7-89 times more frequent in phenacetin abusers than in nonabusers. A survey of patient smoking habits revealed that 98 (22.1 percent) were smokers. In phenacetin-abusing patients with renal pelvis and ureter neoplasms, smoking was as prevalent as in nonabusers. In 12 out of 41 patients (29.2 percent) with bladder tumors, both phenacetin and nicotine abuse were present. Among the nonphenacetin-abusing bladder tumor patients, 62 (19.7 percent) admitted to being smokers. It is concluded that smoking had no effect on the statistically significant incidence of bladder neoplasms in phenacetin abusers.

80–1401. Miller, A. B. **Identification of Adults at High Risk of Lung Cancer.** *Canadian Medical Association Journal* 122(9): 985-987, May 10, 1980.

Data from several studies are examined to consider the risk of lung cancer for 45- to 64-year-old smokers over the next 5 years in order to assess the possibilities for prevention. Data from other studies indicate that smokers exposed to occupational agents (e.g., asbestos, radioisotopes, and nickel) are at increased lung cancer risk. It is stated that screening or antismoking programs are not likely to be effective in reducing lung cancer in high-risk groups (e.g., foundry workers); therefore, the development of measures to prevent lung cancer in such groups is an important and immediate responsibility.

80–1402. National Cancer Institute. **Second Symposium on Epidemiology and Cancer Registries in the Pacific Basin.** National Cancer Institute Monograph 53. U.S. Department of Health, Education, and Welfare, NIH Publication No. 79-1864, November 1979, 208 pp.

The current status of individual cancer registries and population-based studies of the Pacific Basin is discussed in this monograph. One of these studies, the New Zealand registry report, states that in 1976 a question regarding cigarette smoking was included in the national census. Of each 100 males, 38 reported they had never smoked regularly, 22 were former smokers, and 40 were current smokers. In each 100 females, 57 had never smoked regularly, 12 were former smokers, and 31 were current smokers. A cigarette smoking question has now also been incorporated in the New Zealand standard cancer notification form. Another study was concerned with the ethnic differences in smoking and drinking habits of the five major ethnic groups in Hawaii (i.e., Caucasians, Japanese, Chinese, Filipinos, and Hawaiians) by means of questionnaire data obtained from a representative sample of 8,636 Hawaii residents. For men, lifetime cigarette use was greatest among Causasians and lowest among Chinese and Filipinos. For women, smoking cigarettes was highest among Caucasians and lowest among Chinese.

80–1403. Rothman, K. J. **The Proportion of Cancer Attributable to Alcohol Consumption.** *Preventive Medicine* 9(2): 174-179, March 1980.

Existing evidence leaves little room for doubt that alcoholic beverages are carcinogenic. Alcohol is a risk factor for cancer of the oral cavity, larynx, pharynx, esophagus, and possibly the liver. The extent to which carcinogenicity is attributable to the alcohol, or to other components of some or all of the beverages that contain it, is unclear. The alcohol effect, at least for cancers of the upper aerodigestive tract, depends on tobacco consumption. In one oral cancer study, the risk ratio estimate for nonsmoking, nonalcohol drinking subjects was 1.00. For heavy alcohol drinkers (>1.5 oz/day), or heavy smokers (≥40 cigarettes/day), the corresponding risk ratio estimates were 2.33 and 2.43. However, the combined effect of heavy drinking and heavy smoking resulted in a risk ratio of 15.50. About 3 percent of all U.S. cancers in 1974 are attributable to alcohol; the small proportion and the dependence on tobacco mitigate against antidrinking campaigns for the primary prevention of cancer. It is likely that antismoking campaigns would be more effective than antidrinking campaigns in preventing alcohol-related cancers. (Auth. Abs. Mod.)

80–1404. Schmidt, F. **Zwangsrauchen und Krebs. [Passive Smoking and Cancer.]** *Medizinische Klinik* 74(51/52): 1967-1973, December 21, 1979, German.

Numerous research results are briefly summarized to support the conclusion that passive smoking is a genuine health hazard and not merely a nuisance. More than 40 cancerogenic substances detected thus far in tobacco smoke are believed to present sufficient proof of a health hazard to nonsmokers.

Particular attention is devoted to cancerogenic nitrosoamines which are potentially hazardous in doses as low as 1 ppm. At least a dozen nitrosoamines have been identified in tobacco smoke. Numerous experiments are cited in which cancers were induced in animals by passive smoking. Passive smoking cancers were induced in animals by passive smoking. Passive smoking also increases the total cancerogenic burden of men. The need for preventive measures for the protection of nonsmokers, particularly those at work, is stressed.

80–1405. Selikoff, I. J.; Hammond, E. C. **Asbestos and Smoking.** *Journal of the American Medical Association* 242(5): 458-459, August 3, 1979.

A new study confirms previously published findings that asbestos workers who smoke cigarettes have a 90 times greater risk of getting lung cancer than men who neither smoke nor work with asbestos. Asbestos insulation workers (n = 12,051) were followed up from 1967 to 1976, the end of which period culminating in at least 20 years work in the industry. Of the 8,220 workers whose smoking histories were known, 6,841 were cigarette smokers. Death rates for lung cancer (per 100,000 man-years) were found to be as follows: 11.3 for men who neither worked with asbestos nor smoked cigarettes, 58.4 for men who worked with asbestos but did not smoke, 122.6 for cigarette smokers who had not worked with asbestos, and 601.6 for smokers who worked with asbestos. The data also showed that the lung cancer mortality was approximately one-third for workers who had stopped smoking for 10 years compared to those who had continued to smoke.

80–1406. Staszewski, J. **Analiza Kohortowa Umieralnosci na Raka Pluc w Polsce. [Analysis of Age Group Mortality From Lung Cancer in Poland.]** *Nowotwory* 28(2): 187-190, April-June 1978, Polish.

Mortality from malignant lung tumors was analyzed in successive age groups based on 1959-1974 Polish statistics. This study was undertaken to determine future trends in lung cancer incidence. Mortality of people born within 5-year periods was evaluated from mortality data of the corresponding age group in 1959, 1964, 1969, and 1974. A linear relationship was found between increasing age and pulmonary carcinoma mortality for both men and women. In 1974, mortality of Polish men peaked at age 60-65, while that of women was maximum at age 70. Within the succession of analogous age groups mortality increases. Presumably, this finding may be the result either of improved methods of carcinoma detection, or of steady increases in exposure to carcinogenic factors, particularly cigarette smoke. In the forthcoming decades, a threefold increase in lung cancer incidence in men is expected if the exposure to cigarette smoke for the youngest age group remains at the same level as that of the group born around 1930. For women, the increase is expected to be even greater than that for men.

80–1407. Stellman, S. D.; Austin, H.; Wynder, E. L. **Cervix Cancer and Cigarette Smoking: A Case-Control Study.** *American Journal of Epidemiology* 111(4): 383-388, April 1980.

The association between cervix cancer and cigarette smoking was examined among 332 white cases and 1,725 white controls. The analysis was restricted to women who had been married and who had either never smoked regularly or had smoked regularly for at least 10 years. Cases were of lower socioeconomic status (SES) and smoked more than controls ($p<0.001$). After adjustment for both age and SES, no significant association was observed between cervix cancer and cigarette smoking. Many variables related both to cigarette smoking and to known risk factors for cervix cancer are sources of confounding and inadequate control, for these variables may cause an overestimation of the effect of smoking. (Auth. Abs. Mod.)

80–1408. Torjussen, W.; Solberg, L. A.; Hogetveit, A. C. **Histopathological Changes of the Nasal Mucosa in Active and Retired Nickel Workers.** *British Journal of Cancer* 40(4): 568-580, 1979.

Histological examinations were made on nasal biopsy specimens from the middle turbinate in 318 active and 15 retired nickel workers and in 57 controls, to study the prevalence of nasal carcinoma or possible precancerous mucosal changes in nickel-exposed individuals. Two nickel workers from the roasting/smelting department (0.6 percent), both employed 28 years at the plant, had nasal carcinoma. Carcinoma in situ was found in the specimen from one of the men with carcinoma. Epithelial dysplasia was found in about 12 percent of active and 47 percent of retired nickel workers. One of the controls, a male carpenter, had dysplasia. These histopathological changes may be precancerous lesions, as they are almost exclusively found in active and retired nickel workers with enhanced risk of nasal carcinoma. Loss of respiratory epithelium and development of squamous epithelium were seen in all groups, with significantly higher incidence in the nickel-exposed groups. The percentage of smokers was nearly equal among active nickel workers and controls, but lower for retired nickel workers. There was no statistically significant difference between the smoker's average tobacco consumption in active nickel workers and controls ($p>0.1$). The two men with nasal carcinoma were both smokers. However, no correlation was found between tobacco smoking consumption and epithelial dysplasia ($r=0.046$, $n=375$; $p>0.1$). The average histological score was, however, higher in smokers than in nonsmokers, for both active and retired nickel workers and for controls. Furthermore, the correlation between histological score and tobacco consumption was statistically significant ($p<0.0001$). Duration of nickel exposure, type of nickel-refining work, and tobacco consumption were the independent variables that, taken altogether, had the highest explanatory values for the histopathological changes. (Auth. Abs. Mod.)

80–1409. Voirol, M.; Infante, F.; Brahime-Reteno, O.; Raymond, L.; Hollenweger, V.; Loizeau, E. **Consommation d'Alcool, de Tabac et de Nutriments Dans les Affections Pancreatiques. Resultats Preliminaires d'une Etude de 1000 Cas. [Consumption of Alcohol, Tobacco, and Nutriments in Pancreatic Diseases. Preliminary Results of a Study of 1,000 Cases.]** *Schweizerische Medizinische Wochenschrift* 110(22): 854-855, 1980, French.

The incidence of cancer of the pancreas is particularly high in Geneva. To determine the possible effects of tobacco, alcohol, and/or diet, the habits of patients with digestive diseases have been studied at the University Hospital of Geneva since 1977. The present study deals only with pancreatic patients. The tobacco, alcohol, and food consumption of these patients was compared with that of a control group randomly selected from the Geneva population. Lifetime brown tobacco consumption of male patients with pancreatic cancer was double that of male patients with pancreatic disease. In the case of female pancreatic cancer patients, total lifetime tobacco consumption was double that of pancreatic disease patients (84 vs. 46 kg, respectively). In addition, brown tobacco consumption was double, and that of blond tobacco was triple that of female pancreatic disease patients. A relationship between chronic pancreatitis and lifetime alcohol consumption was also found. (Auth. Abs. Mod.)

See also, **80–1389, 80–1394**

NON—NEOPLASTIC RESPIRATORY DISEASES

80–1410. Ashley, M. J.; Corey, P.; Chan-Yeung, M. **Smoking, Dust Exposure, and Serum Alpha$_1$-Antitrypsin.** *American Review of Respiratory Disease* 121(5): 783-788, May 1980.

The relationships of smoking and dust exposure to serum alpha$_1$-antitrypsin concentrations were studied in 1,131 male workers who were PiM phenotypes and who were employed in four different environments (i.e., grain elevators, red cedar sawmills, noncedar sawmills, and municipal service). In each work group, current smokers had higher concentrations than nonsmokers or ex-smokers (grain, $p<0.0001$; cedar, $p<0.0001$; noncedar, $p=0.09$; municipal, $p=0.04$). Among current smokers, concentrations were highest in those who smoked the most and for the longest time. Within the smoking categories the concentrations in most instances were greater in workers exposed to the greatest dust concentrations (noncedar and grain workers). A significant positive relationship with duration of employment was found only in one work group (noncedar workers). The independent effects of smoking and dust exposure persisted when the analysis was repeated using only nonatopic workers who were free of chest symptoms. Although both the biologic significance of these variations and the underlying mechanisms require clarification, the findings suggest that extrinsic factors, such as smoking and industrial dust exposure, may influence serum alpha$_1$-antitrypsin concentrations. (Auth. Abs. Mod.)

80–1411. Bouhuys, A.; Macklem, P. T.; Ghezzo, H. R. **Pulmonary-Function Tests in Smokers. (Letters).** *New England Journal of Medicine* 299(15): 836, October 12, 1978.

A response to an article on the relations between structural changes in small airways and pulmonary function tests (*New England Journal of Medicine* 298(23): 1277-1281, June 8, 1978) points out that the authors did not include measurements of maximum expiratory flow at 50 percent of vital capacity (MEF 50 percent) although this measurement has been found to discriminate between smokers and nonsmokers. In reply, the original authors claim that the MEF 50 percent is not particularly sensitive in detecting airway obstruction and that in cigarette smokers other lung function tests become abnormal before it does. The use of a body plethysmograph to measure volume changes was also discussed. See also 78-1183.

80–1412. Burghard, G.; Cooreman, J.; Perdrizet, S.; Metz-Marcy, G.; Welsch, M. **The Tobacco Habit and Respiratory Symptoms Amongst the Adolescents of a French Department.** *Bulletin of the International Union Against Tuberculosis* 54(1): 83-86, March 1979.

In order to study the relationship between frequency of respiratory symptoms and smoking habit in adolescent students in the Bas-Rhin Department, 29,138 participants (48.2 percent male and 51.8 percent female) were interviewed by means of a questionnaire. Among the students, 33.4 and 27.7 percent of the males and females, respectively, were smokers. The percentage of smokers increased with age, from 16.8 percent at 14 years to 41.3 percent at 18 years. Respiratory symptoms (e.g., cough) were more prevalent in smokers than in nonsmokers. Habitual morning cough occurred in 18.7 percent of the occasional smokers (<1 cigarette/day), in 21.3 percent of the 5-9 cigarettes/day smokers, and in 46.1 percent of the >20 cigarettes/day smokers. Two factors influencing the smoking habit in adolescents were particularly in evidence during this study: the smoking habit in parents and the authorization of smoking in scholastic establishments. When both parents were smokers, 45.4 percent of the children smoked. When the parents were nonsmokers, this proportion was 23.6 percent. When smoking in school was forbidden, 23.2 percent of pupils were smokers, as compared with 38 percent when smoking was not forbidden.

80–1413. Gervois, M.; Dubois, G.; Decherf, D. **Alcool, Tabac et Bronchite Chronique. (Lettre). [Alcohol, Tobacco and Chronic Bronchitis. (Letter).]** *Revue d'Epidemiologie et de Sante Publique* 27(2): 155-157, 1979, French.

The relationship between smoking, chronic bronchitis, and alcohol consumption has been studied using a population sample of 6,191 men 25-59 years of age. A questionnaire was administered and clinical examination of the subjects was subsequently conducted at the Lille Health Center over the 1973-1976 period. Chronic bronchitis was defined as daily cough and expectoration continuing for 3 consecutive months within at least a 2-year period. Alcohol abuse was defined as daily consumption of >2 liters of beer or 1 liter of wine, or by clinical symptoms of intoxication. Among moderate smokers (<20 cigarettes/day), alcohol abuse increased the risk of chronic bronchitis in all age brackets, except in the 25-34 year bracket in which the relative risk was 2.3 percent, regardless of alcohol consumption. Among heavy smokers (>20 cigarettes/day),the relative risk increased by alcohol abuse within all age brackets and by a factor of 4.3 in the 25-34 year bracket. The average relative risk for heavy smokers without alcohol abuse was 7.3 percent. The data indicate a significant relationship between alcoholism and chronic bronchitis in smokers, but no clear relationship in nonsmokers. The effects of obesity and sedentary lifestyle on relative risk were found to be negligible. Age had a minimum confounding effect. A synergic effect of tobacco and alcohol on chronic bronchitis is possible but cannot be ascertained.

80–1414. Gulsvik, A. **Prevalence of Respiratory Symptoms in the City of Oslo.** *Scandinavian Journal of Respiratory Diseases* 60(5): 275-285, 1979.

In the first phase of a two-phase cross-sectional survey conducted in Oslo from 1972 to 1974, a questionnaire was mailed to a random sample of 19,998 persons aged 15-70 years. Information was received from 88.7 percent of the sample population. The completion rate for each of 11 questions on respiratory symptoms in the questionnaire varied between 94 and 98 percent. The crude prevalence rates of the symptoms cough in the morning, breathlessness climbing two flights of stairs, and wheezing were 24 percent, 11 percent, and 17 percent, respectively. Only 27 percent of the men and 46 percent of the women in the survey population were nonsmokers. Cough, phlegm when coughing, wheezing, and periods of cough and/or phlegm were on the average 3 to 5 times more frequent in smokers than in nonsmokers. A dose—response relationship between the number of cigarettes smoked and prevalence was observed for these symptoms. Mild and severe grades of breathlessness yielded also increased prevalence rates with increasing number of cigarettes consumed. Among nonsmokers, phlegm was reported more frequently by men, irrespective of age, whereas breathlessness was reported more often by women than by men. In both sexes of nonsmokers, a linear increase in prevalence of symptoms with age was observed for breathlessness, attacks of breathlessness, and coughing in the morning and during the day. (Auth. Abs. Mod.)

80–1415. Haslam, P. L.; Turton, C.W.G.; Lukoszek, A.; Salsbury, A. J.; Dewar, A.; Collins, J. V.; Turner-Warwick, M. **Bronchoalveolar Lavage Fluid Cell Counts in Cryptogenic Fibrosing Alveolitis and Their Relation to Therapy.** *Thorax* 35(5): 328-339, May 1980.

Bronchoalveolar lavage was used to sample inflammatory cells from the lungs of 51 patients with cryptogenic fibrosing alveolitis (CFA) (24 smokers, 12 ex-smokers, and 15 nonsmokers). The smokers with CFA have been compared with 15 smoking control subjects in whom there was no radiographic abnormality or clinical evidence of chronic bronchitis. Significantly lower volumes of lavage fluid were recovered from the smokers with CFA ($p<0.001$) and the fluid contained lower percentages of macrophages ($p<0.01$), reflecting increased percentages of eosinophils ($p<0.001$) and neutrophils ($p<0.01$). Similar changes were seen in the ex-smokers and nonsmokers. There was also an increase in the percentages of lymphocytes when the whole group of CFA patients was compared with the control subjects ($p\leq0.05$). No significant differences were found when patients with "lone" CFA were compared with those having associated systemic disease. The only feature distinguishing smokers from nonsmokers with CFA was the presence of pigmented cytoplasmic inclusions in the macrophages from the smokers ($p<0.001$). However, there were lower numbers of pigmented macrophages in the smoking CFA patients by comparison with the control subjects suggesting either a change in phagocytic capacity or turnover rate in this disease. Profiles of differential cell counts in individual patients showed that increases of eosinophils over 3 percent or neutrophils over 4 percent or both with lymphocyte counts of less than 11 percent related to a poor clinical response to corticosteroids, but lymphocyte percentages greater than 11 percent related to improvement ($p<0.05$). (Auth. Abs.)

80–1416. Holma, B.; Kjaer, G.; Stokholm, J. **Air Pollution, Hygiene and Health of Danish Schoolchildren.** *Science of the Total Environment* 12(3): 251-286, 1979.

A comprehensive investigation forming part of a joint European study under the auspices of "WHO Long-Term Air Pollution Programme" uses 7- to 13-year-old schoolchildren as the target group. The study has included social, housing, hygienic, and epidemic factors as well as family smoking habits. The results indicate that, at exposure to low levels of air pollution, these factors dominate as causes for the impairment of health, especially that of respiratory health. For example, raw and sore throat incidence in children increased from 75.5 to 87.5 percent when the mother smoked. Corresponding percentages for father's smoking habits were 73 and 87. Bronchial symptoms and wheezing were also more frequent in children of smoking than of nonsmoking mothers. (Auth. Abs. Mod.)

80–1417. Horton, F. O., III.; Mackenthun, A. V.; Anderson, P. S., Jr.; Patterson, C. D.; Hammarsten, J. F. **Alpha$_1$ Antitrypsin Heterozygotes (Pi Type MZ). A Longitudinal Study of the Risk of Development of Chronic Air Flow Limitation.** *Chest* 77(2): 261-264, February 1980.

The role of the MZ phenotype as an independent risk factor for the development of chronic airflow limitation (CAL) was studied over a 6-year period. Fourteen men and 14 women identified with PiMZ were matched by age, sex, race, and smoking history (number of cigarettes per day) with a group from the same population with PiM. Symptoms were evaluated by respiratory symptom questionnaires and standard spirometric tests of pulmonary function were performed in 1971 and 1978. There were no statistically significant differences between the mean spirometric parameters of the PiM and PiMZ subjects even when separated into groups of smokers, nonsmokers, and ex-smokers. Smokers and ex-smokers expressed more symptoms in the respiratory questionnaires. There were no significant differences in symptoms observed between PiM and PiMZ and no particular pattern of change from 1971 to 1978. When spirometric test results for PiM and PiMZ were combined, statistically significant changes were seen between smokers and nonsmokers. These results indicate that over a 6-year period PiMZ does not appear to be a significant independent risk factor for the development of CAL.

80–1418. Jedrychowski, W. **Wplyw Czynnikow Zawodowych na Przebieg Naturalnej Historii Przewleklego Niezytu Oskrzeli w Obserwacji Prospektywnej Populacji Przemyslowej. [The Effect of Occupational Factors on the Natural Course of Chronic Bronchitis in a Prospective Observation of an Industrial Population.]** *Medycyna Pracy* 29(4): 335-342, 1978, Polish.

Questionnaires were administered in 1968 and again in 1973 to 248 and 197 male workers, respectively, average age 42.2 years, in a Polish plant. Simultaneously, spirometric and anthropometric measurements were made. Chronic cough and expectoration were considered to be the symptoms of chronic bronchitis. Spirometric measurements of the forced expiratory volume in 1 sec (FEV$_1$) and FEV$_1$ percent were used to evaluate variations in lung function compared to age and smoking habits of the subjects, as well as to air pollutant levels in the workplace. Workplaces were classified into three categories according to pollution level. The percentage of ex-smokers was highest (30 percent) among workers employed in the moderately polluted places (category 2). The percentage of nonsmokers was the same (15 percent) in all three categories. The average percentage of smokers was 69 for the three categories. In nonsmokers and in smokers, respectively, the incidence of chronic bronchitis symptoms in 1968 was 14 and 29 percent, and in 1973, was 7 and 27 percent. The mean FEV$_1$ value in nonsmokers and smokers was somewhat lower in 1973 than in 1968. The barrier syndrome (the FEV$_1$ percent $\leq$70) was found in 23 percent of the ex-smokers, in 15 and 4 percent of the nonsmokers, and in 19 and 16 percent of smokers in 1968 and 1973, respectively. The indicator of FEV$_1$ drop within the 5-year period was 3 and 7 percent for nonsmokers and smokers, respectively. It is concluded that the incidence of chronic bronchitis and its variation over the 5-year observation period correlate mainly with the smoking habits and, to a lesser degree, with the age of the subjects. A direct relationship between air pollution, incidence and persistence of chronic bronchitis symptoms, and FEV$_1$ value drop over the 5-year period was also established, particularly among smokers.

80–1419. McMillan, G.H.G.; Sheers, G.; Pethybridge, R. **A Radiological Follow-Up Study of the Effect of Asbestos in Dockyard Workers at Devonport.** *Journal of the Royal Naval Medical Service* 64(2): 88-104, Summer 1978.

The 10 percent random sample (n = 1,414) of industrial employees at Devonport Dockyard, first examined in 1966, has been re-examined to determine the attack and progression rates of asbestos-related lesions. Ten percent of those with no radiologically apparent asbestos-related lesion in 1966 were found to have such a lesion, and 10 percent of those with a pleural lesion in 1966 had developed pulmonary fibrosis. The overall prevalence of asbestos-related lesions (i.e., simple plaques, calcified plaques, pulmonary fibrosis, mesothelioma) in smokers and ex-smokers was almost three times that of nonsmokers (16.3, 15.0, and 5.9 percent, respectively), and no cases of asbestos-related disease as distinct from pleural abnormalities were found among the nonsmokers. (Auth. Abs. Mod.)

80–1420. Perdrizet, S.; Liard, R.; Cooreman, J. **Symptomes Respiratoires Chez les Enfants d'Age Scolaire, Dans 7 Zones d'Enquete Francaises. [Respiratory Symptoms in Schoolchildren in 7 French Survey Areas.]** *Archives Francaises de Pediatrie* 36(9): 940-948, November 1979, French.

The effect of atmospheric pollution on the frequency of respiratory symptoms in 6- to 10-year-old children was analyzed using the results of a 1975 survey of the children's parents. The survey covered 6,918 schoolchildren (3,537 boys and 3,381 girls) in seven French regions with different pollution levels. The frequency of all respiratory symptoms (cough, wheezing) and other respiratory problems (dyspnea, colds, bronchial complications) except for wet cough was higher in Paris, where the air was the most polluted, than in the other six areas. The prevalence of respiratory symptoms was related to age, allergy, and socioeconomic factors. After adjustments were made for these factors, however, the difference in respiratory symptoms between Paris and the other geographic areas remained statistically significant

($p<0.001$). The proportion of children with respiratory symptoms was higher for those living with a smoker in a small dwelling (three rooms maximum) rather than in a larger dwelling. Only in the case of children with wet cough was the difference significant: 13.8 percent of those who had this symptom lived with a smoker vs. 8.1 percent who had this symptom and did not live with a smoker ($p<0.05$).

80–1421. Petty, T. L.; Silvers, G. W.; Stanford, R. E.; Baird, M. D.; Mitchell, R. S. **Small Airway Pathology is Related to Increased Closing Capacity and Abnormal Slope of Phase III in Excised Human Lungs.** *American Review of Respiratory Disease* 121(3): 449-456, March 1980.

Single-breath nitrogen and static pressure—volume curves were obtained from 29 excised human lungs. The lungs were separated into two groups based on values predicted for closing capacity (CC): "normal" lungs ($n=19$) had normal closing capacities, and "abnormal" lungs ($n=10$) had closing capacities that were larger than normal. Of the 26 deceased subjects in this study for whom smoking histories were available, 15 were smokers. Chi-square analysis, used to examine the relationship between smoking and abnormal CC, revealed a mild association ($p=0.08$) between smoking and abnormal CC. When the mean CC of smokers of more than 10 pack-years (heavy smoking exposure) was compared with that of nonsmoking subjects, the association approached significance ($p=0.07$). There was no correlation between the CC of light smokers and nonsmokers. One subject who rarely smoked a cigar or pipe was considered to have a slight exposure to smoking. There was a significantly greater closing pressure in smokers compared to nonsmokers ($p<0.03$), but there was no significant difference in closing pressure between smokers with light versus heavy exposure to smoking. (Auth. Abs. Mod.)

80–1422. Radwan, L. **Stan Czynnosciowy Drobnych Drog Oddechowych u Chorych na Przewlekle Zapalenie Oskrzeli bez Spirograficznych Objawow Obturacji. [Functional State of the Small Airways in Patients With Chronic Bronchitis Without Spirographic Symptoms of Obstruction.]** *Pneumonologia Polska* 46(12):837-842, 1978, Polish.

Spirometic indicators (vital capacity (VC), forced expiratory volume in 1 sec (FEV_1)), closing lung capacity (CC), nitrogen concentration increase in expiratory air (percent N_2/l), and mid-expiratory flow (MEF) were measured in 30 male and 10 female patients (with an average age of 38.0 and 42.3 years, respectively) with chronic bronchitis but without spirographic symptoms of pulmonary obstruction. The sample included 23 smokers and ex-smokers and 17 nonsmokers. Vital capacity and FEV_1 values were normal in all patients. However, abnormal MEF values at 50 percent of VC (MEF_{50}) and N_2/l percent values were found in 52.5 percent and 42.5 percent of all the patients, respectively. Mean MEF_{50} and N_2/l percent values were more often and more strongly abnormal in cigarette smokers and ex-smokers than in nonsmokers, although the differences were statistically not significant. Among the smokers and ex-smokers, abnormal MEF_{50}, MEF_{75}, CC/VC percent, and N_2/L percent values were found in 56.5, 34.7, 21.7, and 47.8 percent of all cases, respectively. The corresponding figures for nonsmokers were 47.0, 29.4, 0, and 32.5 percent. The results indicate that early functional disorders of the small peripheral airways may be detected in spirographically asymptomatic patients with chronic bronchitis. A somewhat higher degree of functional disorder in the smokers indicates a possible cumulative effect of the factors damaging to small airways.

80–1423. Regli, J. **Die Aufgaben der Bernischen Hohenklinik Heiligenschwendi als Pneumologische Spezialklinik in der Behandlung chronischer obstruktiver und restriktiver Lungenerkrankungen. [Services Offered at the Heiligenschwendi High-Altitude Pneumological Clinic of Bern for Treatment of Obstructive and Restrictive Chronic Lung Diseases.]** *Schweizerische Rundschau fuer Medizin Praxis* 67(48): 1792-1795, November 28, 1978, German.

In the specialized clinic, modern cardiorespiratory equipment permits a comprehensive diagnosis of specific lung diseases by determining lung function changes, blood gases, electrocardiogram, exercise effects, and by performing X-ray, clinical, and bacteriological laboratory tests. The main service offered by the clinic is a highly specialized program of therapy, which includes smoking withdrawal in addition to drug treatment, health information, and patient motivation. Specialized individual and group therapy proceeds in an ideal nonsmoking environment. The patient is actively helped to overcome his or her smoking habit in smoking withdrawal groups under the supervision of a physician. Individual aerosol inhalation treatment and a breathing education program are available. Patients with limited work ability are helped with ergotherapy.

80–1424. Rosenberg, D. M.; Ferrans, V. J.; Fulmer, J. D.; Line, B. R.; Barranger, J. A.; Brady, R. O.; Crystal, R. G. **Chronic Airflow Obstruction in Fabry's Disease.** *American Journal of Medicine* 68: 898-905, June 1980.

Seven patients with Fabry's disease, an x-linked sphingolipid storage disorder, were evaluated for the presence and extent of airway obstruction. Five of the seven had a history of smoking cigarettes (mean 16±4 pack-years): two were current smokers and three had stopped smoking from 1 to 15 years previously. All were found to have significant obstruction to airflow. In addition, evaluation of their airway epithelial cells obtained by bronchoscopy demonstrated that these cells contained inclusion bodies consistent with deposits of ceramide trihexoside, suggesting that part of their functional obstruction to airflow may be secondary to intrinsic airway disease. Although all of the study population had evidence of airflow obstruction, the impairment was much worse in those who smoked, implying that even mild cigarette smoking is particularly hazardous to patients with Fabry's disease. (Auth. Abs. Mod.)

80–1425. Schoenberg, J. B.; Beck, G. J.; Bouhuys, A. **Growth and Decay of Pulmonary Function in Healthy Blacks and Whites.** *Respiration Physiology* 33(3): 367-393, 1978.

Maximum expiratory flow-volume curves were recorded in 3,046 healthy persons, 1,229 blacks and 1,817 whites, age 7 and over—a representative population of lifetime nonsmokers except for some black adult males, who were healthy smokers or ex-smokers. All but 19 of the 120 black male smokers were light smokers ($\leq$ 10 pack-years). Regression equations for lung function measurements as a function of age, height, and weight were computed for eight subgroups (by sex and race, and for children or adults). Objective statistical criteria were used to select the optimal equations. Simple linear regressions on age and height are inaccurate, in particular for young adults and for the elderly. Weight affects most function measurements: lung function first increases with weight (muscularity effect) and decreases with further increase in weight (obesity effect). The regression equations allow more accurate prediction of normal lung function. In addition, the lower 95 percent confidence limits are closer to the predicted values and are valid regardless of height, weight, and age within each subgroup. Comparisons of lung function residuals in healthy adult subjects with and without histories of occupational exposures showed no systematic or significant differences. Comparisons of residuals in healthy nonsmoking children according to the number of smokers in the home showed minimal differences between children living in homes with 0, 1, or more smokers. (Auth. Abs. Mod.)

80–1426. Shephard, R. J.; Collins, R.; Silverman, F. **Responses of Exercising Subjects to Acute "Passive" Cigarette Smoke Exposure.** *Environmental Research* 19: 279-291, 1979.

Responses to 2 hours of "passive" cigarette smoke exposure have been tested in 23 healthy young men and women (all of whom were nonsmokers) who were performing intermittent bicycle ergometer work sufficient to increase respiratory minute volumes by a factor of 2.5. A simple crossover design compared data with reactions to sham exposures of similar duration. Cigarettes were smoked by a standard machine; chamber carbon monoxide concentrations were 20 (moderate dose) or 31 ppm (heavy exposure). Symptoms were much as in moderate exposures without exercise. The main complaints were of odor and eye irritation. Cough, nasal discharge or stuffiness, and throat irritation were also reported, but wheezing, shortness of breath, and tightness in the chest were both uncommon and unsupported by objective evidence of bronchospasm. A small increase of tidal volume and respiratory minute volume seemed due to anxiety rather than airway irritation. Static lung volumes were unchanged, but there were small (3-4 percent) decreases of forced vital capacity, forced expiratory volume in 1 sec, and the maximum flow at 25 and 50 percent of the vital capacity. The changes of dynamic lung volumes were of the order anticipated from the "cigarette equivalent" encountered by the passive smoker ($<$1/2 cigarette in 2 hours). (Auth. Abs.)

80–1427. Sinclair, D.J.M.; Ingram, C. G. **Controlled Trial of Supervised Exercise Training in Chronic Bronchitis.** *British Medical Journal* 280(6213): 519-521, February 23, 1980.

In a controlled trial of exercise retraining in patients with severe chronic bronchitis, 33 subjects were followed for a mean period of 10.3 months. All the patients had been heavy cigarette smokers. Daily training comprised a 12-minute walk and simple stair climbing exercises. The subjects in the exercise group showed a highly significant improvement in their walking distance, attaining a maximum increase of 24 percent after 8 to 12 months. There was also considerable subjective improvement. The control group did not improve. No significant changes in cardiorespiratory function or muscle strength were seen. More patients in the exercise group gave up smoking than in the control group, but analysis of the 12-minute distance results according to smoking habit showed no statistical differences; cessation of smoking was therefore not a major factor in the overall improvement in the exercise group. (Auth. Abs. Mod.)

80–1428. Suzuki, S. **Kitsuen to Shin hai Kino. 3. Kokyuki Kei no Shuhosu oto. [Smoking and Mechanical Response of the Respiratory System.]** *Japanese Journal of Thoracic Diseases* 15(9): 565-570, 671, September 1977, Japanese.

The effects of smoking on pulmonary function were investigated by analyzing the mechanical frequency response of the lung and thorax in 13 nonsmokers, 7 ex-smokers, and 14 smokers. Frequency dependence of dynamic compliance was measured by a new method which can analyze it from only one period of flow and transpulmonary pressure curves. Respiratory resistance was measured by forced oscillation technique (3 Hz), and frequency dependence was determined by the difference between respiratory resistance of 3 Hz and 7 Hz. Results of the study indicated that smokers showed more frequency dependence of dynamic compliance than nonsmokers. There were no differences among three groups in regard to respiratory resistance and its frequency dependence. After smoking a filter cigarette, all groups showed more frequency dependence of dynamic compliance and an increment of respiratory resistance. Inhalation of a bronchodilator (orciprenaline) aerosol decreased respiratory resistance which had increased after smoking a cigarette, but did not change frequency dependence of dynamic compliance following smoking a cigarette. These results suggest that chronic effects of smoking exist only in the small airways, and the inhalation of cigarette smoke causes acute changes of both large and small airways. (Auth. Abs.)

80–1429. Toyama, T.; Kagawa, J. **Prevalence of Chronic Bronchitis Symptoms in Japan. A Review of the Nation-Wide**

Surveys on Prevalence of Pulmonary Symptoms Using Standard Questionnaire. *Keio Journal of Medicine* 27: 69-87, 1978.

This paper reviewed studies and surveys on the prevalence of various respiratory symptoms in 22,560 persons in Japan between 1965 and 1973, using a Japanese version of the standard British Medical Research Council questionnaire on chronic bronchitis. Respiratory symptoms were found to be less prevalent in Japan than in England and the United States. The incidence of phlegm was lowest among nonsmokers and increased with the amount smoked. The prevalence of phlegm ranged from 10.1 percent for nonsmokers to 19.5 percent for those who smoked ≥ 25 cigarettes/day; comparable percentages for persistent phlegm were 13.5 and 27.2 and for persistent cough and phlegm, 7.0 and 12.4, respectively.

80–1430. Warr, G. A.; Martin, R. R.; Gentry, L. O. **Alterations des Macrophages Alveolaires Pulmonaires Chez les Jeunes Fumeurs de Cigarettes. [Changes in Alveolar Macrophages in Young Cigarette Smokers.]** *Bulletin of the International Union Against Tuberculosis* 51: 569-577, 1976, French.

Pulmonary cells obtained from 100 smokers, average age 23 years, and 200 nonsmokers, aged 22 years, were examined to detect any cellular alterations. The cells were collected by endobronchial lavage. Significantly, the number of cells collected in 250 ml of lavage from the asymptomatic smokers with a cumulative consumption of 5.5 packs of cigarettes/year was about four times higher than the corresponding number from nonsmokers (i.e., 82×10^6 vs. 22×10^6). The alveolar macrophages (AM) comprised 80-90 percent of the total number of cells in both groups of subjects. A staining test revealed a striking abundance of cytoplasmic inclusions in the AM of smokers. This result was confirmed by examination of the AM ultrastructure using electron microscopy. In addition, a decrease in cytoplasmic membrane was observed on the surface of smokers' AM. The smokers' AM surface appeared smooth in contrast to the wrinkled surface of nonsmokers' AM. Smoker AM function tests showed a reduction in the number of the receptor sites for the component C3 of the activated serum complement and in AM phagocytic ability. Only 15 percent of the AM from the smokers formed rosettes of erythrocytes as compared with 55 percent of the AM from nonsmokers. Over 90 percent of AM of the nonsmokers exhibited phagocytosis of staphylococcus, if incubated with fresh or heated serum, while phagocytosis was reduced to about 70 or 40 percent with AM of the smokers. Moreover, 55 percent of phagocytotic macrophages of the smokers were associated with <4 bacteria, while nearly 80 percent of phagocytotic AM in the nonsmokers were associated with 5-20 or more bacteria. However, the presence of the higher number of cells in the lungs of the smokers may compensate for the reduction in functions of individual cells.

80–1431. Woolcock, A.; Leeder, S.; Peat, J.; Blackburn, C. **The Influence of Bronchitis and Asthma in Infancy and Childhood on Lung Function in Schoolchildren.** *Chest* 77(2): 251, February 1980.

The effect of asthma and bronchitis in infancy on subsequent lung function in schoolchildren is studied. A parental questionnaire was used to obtain a history of asthma and bronchitis, and yearly lung function tests were performed in 10,898 children over a 3-year period. A history of respiratory illness in the previous 12 months and a personal smoking history were recorded at the end of the 3-year period. Significantly lower values of the maximum flow at 50 percent of the forced vital capacity (V_{50}) were found in children with a previous history of asthma and/or bronchitis compared to children with no history of disease. Bronchitics who smoked had lower values of V_{50} than nonsmokers ($p<0.01$). The study suggests that lung function in children may be affected by a history of asthma and bronchitis and by subsequent smoking.

80–1432. Yernault, J.-C.; Zamel, N.; Cherniack, R. M. **Effect of Smoking on the Elastic Recoil Pressures of the Lung. (Letters).** *American Review of Respiratory Disease* 121(3): 607-609, March 1980.

Commenting on two articles on the effect of smoking on the elastic recoil pressures of the lung (*American Review of Respiratory Disease* 119(2): 205-211, February 1979; *American Review of Respiratory Disease* 119(5): 707-716, May 1979), the author disagrees with the data showing substantial decrease in lung recoil pressures in smokers (S) vs. nonsmokers (NS) and quotes data in other studies showing no such difference. He attributes the differences in lung elasticity between S and NS to chronic obstructive lung disease suffered by male smokers. The author of one of the original articles replies that the institution which published the study in support of the argument that S have similar recoil as NS has subsequently published new data showing a significant lower lung recoil in S after a followup period of 4 years from its original study. The author of the second article suggests that age of subjects and concentration of inhaled bronchodilator in the other studies might have caused the differences in findings. See also 79-0469 and 79-0969.

CARDIOVASCULAR DISEASES

80–1433. Bansal, R. C.; Tajik, A. J.; Seward, J. B.; Offord, K. P. **Feasibility of Detailed Two-Dimensional Echocardiographic Examination in Adults. Prospective Study of 200 Patients.** *Mayo Clinic Proceedings* 55(5): 291-308, May 1980.

This study highlights the applicability of two-dimensional echocardiography in a group of 200 unselected adult patients referred to the clinical echocardiographic laboratory. The two-dimensional echocardiographic imaging technique permits one to visualize large portions of cardiac anatomy in the majority of patients. The potential effect of age, sex, smoking, and body surface area on the ability to record various tomographic sections was analyzed. Age appeared to be the most important determinant of a successful complete two-dimensional echocardiographic examination. Smoking became more important in older men, reflecting perhaps a cumulative effect of smoking. (Auth. Abs. Mod.)

80–1434. Bjornheden, T.; Wiklund, O.; Bergstrand, R.; Bondjers, G. **Skin Cholesterol and DNA in Young Patients with Myocardial Infarction.** *Acta Medica Scandinavica* 207(4): 271-277, 1980.

Disturbances in cholesterol metabolism are connected with an increased risk of clinical complications to atherosclerosis. Serum cholesterol has been used as an index of such disturbances. However, recently the significance of local tissue, and cellular factors in cholesterol metabolism and atherogenesis have been better appreciated. As easily accessible sources of cells and tissues, skin biopsies have been suggested to increase the possibilities to assess the extent of atherosclerosis in an individual. In order to test this hypothesis, skin biopsies were taken from 24 male patients who had sustained a myocardial infarction before the age of 40, and from 42 healthy, randomly selected male volunteers, matched for age and serum cholesterol. Smoking habits were assessed at the time of biopsy. There were five subgroups: nonsmokers ($n = 1$), previous smokers abstinent at least 4 months ($n = 12$), smokers of 1-14 g tobacco/day ($n = 4$), smokers of 15-24 g tobacco/day ($n = 3$), and smokers of >25 g tobacco/day ($n = 4$). Cholesterol and DNA contents were measured in epidermis and dermis separately; no significant differences were found between the groups. Dermis DNA contents were, however, significantly ($2p < 0.05$) lower in nonsmokers than in smokers among controls, and a similar trend was seen in patients. A significant, positive correlation between serum and dermis cholesterol was found in nonsmokers of both groups. The data do not support the hypothesis that skin biopsies discriminate individuals with atherosclerosis better than serum cholesterol. It is possible that previous data, suggesting a correlation between skin cholesterol and atherosclerosis, might reflect the well known correlation between serum cholesterol and atherosclerosis. (Auth. Abs. Mod.)

80–1435. Bloch, A.; Girod, M.; Bersier, A.-L.; Beurret, L.; Marc, F.; Maeder, J.-P.; Coulet, B.; Ecklin, G. **Facteurs de Risque et Facteurs Psycho-Sociaux Dans l'Infarctus Myocardique de l'Homme Jeune. [Risk Factors and Psychosocial Factors in the Young Male With Myocardial Infarct.]** *Schweizerische Medizinische Wochenschrift* 109(49): 1963-1966, December 22, 1979, French.

Psychosocial and standard risk factors were assessed in 21 young males aged 32-45 admitted for acute myocardial infarction. All patients underwent extensive clinical, dietary, and psychosocial investigation by a team of cardiologists, psychiatrists, dieticians, social workers, and physiotherapists both during hospitalization and later at home. Most standard risk factors were found infrequently and the estimated risk of coronary disease would have been low. Heavy smokers (1-4 packs of cigarettes/day) totaled 20 patients. In every case, before myocardial infarction, the patients had sustained severe long-term stress in their professional, social, and/or familial environments. With rigid and obsessive personality patterns, they appeared to be the prisoners of their problem situations and totally unable to relax. Severe psychosocial stress and heavy smoking appear to be almost constant findings in young males admitted with acute myocardial infarction. (Auth. Abs.)

80–1436. Buerger, S. B.; Strauer, B. E. **Klinische und experimentelle Untersuchungen zur kontraktilen und koronaren Wirkung von Zigarettenrauchen. [Clinical and Experimental Studies of Contractile and Coronary Effects of Cigarette Smoking.]** *Muenchener Medizinische Wochenschrift* 122(Supplement 1): S33-S39, February 20, 1980, German.

Myocardial contractility, ventricular function, and coronary hemodynamics were studied in nine coronary disease patients before and immediately after smoking one cigarette containing 0.8 mg nicotine and 13 mg smoke condensate. All the patients were chronic smokers (5-30 cigarettes/day). In all cases, systolic left ventricular pressure increased significantly by an average of 4.2 percent after smoking ($p < 0.005$). The heart beat increase of 3 beats/min was insignificant. Isovolumetric rate indices, however, indicated significant myocardial contractility increases between 10.2 and 23.2 percent of the original values before smoking. Cardiac output and tension-time index increased by 7.9 and 10.3 percent, respectively. Thus, cigarette smoking leads to an immediate rise in ventricular function and, particularly, in contractility of the left ventricle. Coronary circulation through the left ventricle increased by 6.9 percent and coronary resistance decreased by 9.3 percent. The arterial-venous oxygen difference and myocardial oxygen consumption increased by 5.6 and 12.9 percent, respectively. The pharmacologically demonstrable cardiac reserve of the left ventricle does not appear to be affected by cigarette smoking to any significant degree. In addition to the clinical measurements, experiments were carried out to determine the contractile effect of nicotine on isolated cat ventricular myocardium. Increased nicotine doses produced increases in isotonic muscle contraction and maximum isotonic contraction rate, whereas the isotonic relaxation rate remained unaffected. It is concluded that nicotine produces a positive inotropic effect in isolated ventricular myocardium. A correlation between the rise in inotropy and the increase in the ventricular function parameters and myocardial contractility in the patients remains doubtful.

80–1437. Bulpitt, C. J.; Clifton, P.; Dollery, C. T.; Harper, G. S.; Beilin, L. J.; Coles, E. C.; Gear, J.S.S.; Johnson, B. F.; Munro-Faure, A. D. **Risk Factors for Death in Treated Hypertensive Patients. Report From the D.H.S.S. Hypertension Care Computing Project.** *Lancet* 2(8134): 134-137, July 21, 1979.

A prospective study was performed to determine factors at entry influencing survival in 2,587 treated hypertensive patients who were followed for an average of 4 years. Of the patients, 86 percent had been referred to hospital clinics with hypertension and 14 percent were seen solely by their general practitioners. The patients who died were older and more often male; they were more likely to be smokers ($p<0.001$), to have retinal changes of accelerated hypertension, and to have a history of cardiovascular disease (with the exception of angina). Of the 156 deaths, 81 percent were from cardiovascular causes. Independent risk factors for cardiovascular death were age, impairment of renal function, smoking habits ($p<0.001$), and systolic blood pressure before treatment. Other independent factors of importance were proteinuria, history of myocardial infarction, and retinal changes of accelerated hypertension. Increased weight, serum cholesterol, and serum uric acid were not independent risk factors. Although these results agree substantially with data for normal populations, notable exceptions were impairment of renal function, which was very important in hypertensives, and raised serum cholesterol, which was not an independent risk factor in this hypertensive population. (Auth. Abs. Mod.)

80–1438. Camm, A. J.; Evans, K. E.; Ward, D. E.; Martin, A. **The Rhythm of the Heart in Active Elderly Subjects.** *American Heart Journal* 99(5): 598-603, May 1980.

The dynamic electrocardiogram (DCG) is an increasingly important investigation for the diagnosis of symptoms which might be ascribed to cardiac arrhythmias. This study was designed to document DCG findings in apparently healthy people over the age of 75 years in order to provide a data base with which to compare DCG results in symptomatic patients. The study was performed on 106 subjects (24 smokers, 82 nonsmokers) at home and consisted of a clinical questionnaire, physical examination, resting 12-lead ECG, and 24-hour taped DCG. The DCG demonstrated isolated ventricular premature beats in 45 subjects and potentially more serious ventricular arrhythmias in 32 cases. Ventricular bradycardias of any variety were rarely seen. Arrhythmias were detected two to four times more frequently by DCG than by 12-lead ECG. Within this sample age, ischemic heart disease, systolic hypertension, smoking habits, and a history of syncope, palpitations, or dizziness did not affect the prevalence of any type of arrhythmia.

80–1439. Dobrowolski, L. A. **Arteriosclerosis and Atherosclerosis.** In: Orimo, H.; Shimada, K.; Iriki, M.; Maeda, D. (Editors). *Recent Advances in Gerontology.* Proceedings of the XI International Congress of Gerontology, Tokyo, August 20-25, 1978, 1979, pp. 150-152.

Arteriosclerosis is an aging process that leads to a gradual loss of elasticity in the artery, while atherosclerosis is not a normal aging process but a pathological degeneration in otherwise normal arteries. Hypertension, smoking, and hyperlipidemia are important determinants in the atherosclerotic process. Other factors that have been implicated, such as diabetes mellitus, obesity, stress, and lack of exercise are probably more important in aggravating rather than in initiating the disease. Cigarette smoking has a negative effect on the circulatory system, predisposing it to blood clotting and in the long term to hypoxia and to edema of the arterial intima, the latter a precursor of lipid accumulation and smooth-muscle cell proliferation.

80–1440. Fager, G.; Wiklund, O.; Olofsson, S.-O.; Wilhelmsson, C.; Bondjers, G. **Serum Apolipoprotein Levels in Relation to Acute Myocardial Infarction and Its Risk Factors. Apolipoprotein A-I Levels in Male Survivors of Myocardial Infarction.** *Atherosclerosis* 36(1): 67-74, May 1980.

The significance of high density lipoproteins in the etiology of clinical complications of atherosclerosis has recently received increased attention. The levels of the major apolipoprotein in high density lipoproteins, apoA-I, have been determined in patients who had had an acute myocardial infarction, and compared with a cholesterol-matched and a randomly selected control group. ApoA-I levels were lower in the patients than in the control groups. ApoA-I levels were also lower in smokers than in nonsmokers. The difference between patients and control groups persisted even when the groups were stratified according to smoking habits. In smoking patients, apoA-I levels were significantly lower than in smoking reference group or matched control subjects ($p=0.004$ and $p=0.015$, respectively). However, apoA-I levels of nonsmoking patients were not significantly lower ($p=0.766$) than those of nonsmoking control subjects. This suggests that low levels of apoA-I as well as α-lipoprotein cholesterol are additional characteristics of the infarction patients, even when the established risk factors smoking and hyperlipidemia are taken into account. (Auth. Abs. Mod.)

80–1441. Friedman, G. D.; Siegelaub, A. B. **Changes After Quitting Cigarette Smoking.** *Circulation* 61(4): 716-723, April 1980.

Changes in cardiorespiratory symptoms and coronary disease risk indicators over an average 1 1/2-year period were assessed in 9,392 persistent cigarette smokers and 3,825 persons who quit smoking between two multiphasic checkups. The prevalence of questionnaire-reported chronic cough fell markedly in subjects who quit a one-or-more-pack/day habit (e.g., from 11.2 percent to 1.8 percent in white men, $p<0.001$). However, chest pain, shortness of breath, and exertional leg pain showed no consistent improvement in quitters compared with persistent smokers. Weight gain was about 2-3 lbs greater in quitters, but changes in blood pressure were small and not consistent across race-sex groups, nor were there consistent differences between persistent smokers and quitters in trends in vital capacity, cholesterol, or prevalence of ECG abnormality. Quitting was associated with increase in serum uric acid levels of about 0.2-0.5 mg/dl and relative falls in hemoglobin, leukocyte count, and serum glucose levels, all consistent with smoker-nonsmoker differences previously found in cross-sectional studies. Except for the small increases in weight and uric acid levels, quitting smoking did not appear to increase risk of coronary heart disease by other mechanisms. (Auth. Abs.)

80–1442. Guzowski, K.; Hulanicka, K.; Piechocki, D. **Czynniki Zagrozenia w Udarze Mozgu. [Risk Factors in Stroke.]** *Neurologia i Neurochirurgia Polska* 12(5): 529-535, 1978, Polish.

Effects of several atherogenic factors including tobacco smoking and symptoms of extracerebral atherosclerosis were studied in 300 clinical cases (150 men and 150 women) of stroke. The clinical material was processed statistically in comparison with 120 controls of similar sex and age distribution. Arterial hypertension was found to be the most significant ($p=0.001$) risk factor as it was found in 37.3 percent of stroke cases and in 9.1 percent of controls. The second most important risk factor was diabetes ($p=0.01$) which was found in 11.3 percent of stroke cases and in 3.3 percent of controls. Comparison of the stroke cases and controls indicated that 34.7 percent vs. 25.0 percent, respectively, were long-term smokers. This difference in percentages was not significant. Also, lipid metabolism disorders and obesity were not significant risk factors. In contrast, extracerebral atherosclerosis was found to be a statistically significant risk factor ($p=0.001$). Coronary arterial disease, myocardial infarction, and aorta calcifications were the most significant symptoms of extracerebral atherosclerosis and were observed in 48.8, 8.3, and 32.9 percent, respectively, of all stroke cases. Very often (in 76.3 percent of all cases) association of at least two risk factors was observed. Three or more factors were present in 45.7 percent of the cases. Smoking, lipid metabolism disorders, and extracerebral atherosclerosis were associated in 13 percent of stroke cases.

80–1443. Hamby, R. I.; Hoffman, I.; Weisz, D.; Garvey, J.; Wisoff, B. G. **Recurrent Angina After Bypass Surgery: Evaluation by Early and Late Arteriography.** *American Heart Journal* 99(5): 607-613, May 1980.

The clinical and anatomical correlates of early and late postoperative angiography are described in 98 patients (aged 33-69) evaluated after successful aortocoronary bypass surgery. The clinical information included data regarding hypertension, diabetes mellitus, cigarette smoking, family history, and blood lipids. A total of 177 grafts, including 6 internal mammary artery grafts, were implanted. Of the 98 patients, 38 were referred for recurrent angina pectoris. There were no significant differences in clinical data between symptomatic or asymptomatic patients. The group with recurrent angina had higher frequencies of (1) progressive coronary disease, (2) incomplete surgical revascularization, and (3) graft closure, as compared to the asymptomatic group. It is concluded that control of risk factors, especially lipid abnormalities, is warranted after bypass surgery.

80–1444. Havlik, R. J.; Garrison, R. J.; Feinleib, M.; Padgett, S.; Castelli, W. P.; McNamara, P. M. **Evidence for Additional Blood Pressure Correlates in Adults 20-56 Years Old.** *Circulation* 61(4): 710-715, April 1980.

The offspring of parents belonging to the original Framingham Heart Study cohort, and spouses of these offspring, were examined beginning in 1971. Cardiovascular examinations similar to those performed on the parents in 1952-1953 were given to offspring and their spouses. The mean blood pressure (BP) appeared to be lower in the female offspring-spouses than in the cohort mothers. Comparable multivariate analyses on 3,588 offspring and spouses of both sexes, ages 20-49 years, and 1,842 parents, ages 30-49 years, confirmed the frequently reported importance of weight, heart rate, alcohol consumption, glucose, and hematocrit or hemoglobin as independent positive correlates, and smoking as an inverse correlate of both systolic and diastolic BP ($p<0.05$). In the offspring-spouse population, the additional measured variables of total serum proteins and plasma triglycerides added significantly to prediction of BP in both sex groups, and a less striking negative association was found for serum phosphorus. The findings for serum proteins and triglycerides were supported in a separate population of middle-aged twin men. Even with the additional measured variables, only 28-34 percent of the total BP variance is explained in these populations. (Auth. Abs.)

80–1445. Hawthorne, V. M.; Gilmour, W. H. **Relationship of Glucose to Prevalence of ECG Abnormalities at Baseline and to 6-Yr Mortality in Scottish Males Aged 45-64 Yr.** *Journal of Chronic Diseases* 32(11/12): 787-796, 1979.

A community study of more than 3,000 persons screened in the Burgh of Renfrew, Scotland, included 1,134 men age 45-64 whose baseline glucose level was measured in 1972 and who were followed for 6 years to determine mortality rate. Glucose was significantly positively correlated with systolic blood pressure ($p<0.001$), with relative weight ($p<0.001$), and with cigarette smoking ($p<0.01$). A positive association was found, in univariate analysis, between baseline glucose level and prevalence of electrocardiogram (ECG) abnormalities as defined both by the London Whitehall Study and the U.S. Pooling Project Study. In multivariate analyses of these cross-sectional data, an association was seen for Whitehall but not Pooling Project abnormalities. Trends for high glucose to be associated with higher abnormality rates among smokers and ex-smokers were not significant, and there were no apparent trends among nonsmokers. Cigarette smoking (present or past) showed positive association with cardiovascular and coronary mortality. No association was found between asymptomatic hyperglycemia and coronary mortality at 6 years, either in univariate, bivariate, or multivariate analyses. The clinically observed excess in coronary mortality among those with symptomatic diabetes mellitus was not in evidence among those with asymptomatic hyperglycemia in this group of middle-aged men. (Auth. Abs. Mod.)

80–1446. Hjermann, I. **Smoking and Diet Intervention in Healthy Coronary High Risk Men. Methods and 5-Year Follow-Up of Risk Factors in a Randomized Trial. The Oslo Study.** *Journal of the Oslo City Hospitals* 30(1): 3-17, January 1980.

A 5-year randomized trial has been performed in healthy, normotensive, coronary high-risk men, age 40-49, with serum cholesterol 7.5-9.8 mmol/l (290-379 mg/dl). Of these men, 80 percent initially were daily smokers. The group was allocated at random, 604 and 628 men to the intervention and control groups, respectively. Only diet-hygienic advice was used in the intervention. The daily consumption of tobacco was reduced about 50 percent in the intervention group; a small reduction also occurred in the control group. During the first year, mean serum cholesterol in the intervention group decreased and remained about 13 percent lower than in the control group throughout the 5 years of the study. Lowering of fasting and nonfasting serum triglycerides amounted to 20-25 percent. Body weight remained 2.5-3 kg lower in the intervention than in the control group. Persons in the intervention group who stopped smoking increased their body weight up to a maximum of 1.4 kg during the first 2 years after quitting, but they achieved their initial body weight again after 3-4 years. Serum uric acid was significantly reduced in men with high initial values ($\geq$416 mmol/l-7 mg/dl). During the trial, high density lipoprotein cholesterol was 20 percent higher in the intervention group compared to well-matched controls. Sedimentation rate and fibrinogen concentration seemed to be favorably influenced by the regimen in the intervention group, probably as a consequence of smoking cessation. Blood pressure was significantly reduced in those subjects who adhered to their diet. It is concluded that dietetic-hygienic intervention in healthy, normotensive middle-aged men results in a considerable change in lipids, in smoking habits, and in other variables with relation to atherosclerotic disease. (Auth. Abs. Mod.)

80–1447. Kornitzer, M.; Dramaix, M.; Kittel, F.; De Backer, G. **The Belgian Heart Disease Prevention Project: Changes in Smoking Habits After Two Years of Intervention.** *Preventive Medicine* 9(4): 496-503, July 1980.

The Belgian Heart Disease Prevention Project is a controlled multifactorial prevention trial involving 19,390 men aged 40-59 years, employed by 30 Belgian industries. These industries were paired and randomized into a control or intervention unit. In each intervention factory, the subjects from the two highest deciles of a coronary risk score distribution curve were given semiannual individual advice. Furthermore, a health education campaign was organized in each intervention factory. In the control group, 10 percent of randomly chosen subjects had the same baseline examination as the whole of the intervention group. After 2 years, high-risk subjects as well as random samples of the control and intervention group were compared regarding smoking behavior. Among the high-risk subjects of the intervention group, 18.7 percent stopped smoking as compared to 12.2 percent in the control group ($p\leq0.05$). The random samples of both the intervention and control groups showed an almost identical quitting rate (12.5 and 12.6 percent). The drop in the number of cigarettes smoked daily by smokers at 2 years was significantly different when comparing the high-risk subjects of the intervention and control groups: 18.8 percent and 9.1 percent ($p\leq0.01$). In a stepwise discriminant function analysis, three baseline factors discriminated significantly between high-risk ex-smokers and smokers of the intervention group at 2 years: number of cigarettes smoked daily at entry, earlier attempts to stop smoking, and geographic location in Belgium. For the control group, two baseline factors discriminated between ex-smokers and smokers at 2 years: study level and earlier attempts to stop smoking. (Auth. Abs.)

80–1448. Kraus, J. F.; Borhani, N. O.; Franti, C. E. **Socioeconomic Status, Ethnicity, and Risk of Coronary Heart Disease.** *American Journal of Epidemiology* 111(4): 407-414, April 1980.

This report focuses on the relationship of socioeconomic status (SES), race/ethnicity, and risk of coronary heart disease (CHD) in a large group of employed men. The risk of CHD was determined using the factors of blood pressure, cholesterol, and cigarette smoking ($\geq$1 pack/day) in a multiple logistic model for 19,141 men aged 35-57 years, residing and employed in the Sacramento, California, Standard Metropolitan Statistical Area. The percentage prevalence of cigarette smoking was 30.2 for whites, 25.7 for blacks, 20.7 for Spanish Americans, and 14.8 for Asians in all socioeconomic groups. Corresponding probabilities of CHD events were 0.023 for whites, 0.024 for blacks, 0.015 for Spanish Americans, and 0.018 for Asians. SES was measured by the modified two factor (education and occupation)

Hollingshead index. Information on education, occupation, race/ethnic group, and number of cigarettes smoked was derived from self-administered questionnaires. The data indicate an inverse relationship between SES and risk of CHD, and the relationship persisted for whites, Asians, and Spanish Americans, but not for blacks. Age adjustment did not significantly alter this finding. (Auth. Abs. Mod.)

80–1449. Lagrue, G.; Marcel, G. A.; Faucher, G.; Gallais, M.; Branellec, A. **Deformabilite Erythrocytaire: Influence du Tabagisme et des Facteurs de Risque Vasculaire. [Red Cell Deformability: The Effects of Smoking and Cardiovascular Risk Factors.]** *Nouvelle Presse Medicale* 8(50): 4079-4081, December 24, 1979, French.

Red cell filterability was found reduced to 81 percent of normal in the subjects who were smoking 20-25 cigarettes/day and who did not present other cardiovascular risk factors. In this population sample of 62 subjects, filterability decreased with the increase in cigarette consumption ($p<0.05$). In a separate experiment with 10 heavy smokers, the erythrocyte filtration rate (EFR) was found to decrease in 8 subjects from $10.8 \pm 1.0\mu$/sec before smoking to $9.5 \pm 1.29\mu$/sec 1 hour after smoking two cigarettes. In a sample of 72 patients who were smoking >20 cigarettes/day, the red cell filterability was correlated with the number of cardiovascular risk factors present (i.e., high blood pressure, obesity, diabetes, hyperuricemia, hyperlipidemia, smoking). There was a statistical difference between the groups with risk factors and those without risk factors ($p<0.01$). The difference is highly significant ($p<0.0005$) between zero and four risk factors.

80–1450. LaPorte, R. E.; Cresanta, J. L.; Kuller, L. H. **The Relationship of Alcohol Consumption to Atherosclerotic Heart Disease.** *Preventive Medicine* 9(1): 22-40, January 1980.

A review of the literature indicated that moderate alcohol consumers appear to have a lower rate of heart disease and a lower mortality than nondrinkers or heavy drinkers. A geographic analysis of 20 countries in 1972 suggested that moderate alcohol consumption appeared to be negatively related to rates of heart disease mortality. A temporal analysis within the United States indicated that changes in alcohol consumption, particularly beer, were highly negatively related to changes in heart disease mortality for 1950 to 1975. The review, in addition to the geographic and temporal analyses, suggests a U-shaped relationship between alcohol consumption and heart disease. Possible reasons for the negative relationship between moderate alcohol consumption and heart disease were discussed, including relationships to high-density lipoprotein cholesterol, changes in food patterns, and stress. On the other hand, per capita cigarette consumption was positively (+0.33) correlated to age-adjusted atherosclerotic heart disease (ASHD) death rates. Also, a graphic lag analysis of ASHD death rates (1950-1975) and cigarette consumption (1945-1975) indicates a positive relationship where increases in cigarette smoking are associated with an increase in ASHD mortality 5 years later. (Auth. Abs. Mod.)

80–1451. Leclercq, J. F.; Samuel, J. L.; Merillon, J. P.; Eschwege, E.; Bertrand, M.; Gourgon, R.; Grosgogeat, Y.; Bouvrain, Y.; Swynghedauw, B. **Enquete Epidemiologique Multicentrique sur les Myocardiopathies Primitives. A Propos de 380 Observations. [Epidemiologic Polycentric Survey of Primitive Cardiomyopathy. Based on 380 Observations.]** *Archives des Maladies du Coeur et des Vaisseaux* 72(12): 1302-1310, December 1979, French.

The evolution of cardiomyopathy incidence over 10 years has been studied in four centers in 335 male and 45 female patients aged 17-68 years. The patients were subdivided for comparative purposes into four groups: valvular cardiopathy, myocardial infarct, cardiomyopathy, and controls. Atherosclerosis risk factors (e.g., cholesteremia, hypertension), occupation, diabetes, tobacco and alcohol consumption, mean corpuscle volume, and γ-glutamyl transpeptidase were examined. Average daily tobacco consumption was 11.4 g for the valvular cardiopathy patients, 18.3 for the cardiomyopathy patients, 20.9 g for the myocardial infarct patients, and 16.0 for the controls. A significant ($p<0.001$) difference in consumption was found between valvular cardiopathy and cardiomyopathy patients.

80–1452. Marmot, M. G.; Syme, S. L.; Sacks, S. T.; Kwok, L. W. **Japanese Culture and Coronary Heart Disease.** In: Orimo, H.; Shimada, K.; Iriki, M.; Maeda, D. (Editors). *Recent Advances in Gerontology.* Proceedings of the XI International Congress of Gerontology, Tokyo, August 20-25, 1978, 1979, pp. 476-479.

Men of Japanese ancestry living in Hawaii and California have a higher rate of coronary heart disease (CHD) incidence and mortality than Japanese living in Japan which may be explained only in part by differences in smoking habits and other risk factors. A study was conducted of 3,809 Japanese-American men in California (of whom 86 percent were American-born) to determine whether social and cultural differences between the Japanese in America and those in Japan could be responsible for the difference in CHD rates. Each man was classified as "traditional" or "nontraditional" on the basis of cultural factors including education, religion, and upbringing. Results indicated that for men less than 55 years old, there was a 2-2.7 times greater CHD prevalence in the nontraditional group which could not be accounted for by their more Westernized diet. The effect of culture and other risk factors on CHD prevalence was examined utilizing a multiple logistic equation. It was found that cigarette consumption/day and culture of upbringing were significantly ($p<0.05$) associated with CHD prevalence. The results are consistent with the hypothesis that the unexplained differences in CHD prevalence may be due partly to the loss of the protective effect of Japanese culture.

80–1453. McGill, H. C., Jr. **Potential Mechanisms for the Augmentation of Atherosclerosis and Atherosclerotic Disease by Cigarette Smoking.** *Preventive Medicine* 8(3): 390-403, 1979.

Cigarette smoking is clearly associated with increased risk of atherosclerotic disease, but the mechanisms underlying the association are not known. In the direction of increasing the risk of atherosclerotic disease, smokers have more extensive advanced atherosclerosis, slightly higher serum concentrations of total cholesterol and low-density lipoprotein cholesterol,slightly lower concentrations of high-density lipoprotein cholesterol, higher erythrocyte counts and hemoglobin concentrations, and higher fasting blood glucose concentrations than do nonsmokers. In the direction of decreasing risk, smokers are leaner and have lower blood pressures than nonsmokers. Observed effects which also may augment atherosclerosis in smokers as compared to nonsmokers include leukocytosis and increased hepatic mixed function oxygenase activity. This review of available evidence indicates that cigarette smoking probably contributes to atherosclerotic disease by a variety of mechanisms. (Auth. Abs.)

80–1454. Nora, J. J.; Lortscher, R. H.; Spangler, R. D.; Nora, A. H.; Kimberling, W. J. **Genetic-Epidemiologic Study of Early-Onset Ischemic Heart Disease.** *Circulation* 61(3): 503-508, March 1980.

A genetic-epidemiologic study of a white Colorado population of 207 patients (174 men and 33 women) who had a myocardial infarction before age 55 years was undertaken. Nineteen independent variables were compared between the 207 cases and 621 controls, matched 3:1. The highest risk ratios were associated with a positive family history for ischemic heart disease (IHD). The heritability of IHD was 63 percent when families with the monogenic forms of hyperlipoproteinemia were included, and 56 percent when they were excluded. Smoking more than half a pack of cigarettes per day had a risk ratio of 4.0. A risk index was developed that incorporates family history into a data base of risk factors, which can be readily assessed by the clinician obtaining a screening history, physical, and standard laboratory tests. A scale of 0-10 was devised and the predictive value of the index was tested against another data set. The efficiency of the index was maximal at a screening level of 5. This study suggests that it is logistically feasible to seek patients at high risk for intensive management in a clinical setting (high-risk strategy) using risk indices similar to the one developed for this study, which emphasizes the very important familial component to IHD. (Auth. Abs. Mod.)

80–1455. Olsson, A. G.; Walldius, G.; Rossner, S.; Callmer, E.; Kaijser, L. **Studies on Serum Lipoproteins and Lipid Metabolism. Analysis of a Random Sample of 40 Year Old Men.** *Acta Medica Scandinavica*(Supplementum 637);3, 5, 7-47, 1980.

The purpose of the present study was to establish normal serum lipoprotein concentrations and distributions in a randomly selected sample of 86 40-year-old men in Stockholm. Among the sample characteristics obtained were weight/height (overweight) index, blood pressure, marital status, and tobacco and alcohol consumption habits. Half (n=43) of the sample subjects were current smokers and of these 20 smoked >20 cigarettes/day. There were 18 ex-smokers. Serum lipoprotein and triglyceride (TG) concentrations, fatty acid composition of serum TG, phospholipids, cholesterol esters, free fatty acids, adipose tissue triglycerides, intravenous fat tolerance test, exercise electrocardiogram, and dietary habits were investigated. It was found that those subjects smoking >20 cigarettes/day had higher low density lipoproteins (LDL) but not very low density lipoprotein (VLDL) cholesterol concentrations than nonsmokers. Heavy smokers also tended to have lower high density lipoprotein cholesterol in relation to VLDL TG than nonsmokers.

80–1456. Omae, T. **Prevention of Ischemic Heart Disease and Stroke in Japan.** In: Hayase, S.; Murao, S.; MacArthur, C. (Editors). *Cardiology.* Proceedings of the VIII World Congress of Cardiology, Tokyo, September 17-23, 1978, 1979, pp. 140-144.

According to vital statistics in Japan, the incidence of death due to ischemic heart disease has recently increased together with that of cerebral infarction, whereas that of cerebral hemorrhage has continuously been reduced and become almost equal to that of cerebral infarction. Rapid changes in life style including diet, effect of medical care, and possible bias in clinical diagnosis reflected in the mortality statistics could account for these changes. Although the pattern of cardiovascular diseases in the Japanese has thus increasingly come to resemble that in Western countries, some characteristics still remain, such as a high incidence of cerebrovascular disease and low incidence of ischemic heart disease. An autopsy-based population study in a selected community was used to investigate cerebrovascular and ischemic heart disease incidence as compared with previous information. There were 1,621 subjects, aged over 40, comprising approximately 90 percent of the total population. During the 15-year period from 1961 to 1976, 503 died, of whom 416 were autopsied. The most frequent cause was found to be malignant neoplasm, followed by stroke and heart disease. The frequency of heart disease was about half that of stroke. Among the heart diseases, congestive heart failure was found to be more frequent than myocardial infarction. Of the risk factors in cardiovascular disease, hypertension was found to predominate. Cerebral infarction developed about five times more frequently in hypertensive than in normotensive subjects. The significance of smoking (≥ 10 cigarettes/day) in the development of cardiovascular disease in males (adjusted for age and blood pressure) was investigated, but the result was negative. A marked reduction in deaths including those due to stroke was shown after a hypertension control program was instituted in 1975.

80–1457. Patel, Y. C.; Kodlin, D.; Strong, J. P. **On the Interpretation of Smoking Risks in Atherosclerosis.** *Journal of Chronic Diseases* 33(2): 147-155, 1980.

A simple two-parameter model for the formation of fatty streaks from normal intimal surface at a constant rate, and with a subsequent conversion to a raised lesion at a constant rate is described. Data from 1,267 autopsied men from the New Orleans area (for whom smoking history and measurements of both fatty streaks and raised lesions of the abdominal and aortic arteries were available) were used to test the validity of the model and to investigate the effect of smoking on atherosclerosis using the model. The transition rate in the abdominal aorta from normal to fatty streak increased from 0.014 in nonsmokers to 0.026 in light smokers and 0.031 in heavy smokers ($p<0.001$). The conversion rates of fatty streaks to raised lesions were also associated with smoking and increased from 0.039 to 0.087 ($p=0.02$). In the coronary arteries only the conversion of fatty streaks to raised lesions was enhanced. Prevalence curves were drawn from autopsy case data. The two-parameter model described appears to give a fairly good account of the atherosclerotic events in time and their modification by smoking.

80–1458. Schievelbein, H. **Evaluation of the Role of Carbon Monoxide and Nicotine in the Pathogenesis of Arteriosclerosis and Cardiovascular Disease.** *Preventive Medicine* 8(3): 379-389, 1979.

The evidence for the role of smoking, carbon monoxide (CO), and nicotine in the etiology of cardiovascular diseases (CVD) is evaluated critically. Positive statistical correlations between smoking and CVD obtained in several epidemiological studies demonstrate the need to investigate possible causative biological mechanisms. Ambient concentrations of CO influence carboxyhemoglobin (COHb) levels in humans. Correlations are reported between the COHb level and CVD, but there is no clearcut evidence of a public health risk from ambient CO concentrations. Assuming that a major part in the etiology of CVD is the development of arteriosclerosis, investigations in this direction have been performed with CO and nicotine in animal experiments. Neither substance has any influence, comparable to human arteriosclerosis, on the development of CVD. (Auth. Abs.)

80–1459. Stamler, J. **What Is Happening With Coronary Heart Disease in the United States?** In: Hayase, S.; Murao, S.; MacArthur, C. (Editors). *Cardiology.* Proceedings of the VIII World Congress of Cardiology, Tokyo, September 17-23, 1978, 1979, pp. 125-130.

A sizable downward trend in coronary heart disease, stroke, cardiovascular disease, and all causes of mortality has in recent years occurred in the United States. Changes in trends of lifestyle and risk factors such as diet and serum cholesterol, exercise habit, detection and control of hypertension may be playing a role in reducing mortality. Cigarette smoking has declined since the Surgeon General's report in 1964. The percentage of adult male (M) and female (F) smokers dropped from 52.4 (M) and 32.5 (F) in 1964-1966, to 39.3 (M) and 28.9 (F) in 1975. However, the problem of teenage smoking (particularly teenage girls) still remains.

80–1460. Stamler, R.; Stamler, J. **Asymptomatic Hyperglycemia and Coronary Heart Disease. A Series of Papers by the International Collaborative Group, Based on Studies in Fifteen Populations. Introduction.** *Journal of Chronic Diseases* 32(11/12): 683-691, 1979.

The relationship between asymptomatic hyperglycemia and coronary heart disease was studied in 15 different populations. Baseline measurements for each study included circulating glucose, electrocardiogram (ECG), and a number of variables relevant to cardiovascular status: relative weight, blood pressure, smoking history, and circulating cholesterol. Each study had the capacity to follow to death those examined at baseline; 11 studies completed 4 or more years of such followup. For the other four studies, only cross-sectional data are presented. The end points for the prevalence analyses were baseline ECG findings. Data on mortality are included for those studies with followup of 4 years or longer. Data on the prevalence of cigarette smoking at entry in the 15 populations, and the average number of cigarettes smoked per day for each group overall and for its smokers only are given. There were sizable interpopulation differences (ranging from 13.4 cigarettes/day in Denmark to 24.9 cigarettes/day in Japan) in reported cigarette smoking.

80–1461. Stamler, R.; Stamler, J. (Editors). **Asymptomatic Hyperglycemia and Coronary Heart Disease. A Series of Papers by the International Collaborative Group, Based on Studies in Fifteen Populations.** *Journal of Chronic Diseases* 32(11/12): 683-837, 1979.

This is a series of papers based on studies in 15 different populations on the association between asymptomatic hyperglycemia and coronary heart disease (CHD). No consistent association between asymptomatic hyperglycemia and CHD was found. Various analyses were performed using blood cholesterol, blood pressure, cigarette smoking, and electrocardiographic data.

80–1462. Stimmel, B. **The Stimulants 2: Caffeine and Nicotine.** In: *Cardiovascular Effects of Mood-Altering Drugs.* New York, Raven, 1979, pp. 253-278.

Studies of the cardiovascular effects of caffeine and nicotine show that the consumption of tea, cocoa, and cola drinks is not associated with any definite risk in individuals with cardiac disease, whereas all available statistical and biological evidence demonstrates a strong association between smoking and cardiovascular disease. Cigarette smoking has been shown to be related to the occurrence of angina, myocardial infarction, and mortality due to coronary artery disease. Smoking is not only an independent factor but can act cumulatively with other major risk factors. Consumption of low-tar and low-nicotine cigarettes, although reducing the amount of nicotine absorbed, still is associated with a risk due to carbon monoxide. Studies reviewing risk in ex-smokers have revealed a marked diminution in cardiovascular disease risk in patients under age 65 upon ceasing smoking.

80–1463. Studer, A.; Baumgaertner, R.; Siebenschein, R.; Satz, N.; Grimm, J.; Siegenthaler, W.; Vetter, W. **Pravalenz der Hypertonie und Grenzwerthypertonie bei Studenten. Vergleich mit einem nicht-studentischen Kollektiv. [Prevalence of Hypertension and Borderline Hypertension. Comparison With a Non-Student Group.]** *Schweizerische Medizinische Wochenschrift* 110(9): 338-346, 1980, German.

A blood pressure detection program was conducted at the University of Zurich in June 1978. Three consecutive blood pressure measurements were performed in 1,364 students and 440 other subjects. Height and body weight were also measured and used to calculate the body mass index. Personal history, physical activity, smoking, and dietary habits were analyzed with the aid of a questionnaire. Based on the mean value of all three measurements, hypertension was observed in only 1.7 percent of the male and 0.9 percent of the female students, compared to 7.9 and 4.5 percent, respectively, in the other subjects. The prevalence of borderline hypertension was 7.4 percent in male and 3.0 percent in female students; corresponding values for the other subjects were 18.7 and 11.6 percent. The percentage of smokers was 31.9 percent in male and 24.1 percent in female students. Comparable differences between both sexes were observed in nonstudents (males 28.9 percent, females 19.7 percent). Female students taking oral contraceptives ($n=139$, 31.6 percent) showed significantly higher systolic (111.5 mm Hg, $p<0.005$) and diastolic (72.5 mm Hg, $p<0.05$) blood pressure values than nontakers (systolic 108.4, diastolic 70.2 mm Hg), whereas these two groups showed no differences in body mass index. The percentage of smokers among female students taking oral contraceptives (32 percent) was as high as in male students. Statistical analysis showed no significant correlations between blood pressure and smoking habits, salt intake, frequency of meals, and physical activity. Significant relationships, however, were observed between blood pressure and age and between blood pressure and body mass index ($p<0.001$). (Auth. Abs. Mod.)

80–1464. Townsend, J. L.; Meade, T. W. **Ischaemic Heart Disease Mortality Risks for Smokers and Non-Smokers.** *Journal of Epidemiology and Community Health* 33(4): 243-247, December 1979.

Although many studies have shown that smoking is associated with an increased risk of death from ischemic heart disease (IHD), and that the increase appears to vary with age and amount smoked, there has been little formal specification or estimation of the relationship. In this paper, two alternative models are tested, using data for different ages and levels of smoking from four major studies in three countries. One model explains 80 percent of the variation in mortality in terms of a positive linear function of the number of cigarettes smoked, the parameters of which decrease with age. In the second model, it is estimated that every cigarette smoked per day increases the risk of dying from IHD by as much as 35 percent at ages 35-44, and reduces the risk to 2 percent at ages 65-74. The risk attributable to smoking may account for more than 80 percent of IHD deaths of men aged 35-44, and 27 percent of those of men aged 45-64. Although the relative risk is highest for younger age groups, the absolute risk of death from IHD that is attributable to smoking increases with age. The evidence suggests that both are increasing with time. (Auth. Abs.)

80–1465. Trap-Jensen, J.; Carlsen, J. E.; Svendsen, T. L.; Christensen, N. J. **Cardiovascular and Adrenergic Effects of Cigarette Smoking During Immediate Non-Selective and Selective Beta Adrenoceptor Blockade in Humans.** *European Journal of Clinical Investigation* 9(3): 181-183, June 1979.

The cardiovascular and adrenergic responses to cigarette smoking during acute selective and nonselective beta adrenoceptor blockade were studied in seven young healthy volunteers (one female and six males, aged 23-45 years) in a double-blind crossover study. All subjects smoked 10-20 cigarettes daily. Heart rate, arterial blood pressure, forearm blood flow, and plasma levels of adrenaline and noradrenaline were determined before and during the terminal 5-min period of the 15-min smoking test. During smoking, plasma concentrations of adrenaline increased markedly and evenly by approximately 0.3 ng/ml in all three experimental sessions. Plasma concentrations of noradrenaline remained unchanged. Propranolol, a nonselective beta blocker, caused a marked rise in diastolic and mean blood pressure and forearm vascular resistance during smoking. This response was not seen in the control series or after selective beta-1 blockade with atenolol. This difference is attributable to propranolol's blockade of adrenaline's vasodilating effect mediated by beta-2 receptors in the resistance vessels. Furthermore, atenolol attenuated the systolic blood pressure and tachycardiac responses induced by cigarette smoking by comparison with placebo. This study suggests that selective beta-1 blockers are preferable in the management of patients who are habitual smokers. (Auth. Abs. Mod.)

80–1466. Williams, R. S.; Logue, E. E.; Lewis, J. L.; Barton, T.; Stead, N. W.; Wallace, A. G.; Pizzo, S. V. **Physical Conditioning Augments the Fibrinolytic Response to Venous Occlusion in Healthy Adults.** *New England Journal of Medicine* 302(18): 987-991, May 1, 1980.

The effects of a 10-week physical conditioning program on fibrinolytic activity at rest and after stimulation by venous occlusion were studied in 69 healthy adults 25-69 years old, of whom 47 were smokers. Physical conditioning was documented by treadmill performance, and fibrinolysis was measured with a newly developed radioenzymatic assay. Whereas fibrinolysis declined at rest from 16.2 ± 1.3 to 11.4 ± 0.8 units (mean ± S.E.M.) ($p=0.0017$), the increment in fibrinolysis produced by venous occlusion was increased from 21.7 ± 2.9 to 33.8 ± 4.7 units ($p=0.0037$). This augmentation was most marked in women, persons with low initial levels of stimulated fibrinolysis, and persons with low initial physical fitness. Since only five subjects reported a change in smoking habits between their enrollment and the end of the study, it is unlikely that the observed increase in stimulated fibrinolysis could be attributed to changes in smoking habits that occurred during the study. It is concluded that physical conditioning can enhance the augmentation of fibrinolytic activity that occurs in response to venous occlusion. Enhanced fibrinolysis in response to thrombotic stimuli could be an important mechanism in the beneficial effect of habitual physical activity on the risk of cardiovascular disease. (Auth. Abs. Mod.)

80–1467. Wong, J.; Lam, S.T.K.; Ong, G. B. **Buerger's Disease—A Review of 105 Patients.** *Australian and New Zealand Journal of Surgery* 48(4): 382-387, August 1978.

One hundred and five cases (101 males and 4 females) of Buerger's disease occurring in Hong Kong have been reviewed. The digits in the foot that were affected were predominantly the big, second, and little toes, whereas in the hand the index and middle fingers were most commonly involved. There was no selective involvement of the left or right side in the upper or lower extremities. All but four of the patients (two men and two women) were cigarette smokers; most smoked 11-20 cigarettes per day. Patients were treated conservatively at first, including abstinence from smoking, analgesics, vasodilators, and care to the ischemic digits. The majority of patients needed surgery and had amputation, sympathectomy, or both procedures performed. Major lower limb amputation was carried out in 22.6 percent of patients with lower limb involvement. Reconstructive arterial surgery was possible in only four patients, and early failure of grafts in three patients resulted in major amputation. (Auth. Abs. Mod.)

See also, **80–1384, 80–1468**

PREGNANCY AND INFANT HEALTH

80–1468. Asmussen, I. **Effects of Maternal Smoking on the Fetal Cardiovascular System.** *Cardiovascular Medicine* 4(7): 777-790, July 1979.

As a coronary risk factor, maternal smoking may produce its initial vessel lesions in humans during the 9 months of fetal life. Vessel wall damage comprising intima and media changes influenced by maternal smoking has been demonstrated in biopsies from the fetal cardiovascular system at birth. Heavy smokers ($n=13$, ≥ 10 cigarettes/day) were compared with nonsmokers ($n=15$), and the vessel damage was demonstrated in smokers only. In biopsies taken from the umbilical artery and vein and the fetal capillaries in the placenta, changes were demonstrated in the vessels that had conducted the blood to and away from the fetus. It seems reasonable to conclude that, in the infant, the vasculature between these two vessels has developed some degree of morphologic change that may give rise to sequelae later in life. (Auth. Abs.)

80–1469. Asmussen, I. **Ultrastructure of the Villi and Fetal Capillaries in Placentas From Smoking and Nonsmoking Mothers.** *British Journal of Obstetrics and Gynaecology* 87(3): 239-245, March 1980.

The ultrastructure of placental villi from 10 heavy smokers (≥ 10 cigarettes per day) was compared with that of 16 nonsmokers (never smoked). Thickening of the basement membrane and increase in collagen content of the villous stroma was found. The fetal capillaries were fewer in number and with decreased lumen either due to edema or contraction of the endothelial cells. All cells, both in the fetal capillaries as well as syncytiotrophoblast and cytotrophoblast contained large amounts of filaments and fibrillae possibly capable of cell contraction. Thus a result of these findings might be a decreased placental flow in smokers. (Auth. Abs.)

80–1470. Estel, C.; Kadner, J. **Rauchen in der Gravidität. [Smoking During Pregnancy.]** *Zentralblatt fuer Gynaekologie* 100(9): 579-582, 1978, German.

A sample of 1,658 women was surveyed immediately postpartum about their smoking habits by means of a questionnaire. Of the women, 28 percent had been smokers who stopped during pregnancy, and 11 percent were smokers who continued during pregnancy. In this latter category, 61.7 percent of the 175 women smoked 1-5 cigarettes/day, 30.3 percent 6-10, and 8 percent more than 10 cigarettes/day. Premature labor occurred in 8.5 percent of the women who smoked during pregnancy vs. 4.9 percent of the nonsmokers. Among the premature newborns of nonsmoking mothers, only 1.0 percent were dystrophic compared to 2.3 percent of the premature infants born to mothers who had smoked during pregnancy. Of the full-term dystrophic infants, 5 percent were born to nonsmoking mothers vs. 12.6 percent to smoking mothers. These differences are statistically significant ($p<0.01$).

80–1471. Gibel, W.; Blumberg, H.-H. **Die Auswirkungen der Rauchgewohnheiten von Eltern auf das ungeborene und neugeborene Kind. [Effect of the Parents' Smoking Habits on the Unborn and Newborn Child.]** *Zeitschrift fuer Aerztliche Fortbildung* 73(7): 341-342, April 1, 1979, German.

A survey of recent literature shows that women who smoke during pregnancy have a 2-3 times higher probability than nonsmoking women of having a newborn infant who will be 150-250 g underweight, depending on the number of cigarettes smoked from the fourth month of pregnancy. Mental retardation was found in a study of 17,000 children up to 11 years old who were born to smoking mothers. The possibility of an abortion or a stillborn child was twice as high in smoking as in nonsmoking women. In a study of 5,200 cases, greater perinatal mortality was found in children whose fathers smoked >10 cigarettes/day even when the mother was a nonsmoker, than in children whose parents were nonsmokers. The incidence of severe malformations in children of fathers who are heavy smokers was double that found in children of nonsmoking fathers, regardless of the parents' age and social position. There are presently 1.3 million smoking women and 3.2 million smoking men in East Germany. Since these numbers grow steadily, one must anticipate a still higher percentage of smoking parents in the total population. Interested persons should be advised about the possible risks of smoking to unborn children in the framework of popular science lectures and marriage and pregnancy counseling.

80–1472. Johnson, J.W.C.; Dubin, N. H. **Prevention of Preterm Labor.** *Clinical Obstetrics and Gynecology* 23(1): 51-73, March 1980.

Attributable risks for preterm delivery include low socioeconomic status, repetitive prematurity (and abortions), unwanted pregnancies, smoking during pregnancy, placenta praevia and abruptio placentae, multiple pregnancies, maternal illnesses (e.g., hypertension, cardiac, urologic, pulmonary), and fetal malformations. Each of these risks is briefly reviewed. The highest percentage of preterm deliveries is attributed to low socioeconomic status (40-60 percent); smoking as a risk factor was in the median range (4-14 percent). Mechanisms of preterm delivery such as socioeconomic status, fetal hypoxia, hormones, and cervical factors, are also discussed.

80–1473. Martin, D. C.; Martin, J. C.; Streissguth, A. P. **Sucking Frequency and Amplitude in Newborns as a Function of Maternal Drinking and Smoking.** In: Galanter, M. (Editor). *Currents in Alcoholism.* Biomedical Issues and Clinical Effects of Alcoholism, Volume 5, New York, Grune and Stratton, 1979, pp. 359-366.

A sample of 151 unselected pregnant women who were in prenatal care by the fifth month of pregnancy were interviewed during pregnancy regarding their use of alcohol, caffeine, drugs, and medication. Maternal alcohol use during and prior to recognition of pregnancy, and cigarette use during pregnancy, were the criteria for infant selection for this study. Heavy nicotine intake was defined as ≥16 mg/day, light intake was ≤6 mg/day, and no intake was 0 mg/day. Corresponding percentages of smokers found in the sample are 8.6, 10.6, and 80.8. The 151 2-day-old infants from these mothers were seen for 75 min for their experimental sucking trial. Trials were terminated after 10 responses on a nonnutritive nipple or after 30 sec had elapsed with no response. Data such as latency to first suck, number of sucks emitted, number of trials, and trial length were recorded. Both sucking pressure and whole-trial average pressure decreased significantly with alcohol ($p=0.03$) and with nicotine ($p=0.01$). Both latency and trial length increased significantly ($p=0.01$) with increasing nicotine scores.

80–1474. Naeye, R. L. **Abruptio Placentae and Placenta Previa: Frequency, Perinatal Mortality, and Cigarette Smoking.** *Obstetrics and Gynecology* 55(6): 701-704, June 1980.

Abruptio placentae and placenta praevia are responsible for at least half the excessive fetal and neonatal deaths associated with maternal smoking during pregnancy. Data from a large prospective study were analyzed to determine if stopping smoking reduced the frequency of these disorders. Mothers who stopped smoking had a 23 percent lower frequency of abruptio placentae and a 33 percent lower frequency of placenta praevia than women who continued to smoke during pregnancy. The mothers who stopped smoking had only about half as many fetal and neonatal deaths due to abruptio placentae and one-third fewer losses to placenta praevia than did mothers who continued to smoke. These seeming benefits of stopping smoking were greatest in mothers 30 years of age and older. (Auth. Abs.)

80–1475. Naeye, R. L. **Cigarette Smoking and Pregnancy Weight Gain. (Letter).** *Lancet* 1(8171): 765-766, April 5, 1980.

In order to determine whether raising birthweights through increasing maternal weight gains might protect the fetus against the unfavorable effects of smoking, data from 45,113 pregnancies were analyzed. Maternal weight gains were calculated in percent of "optimal" gains. The frequencies of abruptio placentae, placenta praevia, and large placental infarcts were determined for various maternal weight gains because these disorders are responsible for most of the increased fetal and neonatal deaths associated with maternal cigarette smoking during pregnancy. The study showed that the frequencies of the placenta disorders were greater in smokers (>10 cigarettes/day) than in nonsmokers and changed little as pregnancy weight gains increased. In the case of large placental infarcts, for example, where the weight gain was <45 percent of the optimal gain, the number of cases/1,000 births was 29.0 for nonsmokers and 49.5 for smokers ($p<0.001$). The comparable figures where the weight gain was more than 75 percent were 29.3 and 39.3. It is concluded that increasing maternal weight gain is unlikely to protect the fetus against the adverse effects of maternal smoking.

80–1476. Naeye, R. L. **The Duration of Maternal Cigarette Smoking, Fetal and Placental Disorders.** *Early Human Development* 3(3): 229-237, September 1979.

Data from a large prospective study of pregnancy were used to determine whether the number of years a mother had smoked cigarettes influenced the development of seven common fetal and placental disorders. Three disorders increased in frequency when mothers had smoked for more than 6 years: placenta praevia 143 percent, abruptio placentae 72 percent, and large placental infarcts 37 percent (all $p<0.05$). Although mothers' current smoking habits had a smaller influence on the frequency of these disorders, all seven disorders were more prevalent in smokers than nonsmokers ($p<0.1$). The effects of smoking were largely independent of maternal pregnancy weight gain. The placentas of smokers had microscopic evidences of underperfusion from the uterus. The placental abnormalities were influenced by both the number of years mothers had smoked and by their current smoking habits. (Auth. Abs. Mod.)

80–1477. National Center for Health Statistics. **Factors Associated With Low Birth Weight, United States, 1976.** U.S. Department of Health, Education, and Welfare, Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, DHEW Publication No. (PHS) 80-1915, April 1980, 42 pp.

This report describes the pattern of occurrence of low birthweight in the United States relative to demographic, socioeconomic, and health characteristics recorded on birth certificates. In addition, recent trends in low birthweight incidence are examined.

80–1478. Rush, D.; Stein, Z.; Susser, M. **A Randomized Controlled Trial of Prenatal Nutritional Supplementation in New York City.** *Pediatrics* 65(4): 683-697, April 1980.

This randomized controlled trial of nutritional supplementation in pregnancy, in a poor black urban population in the United States, aimed to increase the birthweight and influence the postnatal development of the offspring of mothers at high risk of having low birthweight infants. The execution of the research design and adherence to the treatment regimen among the experimental population appeared adequate for a reasonable test of the treatments. At birth, the only significant favorable effect of supplementation observed was the prevention of depressed birthweight among the offspring of mothers who smoked heavily (≥ 15 cigarettes/day). With balanced protein-calorie supplementation, length of gestation was increased, the proportion of low birthweight infants reduced, and mean birthweight raised by 41 gm (not statistically significant). With high protein supplementation, there was an excess of very early premature births and associated neonatal deaths, and there was significant growth retardation up to 37 weeks of gestation. At 1 year of age, significant effects of high protein supplement were found on three psychological measures: visual habituation, visual dishabituation, and mean length of free play episodes. These measures were unrelated to measures of growth at birth and at 1 year of age. There were no detectable residual adverse effects of high protein supplementation at 1 year of age. (Auth. Abs.)

80–1479. Whichelow, M. J. **Breast Feeding in Cambridge, England: Factors Affecting the Mother's Milk Supply.** *Journal of Advanced Nursing* 4(3): 253-261, May 1979.

Mothers who were breast feeding 2 weeks after delivery were studied. One group (106 mothers) were advised to "eat for two" during lactation to sustain their milk supply. They were compared at 3 months with a control group of 152 mothers. At 3 months only half as many study group mothers had weaned their babies due to insufficient milk as control mothers. The numbers weaning for other reasons were similar in both groups. More breast feeding mothers reported an increased appetite during lactation than did mothers who had changed to artificial feeding. Smokers were more likely to have given up breast feeding between 2 weeks and 3 months than nonsmokers, but smoking was not associated with any particular weaning reason. Women taking a contraceptive pill during lactation more often experienced a diminution of milk supply than those not taking pills. Sustained lactation benefits the mother since those who were still breast feeding were less likely to suffer from depression or fatigue at 3 months, and were more likely to have lost weight. The early introduction of solids was less frequent among infants being breast fed at 3 months. (Auth. Abs.)

See also, **80–1338, 80–1369, 80–1524**

OTHER DISEASES AND CONDITIONS

80–1480. Campbell, J. M.; Harrison, K. L. **Smoking and Infertility. (Letter).** *Medical Journal of Australia* 1(8): 342-343, April 21, 1979.

In order to determine whether nicotine has a negative effect on sperm motility and density, the semen analyses of the male partner of 253 couples at an infertility clinic were assessed in relation to the husband's smoking habits. Of the men, 53 percent were regular smokers. The results suggest an association between smoking and reduced sperm motility and density. Reduced motility was found in 35 percent of the smokers and 25 percent of the nonsmokers; reduced density in 41 and 26 percent, respectively.

80–1481. Garn, S. M.; Shaw, H. A.; McCabe, K. D. **Maternal Smoking as a Nutritional Variable.** *Ecology of Food and Nutrition* 7(3): 143-145, 1978.

As shown in 17,649 pregnancies (followed in the National Collaborative Survey of the National Institute of Neurological and Communicative Disorders and Stroke), prepregnancy weights of smoking mothers are systematically lower than comparable weights of nonsmoking mothers appropriately corrected both for weight and per capita income. In similar fashion, weight gain between pregnancies is considerably smaller for women who smoke than for nonsmoking women of the same race. (Auth. Abs. Mod.)

80–1482. Giger, M.; Gonvers, J.-J.; Weber, K. B.; Sonnenberg, A.; Birchler, R.; Mattle, W.; Hofstetter, J.-R.; Blum, A. L. **Pirenzepin-Behandlung des Ulcus duodeni: Vergleich mit Cimetidin und Placebo. [Duodenal Ulcer Treatment With Pirenzepine: Comparison With Cimetidine and Placebo.]** In: Blum, A. L.; Hammer, R. (Editors). *Die Behandlung des Ulcus pepticum mit Pirenzepin.* [*The Treatment of Peptic Ulcer with Pirenzepine.*] Report on the First International Pirenzepine Symposium, November 10-11, 1978, Titisee, Verhandlungsband 15, Federal Republic of Germany, Grafelfing, Demeter Verlag, 1979, pp. 216-220, German.

In a double-blind study, 50 randomly selected ambulatory patients, 37 men and 13 women, average age 44 years, were treated for acute duodenal ulcer with pirenzepine, cimetidine, and placebo. There were 30 smokers and 20 nonsmokers in the group. Two groups of 16 patients each were treated with 75 mg/day and 1 g/day doses of pirenzepine and cimetidine tablets, respectively; the remaining 18 patients received placebo. Patients complaining about pain received aluminum hydroxide-base antacid. No diet was prescribed, nor were smoking or alcohol consumption restricted. After 4 weeks of treatment, 11 patients receiving placebo, 12 receiving cimetidine, and 11 treated with pirenzepine were cured, i.e., they did not exhibit ulcer symptoms. The results in favor of pirenzepine were statistically insignificant. The ulcer healing rate was significantly lower for smokers ($p<0.05$). A favorable effect on ulcer healing in smokers was observed with cimetidine but not with pirenzepine (the ratio of smokers to nonsmokers among the cured patients was 8 to 4 with cimetidine and 4 to 7 with pirenzepine). Dryness of the mouth was significantly more frequent with pirenzepine than with the two other types of treatment. There was no difference in the incidence of other side effects in the three groups studied. It is concluded that the effects of all three substances tested are identical with respect to healing and symptoms of duodenal ulcer.

80–1483. Luria, S. M.; McKay, C. L. **Visual Processes of Smokers and Nonsmokers at Different Ages.** *Archives of Environmental Health* 34(6): 449-454, November-December 1979.

Forty smokers and 40 nonsmokers (of whom 27 had never smoked) were matched for age over the range from 21 to 76 years. The battery of tests included optometric refraction for near vision, scotopic sensitivity, simple and choice reaction time, visually evoked cortical potentials to targets flashing at 1 and 16 times/sec, electroencephalogram (EEG), and a perimetric test of color detection. Roughly the same number of smokers and nonsmokers required spectacle corrections, but smokers tended to require more plus correction. The mean scotopic sensitivity of the smokers was significantly worse than that of the nonsmokers ($p<0.01$). Reaction time tended to be poorer for the smokers. There were no differences on the perimetry test and in the EEGs of the smokers and nonsmokers, and no significant changes in the visually evoked responses to either 1 or 16 flashes/sec. There was virtually no evidence of an increasing difference between the smokers and nonsmokers with increasing age. (Auth. Abs. Mod.)

80–1484. Nilsson, B. **Taste Acuity of the Human Palate. II. Studies With Electrogustometry on Subjects in Different Age Groups.** *Acta Odontologica Scandinavica* 37(4): 217-234, 1979.

The electrical taste acuity of the hard and soft palate and of the tip of the tongue was determined in 140 subjects aged 13-81 years using an electrogustometer constructed for the study. The possible influence of several variables including age, sex, wearing of a full upper denture, smoking, and medical and social factors on the threshold values was also studied. Taste perception was demonstrated on the soft palate and on an area of the hard palate close to the junction of the hard and soft palate in most subjects. No perception of the electrical taste was found in 11 subjects (8 percent) on either the hard or the soft palate, and in an additional 33 subjects (24 percent) on the hard palate alone and 2 subjects (1 percent) on the soft palate alone. The taste thresholds on the hard palate were very high compared to those on the soft palate and those on the tongue. Older subjects generally had higher thresholds than did younger subjects, and the differences were greater for the thresholds on the hard and soft palate than for those on the tongue. No statistically significant differences in threshold values were found between women and men, denture wearers and nondenture wearers, or smokers ($n = 46$) and nonsmokers. (Auth. Abs.)

80–1485. Noppa, H.; Bengtsson, C. **Obesity in Relation to Smoking: A Population Study of Women in Goteborg, Sweden.** *Preventive Medicine* 9(4): 534-543, July 1980.

From a population sample of 1,462 middle-aged women (participation rate 90.1 percent) studied in 1968 and 1969, 1,302 (participation rate 80.3 percent) were reexamined in 1974 and 1975. Cigarette smoking was more frequent ($p<0.001$) among younger women. Current smokers were leaner than nonsmokers. A U-shaped relationship was found between obesity index and cigarette consumption. This relationship was not explained by differences in energy intake, physical activity, or social class. Cessation of smoking was correlated with weight gain and commencement of smoking with weight loss. Of those who quit, 13 percent gained ≥ 10.0 kg, and 22 percent gained between 5.0 and 9.9 kg, compared with 4 and 14 percent, respectively, among those who did not change smoking habits. The average weight change was +3.5 and -0.7 kg among those who quit smoking and started smoking, respectively, compared with +1.4 kg among those who did not change smoking habits. A greater weight gain with the amount smoked was found both in those who stopped smoking and in those who continued smoking. Increased physical activity and reduced energy intake need to be considered at cessation of smoking, especially among heavy smokers. The quantitative aspects of the relationship between smoking and obesity, and between cessation of smoking and weight gain, are considered to be of special interest for health personnel engaged in antismoking campaigns. (Auth. Abs.)

80–1486. Pindborg, J. J.; Reibel, J.; Roed-Petersen, B.; Mehta, F. S. **Tobacco-Induced Changes in Oral Leukoplakic Epithelium.** *Cancer* 45(9): 2330-2336, May 1, 1980.

A study of 112 biopsy specimens from Danish oral leukoplakia patients (of whom 10 were nonsmokers and 15 were ex-smokers) and 145 biopsy specimens from Indian leukoplakia patients (all of whom smoked or chewed tobacco) revealed a chevron-like keratinization of the epithelium in a number of cases. Clinically, this type of keratinization was often characterized by a pumice-like appearance. The clinical and histologic changes were only observed in tobacco users, and appeared to be associated particularly with use of pipes, snuff, and hooklis. (Auth. Abs. Mod.)

80–1487. Purchase, I.F.H.; Richardson, C. R.; Anderson, D.; Paddle, G. M.; Adams, W.G.F. **Chromosomal Analyses in Vinyl Chloride-Exposed Workers.** *Mutation Research* 57(3): 325-334, 1978.

Chromosomal morphology from cultured peripheral lymphocytes was studied in 81 men; 57 of the men were employed in plants manufacturing vinyl chloride or polyvinylchloride, 19 were on-site controls, and 5 were off-site controls. There was a significant increase in chromosomal abnormalities in the exposed workers when compared with the controls. The greatest statistically significant increase in total B and total C cells occurred in autoclave operators ($p<0.01$), with smaller increases in other job categories. The increase in chromosomal aberrations was correlated with the length of exposure and with a history during the year prior to sampling (1973-1974) of exposure to excursion levels of vinyl chloride. Information on smoking habits was obtained 18 months after blood sampling and a positive correlation between these and total C cell abnormalities was found. The mean values of percentage of C cell abnormalities, irrespective of their occupational history, was 1.80 for present smokers and 1.06 for nonsmokers. There was no positive correlation with various other parameters (bilirubin, platelets, γ-glutamyltranspeptidase, alkaline phosphatase, alanine transaminase, and aspartate transaminase). It was not possible to estimate which of the three parameters (smoking history, length of employment, or exposure to excursion levels) was the most important. (Auth. Abs. Mod.)

80–1488. Stekhun, F. I. **Alkogol' i Tabakokurenie Kak Vozmozhnye Prichiny Besplodiia Muzhchin. [Alcohol and Tobacco Smoking as Possible Causes of Male Sterility.]** *Vestnik Dermatologii i Venerologii* (7): 61-65, 1979, Russian.

The influence of alcohol and smoking on the spermatogenic function of the testes and the prostate gland, on the content of 17-ketosteroids (17-ks) in the urine, and on the activity of blood phosphatases was studied in 1,104 men varying in ages from 18 to 40 years. The control group included 209 men. The results showed that the alcohol and smoking toxicity led to a decrease in the number of spermatozoids in 10^6/ml by 43.1-53.3 percent and a decrease in their mobility by 22.6-35.5 percent. Disorders in the prostate gland function decreased by 32.3-55.4 percent, the 17-ks concentration in the urine decreased by 59.7 percent, and blood acid and alkaline phosphatase activity decreased by 18.4 and 52.2 percent, respectively. These changes depend not only on the amount and duration of drinking and smoking, but on the onset of toxicity, and they may lead to reduced or complete sterility. (Auth. Abs.)

80–1489. Thomas, J.; Greig, M.; McIntosh, J.; Hunt, J.; McNeil, D.; Piper, D. W. **The Location of Chronic Gastric Ulcer.** *Digestion* 20(2): 79-84, 1980.

Ulcer size, age, sex, and alcohol, analgesic, and cigarette consumption (0, <20, and ≥20 daily) were studied in relation to the site of the ulcer in 215 patients with chronic gastric ulcer. Ulcer site was classified into upper (U), middle (M), and lower (L) thirds of the stomach, on X-ray films of air-contrast barium studies. The ulcers were situated in the upper third in 37 patients (17 percent), middle third in 90 patients (42 percent), and lower third in 88 patients (41 percent), i.e., significantly more in M and L compared to U ($p<0.001$). Initial ulcers did not differ from recurrent ulcers except in women where initial ulcers were more common in M and L, whereas recurrent ulcers were more common in U. The lesser curve position was more common for smokers as compared to nonsmokers ($p<0.05$). The independent variables, namely, alcohol, and analgesics (with the exception of smoking), were not determinants of ulcer site, size, or position once the patients were segregated by sex. In men only, there were interactions between L and lesser curve site ($p<0.004$), and L and smoking ($p<0.03$). (Auth. Abs. Mod.)

80–1490. West, K. M.; Erdreich, L. S.; Stober, J. A. **Absence of a Relationship Between Smoking and Diabetic Microangiopathy.** *Diabetes Care* 3(2): 250-252, March-April 1980.

The relationship between cigarette smoking and retinopathy and proteinuria was examined in a group of 973 subjects with diabetes (323 smokers, 650 nonsmokers). Other variables known to influence the risk of microangiopathy were also measured in a standardized fashion (e.g., duration of diabetes, blood glucose, blood pressure). The characteristics of smokers and nonsmokers were studied in detail. It was therefore possible to take into account the effects of confounding variables on the relationship between smoking and risk of microangiopathy. Rates of retinopathy, proliferative retinopathy, and proteinuria were very similar in smokers and nonsmokers. No significant differences in rates of microangiopathy by subgroups (light, moderate, heavy, and former smokers) were found. Indeed, heavy proteinuria was somewhat less common in smokers (12 percent) than in nonsmokers (15 percent). Smokers had slightly lower blood glucose values and lower blood pressure ($p=0.03$). (Auth. Abs. Mod.)

See also, **80–1399**

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–1491. Akinkugbe, O. O. **The Smoking Problem in Nigeria.** WHO Expert Committee on Smoking Control, Geneva, October 23-28, 1978, 1978, 2 pp.

Consumption of cigarettes in Nigeria is estimated to be 12.50 billion in 1977-78. Most smokers are male and between 18 and 40 years of age. Only 1 percent of the women smoke. Illegally imported cigarettes represent about 20 percent of the total consumption. Approximately 75 percent of the cigarettes smoked are of the filter variety. So far the public has not been sufficiently alerted to the dangers of smoking. Health education efforts have not met the smoking challenge, and there is no legislation against smoking in Nigeria.

80–1492. Allegrante, J. P.; O'Rourke, T. W.; Tuncalp, S. **A Multivariate Analysis of Selected Psychosocial Variables on the Development of Subsequent Youth Smoking Behavior.** *Journal of Drug Education* 7(3): 237-248, 1977-1978.

The purpose of this study was to determine if interaction effects of selected psychosocial variables could be utilized as indicators of subsequent smoking behavior among 2,090 (864 male and 1,226 female) 7th grade students who had originally identified themselves on a survey as having never smoked. Interaction effects of several psychosocial variables selected for study (e.g., parental and peer smoking habits, parental attitude toward smoking, knowledge of the Surgeon General's Report on Smoking) were found to be indicative of subsequent smoking behavior. (Auth. Abs. Mod.)

80–1493. Batsch, F.; Mueller, G. **Nichtrauchen am Arbeitsplatz — Eine Umfrage unter Rauchern und Nichtrauchern im Verkehrswesen. [Nonsmoking in the Workplace. A Survey of Smokers and Nonsmokers in Transportation.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 153(2): 218-222, 1979, German.

In the framework of a campaign for the elimination of smoking in the workplace, about 3,000 employees (of all ages and occupational categories) of the East German State Railways answered a questionnaire about their attitudes toward smoking. Specific questions concerning transportation, but also general questions about further restrictions of smoking, were asked. As expected, the nonsmokers (54 percent of the 2,500 usable answers) gave positive answers to all questions on smoking restriction. On the average, 50 percent or more of the smokers gave positive answers. For example, 70 percent of the smokers would cease smoking at work if their nonsmoking colleagues requested it. It is concluded that, in general, most smokers are not against sensible measures of reducing smoking in public places. On the basis of this opinion survey, a program for further reduction of smoking in the transportation sector is presented.

80-1494. Battegay, R.; Muehlemann, R.; Hell, D.; Zehnder, R.; Hoch, P.; Dillinger, A. **Alkohol, Tabak und Drogen im Leben des jungen Mannes. Untersuchung an 4082 Schweizer Rekruten betreffend Suchtmittelkonsum im Zivilleben und wahrend der Rekrutenschule. [Alcohol, Tobacco and Drugs in the Lives of Young Men. A Study of Drug Abuse in 4082 Swiss Recruits in Civilian Life and During Military Training.]** Sozialmedizinische und Paedagogische Jugendkunde, Volume 14, Basel, 1977, 149 pp., German.

To estimate the extent of drug abuse in Swiss youth, two multiple choice questionnaires were submitted to 4,082 healthy recruits at the beginning of their military schooling, and to 2,626 original recruits (64.3 percent) during the last 3 weeks of school. A standardized personal interview was conducted by a physician with 1,166 recruits (28.5 percent of the original sample) 4-8 weeks after the first questionnaire was filled out. The data show that 60.2 percent of the original sample smoked in civilian life and that 17.4 percent of them smoked >24 g tobacco/day. The difference between the smoking habit in civilian life and in the military school was insignificant. Family problems (divorced or separated parents, forced marriage) increased the risk of higher drug consumption by the recruits. The influence of the parents was evident in that the percentage of recruits who smoked >24 cigarettes/day increased from 7.3 to 25.8, when the number of smokers in their families increased from 0 to 100 percent. The percentage of heavy smokers (>24 g tobacco/day) was higher (47.6) among subjects who started smoking at age 13-15 than among those (4.5) who started smoking at age 20 or older. The percentage of heavy smokers increased from 6.21 in subjects who did not drink alcohol to 48.7 in those who consumed over 350 g alcohol/week. Subjects with a relatively high income, representatives from the lower socioeconomic classes, and subjects who left their parents' house at an early age run the highest risk of becoming addicted to tobacco. The overwhelming majority of smokers were motivated by imitating other smokers or by habit. It is concluded that the smoking habit presents one of the most serious social and medical problems in Switzerland.

80-1495. Bennett, D.; Levy, B. S. **Smoking Policies and Smoking Cessation Programs of Large Employers in Massachusetts.** *American Journal of Public Health* 70(6): 629-631, June 1980.

To assess the current extent of programs and policies to facilitate smoking cessation among employees, 128 large employers in Massachusetts were questioned in mid-1978. Eight-four (66 percent) responded. Fifty-four (64 percent) of the respondents had designated jobs or work areas in which smoking was prohibited usually because of potential danger to products or equipment. Seven (8 percent) of the employers provided counseling, and ten (12 percent) provided smoking cessation programs for those employees who wanted to quit smoking. There is a clear need for the development and evaluation of workplace policies and programs aimed at reducing smoking. (Auth. Abs.)

80-1496. Binnie, C. D.; Comer, A. K. **The Effect of Cigarette Smoking on the Contingent Negative Variation (CNV) and Eye Movement.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 69-75.

The contingent negative variation (CNV) and eye movement of 18 male and 18 female cigarette smokers (aged 19-55 years) were recorded before and after smoking one king-size filter-tipped cigarette. Electroencephalogram (EEG) measurements were taken and CNV was recorded between the vertex and the left mastoid sites using chlorided silver stick-on electrodes. Smoking behavior (e.g., puff number, volume, duration; interpuff interval; butt length) was also recorded. A mood adjective check list was administered before and after the experiment. Scores were obtained for the factors of aggression, anxiety, surgency, pleasantness, concentration, fatigue, vigor, and sadness. Eighteen subjects showed an increase in CNV magnitude after smoking (mean 104.7 percent increase), and 18 subjects showed a decrease (mean 29.6 percent decrease). Both these changes were statistically significant ($p \leq 0.05$), and the mean increase was significantly greater than the mean decrease ($p \leq 0.05$). The mean presmoking CNV and eye movement magnitudes and number of eyeblinks were compared with the postsmoke mean values. In a second experiment with 35 subjects, 3 showed a decrease and 7 an increase in CNV magnitude ($p \leq 0.1$), all other subjects showing no significant change. For eye movement during the CNV, eight subjects showed a significant decrease and two an increase. Five subjects showed a significant increase in eye blinks during postsmoking blocks and four showed a decrease. There was a tendency for subjects showing a significant CNV change to have smoked more intensely than those showing no significant change, and a CNV increase was associated with greater smoking intensity than a CNV decrease. There was no apparent relationship between smoking behavior and personality for these subjects. The only scores on the mood adjective checklist which changed significantly were those for concentration and vigor, both of them showing a decrease.

80-1497. Chambliss, C.; Murray, E. J. **Cognitive Procedures for Smoking Reduction: Symptom Attribution Versus Efficacy Attribution.** *Cognitive Therapy and Research* 3(1): 91-95, 1979.

The purpose of the present study was to evaluate two cognitive procedures for reducing smoking. In the first phase of the study, a procedure was used to get the subjects (28 male and 18 female college students of whom half were Internals (9 and below) and half were Externals (11 and above) on Rotter's Locus of Control Scale), to attribute withdrawal symptoms to a placebo pill rather than smoking reduction. None of the main effects or interactions reached statistical significance. In the second phase of the study, a procedure was used to get the subjects to attribute their partial success to themselves rather than the placebo pill (drug vs. self-efficacy). The self-efficacy condition resulted in much greater reduction in smoking than the drug efficacy condition. This result was statistically significant, with an alpha of 0.05. There was also a significant interaction between the efficacy manipulation and locus of control. For the Internal subjects, reduction of smoking was significantly greater in the self-efficacy condition than in the drug efficacy condition. (Auth. Abs. Mod.)

80–1498. Cherry, N.; Kiernan, K. E. **A Longitudinal Study of Smoking and Personality.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 12-18.

To demonstrate the correlation between personality scores collected in late adolescence and the smoking behavior in the following years, 2,753 survey members were followed up every 2 years since their birth. They completed questionnaires for personality and smoking behavior at the ages of 16, 20, and 25. Extraverts and neurotics started smoking earlier (before the age of 16) and became heavy smokers at the age of 25 (73 percent male and 62 percent female compared to 44 percent male and 32 percent female stable introverts). Stable extraverts were more likely to quit smoking (or at least inhale less) at the age of 25 than neurotic introverts. Neuroticism was not related to changing the smoking habits in women, but emotionally stable men were more likely to quit smoking at the age of 25. Both men and women extraverts were more likely to give up the habit than introverts. Heavy smokers (>20 cigarettes/day) at the age of 20 were less likely to quit at the age of 25 than light smokers.

80–1499. Colletti, G.; Kopel, S. A. **Maintaining Behavior Change: An Investigation of Three Maintenance Strategies and the Relationship of Self-Attribution to the Long-Term Reduction of Cigarette Smoking.** *Journal of Consulting and Clinical Psychology* 47(3): 614-617, 1979.

After receiving an identical, nonaversive treatment package, 42 subjects (mean age, 37.68; mean number of years smoked, 20.66; mean baseline smoking rate, 32.11 cigarettes/day) were randomly assigned to one of three maintenance strategies: modeling, participant observing, or a self-monitoring control. These subjects showed a mean smoking rate of 46 percent of baseline at 1 year and no significant relapse between 6 months and 1 year. Significant group differences and correlations with attribution and other questionnaire measures were found. A second sample of 32 treated subjects, who observed the original subjects during their maintenance strategy sessions, achieved highly similar results on correlational smoking and abstinence rate measures, providing some support for the replicability of the results. (Auth. Abs. Mod.)

80–1500. Comer, A. K.; Creighton, D. E. **The Effect of Experimental Conditions on Smoking Behaviour.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 76-86.

The effect of various experimental and social conditions on smoking characteristics was investigated. Subjects smoked one type of cigarette (1) while being observed through a one-way mirror in a relaxed atmosphere; (2) while electroencephalographic (EEG) recordings were being made; (3) 1 year after initial EEG recording, either under relaxed laboratory conditions or during EEG recording; and (4) while being surreptitiously observed in a coffee lounge. The effect of smoking deprivation was also studied in the experiment conducted 1 year after initial EEG recording. Observations confirmed that people smoke the same type of cigarette in different ways under different conditions, as has always been suspected. Intensity of smoking was increased during an EEG experiment and decreased in a social situation, when compared to smoking in a solitary, relaxed laboratory environment. Smoking behavior was stable with time and unaffected by a short period of smoking deprivation. It seems reasonable to suggest that people tailor their smoking behavior to obtain a desired physiological effect and that their requirements will differ according to the situation in which they find themselves. Care must always be taken if results of laboratory smoking, during an experiment, are extrapolated to a wider context, and it is always necessary to consider the ways in which the experimental procedure may itself alter smoking behavior.

80–1501. Creighton, D. E.; Lewis, P. H. **The Effect of Smoking Pattern on Smoke Deliveries.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 301-314.

The effects of puff shape, puff volume, and interval between puffs on deliveries of total particulate matter (TPM), nicotine, and carbon monoxide (CO), the ratios of the deliveries of these components, and the filtration efficiencies for TPM and nicotine were analyzed by use of a puff duplicator which smokes cigarettes to measure smoking patterns recorded on paper tape by the smoking analyzer and data logger. A king-size filter-tipped cigarette typical of those found on the U.K. market was used in this study. The data from four puff volumes and four puff intervals (i.e., sine-shaped, early peak triangle, late-peaked triangle, and square puff profiles) were tabulated. The nicotine delivery was almost independent of puff shape at least when averaged over all levels of interval and volume. For TPM and CO the deliveries are greatest from the late triangle shape and least for the early triangle shape. Examination of the delivery ratios revealed that the TPM to nicotine and CO to nicotine ratios increased with increasing puff volume, while the TPM to CO ratio is approximately constant. The delivery of carbon monoxide was shown to increase as the interval between puffs increased up to a maximum interval of 1 min and then decreased again. The TPM to nicotine ratio decreased systematically with increasing puff interval. The filtration efficiency of the filter to nicotine did not change with increasing intervals between puffs, whereas the filtration efficiency to TPM increased almost linearly with increasing intervals between puffs.

80–1502. Dodds, A. M.; Rankin, D. W.; Hill, D. J.; Gray, N. J. **Attitudes and Smoking Habits of Doctors in Victoria.** *Community Health Studies* 3(1): 28-31, 1979.

A 1977 survey of 275 physicians in the State of Victoria, Australia, revealed an overall cigarette smoking rate of 14 percent, the same percentage as in the national survey in 1974. The rate in the general population was 35 percent. Forty-six percent of the physicians reported that they never smoked regularly and a further 30 percent were ex-smokers. The main reasons given by ex-smokers for stopping were predominantly concerned with health. Among these reasons were the general nature of the health risk (65 percent), the observed effect of smoking on their own health (51 percent), and to set an example for their patients or their own children (20 percent). Of the 39 physicians still smoking cigarettes in the recent survey, nearly one-fifth had switched to a low-tar brand in the past 2 years. The survey also showed that in 1977 physicians believed still more strongly in the role of cigarettes in the causation of heart disease, lung cancer, and chronic bronchitis than physicians in the 1974 survey.

80–1503. Einstein, S.; Epstein, A. **Cigarette Smoking Contagion.** *International Journal of the Addictions* 15(1): 107-114, 1980.

A ministudy was carried out in Tel-Aviv, Israel, with 50 male and female adults 17-64 years of age who were regular cigarette smokers (≤ 5 cigarettes/day to ≥ 80 cigarettes/day for 1-39 years, median 11 years) to investigate the potential role(s) which contagion plays in smoking. Notwithstanding their reports that they were introduced to smoking by others 40 percent of the time, only 12 percent reported initiating someone else to smoking. The report of self-initiated cigarette use is congruent with most views about smoking, but not regarding drug use. As long as drug use intervention utilizes contagion as a concept, deviance as a classification and varieties of isolation as control techniques will be associated with drugs, drug users, and drug use. By definition, socially accepted substances (e.g., tobacco, alcohol, food) have no source of contagion by others. (Auth. Abs. Mod.)

80–1504. Eiser, J. R.; Gossop, M.; van der Pligt, J. **Drug Attitudes and Discrimination Between Drugs Among a Group of English Schoolchildren.** *Drug and Alcohol Dependence* 5(1): 57-62, January 1980.

This study investigates the attitudes of a group of 185 London schoolchildren (80 boys and 105 girls) aged 15-16 years toward a number of legal and illegal drugs, and examines the ways in which they discriminate between these drugs. Heroin and LSD were seen as the most dangerous drugs, and alcohol and cigarettes as the safest. Subjects who had a negative attitude toward drugs in general regarded amphetamines and alcohol (both $p<0.05$) and cannabis and tobacco (both $p<0.01$) as more dangerous than subjects with a neutral or prodrug attitude. There was also a sex difference: boys claimed to know more about drugs, were more likely to know a cannabis user and to have been "really drunk" than the girls. The implications of these results for drug and alcohol education programs are discussed. (Auth. Abs. Mod.)

80–1505. Ek, S.; Froberg, J.; Kagan, A.; Karlsson, C.-G.; Levi, L.; Palmblad, J. **Cigarette Smoking, Nicotine Content, Cognitive Factors and Psychosocial Stressors: An Experimental Study of Physiological and Psychological Effects in Smokers, Non-Smokers and Abstainers.** Reports From the Laboratory for Clinical Stress Research, Karolinska Institute, Report No. 61, Stockholm, August 1977, 29 pp.

The present study was carried out to test hypotheses of the effect of nicotine on physiological and psychological variables under conditions that could be relevant to real life. It aimed to simultaneously measure several variables believed to be (a) interrelated, (b) influenced not only by nicotine but also by psychosocial factors, and (c) to be of potentially pathogenic significance. The following five hypotheses were tested: (1) smokers differ from nonsmokers with respect to their baseline and "stress" levels of psychosocial variables; (2) the nicotine content (high or low) of cigarettes affects the magnitude of psychosocial reactions; (3) smoking high-nicotine cigarettes increases catecholamine excretion, thyroid activity, plasma lipids, and self-ratings of subjective arousal, and decreases serum iron concentration; (4) smoking low-nicotine cigarettes induces some or all the reactions mentioned above, but to a lesser degree; and (5) those smoking high-nicotine cigarettes (as compared to low nicotine cigarette smokers or abstainers) are less "stressed" by psychosocial stressors. The study subjects included 129 men aged 20-24 years, defined either as smokers (5-15 cigarettes/day), or as nonsmokers (former smokers of ≤ 5 cigarettes/day who had not smoked for ≥ 3 months). Study data were tabulated and discussed. Very little support for the first hypothesis was found. The second hypothesis was incorrect. As for the third hypothesis, it was correct for adrenaline excretion, thyroid activity, and self-ratings of subjective arousal. However, it was incorrect for noradrenaline and thyroid activity. The fourth hypothesis was true for adrenaline excretion and fatigue, out of the factors mentioned for hypothesis three, and it was also true for dizziness and nausea. Finally, the fifth hypothesis was found to be incorrect.

80–1506. Glover, E. D. **Decreasing Smoking Behavior Through Subliminal Stimulation Treatments.** *Journal of Drug Education* 9(3): 273-283, 1979.

The general purpose of the study was to determine whether subliminal perception could be used as a means for altering cigarette smoking behavior. The study contained a total population sample of 72 female subjects. The experimental situation consisted of three different films, shown at three different sessions, with subliminal stimulation for the treatment group. The control group viewed the three films without the subliminal stimulation. The entire association between smoking and quitting smoking was presented subliminally. After an analysis of the data collected, a comparison of the differences between the control and experimental groups revealed that there was no significant difference in regard to base cigarette count between groups after the treatment had been administered. Thus, smoking behavior was not altered through subliminal perception as carried out in this study. There was evidence that smoking behavior was altered, but it was an unpredicted change, as some of the subjects decreased their smoking patterns. (Auth. Abs.)

80-1507. Goldberg, J. **Raucherberatung in der Schule—Erfahrungen in einer Stadt der DDR. [Influencing Smoking Habits at School—Experiences in a Town of the German Democratic Republic.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 148: 35-43, 1977, German.

Attempts to modify the smoking habits of high school students in the East German city of Schwedt were initiated in 1974 by the head of the local chest clinic. In May 1974, a survey of all 998 8th and 10th grade students in 12 high schools showed that 50.2 and 54 percent of the students, respectively, were smokers. Of the smokers in grades 8 and 10, 17 and 47 percent, respectively, smoked >5 cigarettes/day. These findings led to the inclusion of a lecture on nicotine abuse in 8th grade curriculum. The students learned about the risks of smoking, the difficulties of smoking cessation, and the increasing number of young smokers. In the following two academic years, the teachers, with the help of a lung specialist, began discussing smoking problems with students. In addition, seminars for teachers and parents were conducted to provide basic information for educating children about smoking. Influencing parents to change their attitudes toward their children's smoking became necessary when it was found that 27 percent of all 14-year-olds and about one-half of all the 16-year-olds were offered cigarettes by their parents. Overall, the percentage of smokers in the 8th grade fell from 50.2 percent in 1974 to 37.8 percent in 1976. From this 3-year-long health education program, it is concluded that a far-reaching change in the smoking habits of adolescents can be achieved only by continuous, systematic, and close cooperation of the medical staff with teachers and parents.

80-1508. Golli, V. **Enquete sur l'Habitude de Fumer Chez les Etudiants. [Survey of Smoking Habits in Students.]** *Bulletin of the International Union Against Tuberculosis* 51: 579-585, 1976, French.

The most important results of a smoking survey of the Craiova University, Romania, students are analyzed. About one-third (29 percent) of the 2,321 students surveyed were smokers. While only one out of five (21.3 percent) of the freshmen were smokers, the proportion of smokers in the 5th school year was double (about 39 percent). Approximately 5 percent of the smokers started smoking before age 15 and about 42 percent started smoking before enrolling at the University, that is, at or before age 18. Of the female students, 21 percent started smoking at 18 years of age. The regular cigarette consumption of 56 percent of the smokers was <10 cigarettes/day, and 8 percent smoked >1 pack/day. The ratio of male:female smokers of 16 to 20 cigarettes/day was nearly 6:1 (21.43 vs. 3.82 percent). Among the smokers of >20 cigarettes/day, the male:female ratio was 9.40:1.53 percent. Curiosity was cited most frequently (by 63 percent of all smokers) as the initial motivation for smoking. More female than male smokers indicated boredom and disappointment as their initial motivations. Enjoyment was the factor predisposing about 53 percent of male smokers to continue their tobacco habit, while 18.32 percent of female smokers vs. 9 percent of male smokers indicated desire to be that factor. A large proportion (81 percent) of the students, the nonsmokers significantly more often than the smokers, estimated that smoking cigarettes was dangerous to your health. A smaller proportion (75 percent) of the students answered yes to the question of whether smoking shortens one's life. About 84 percent of the smokers and 81 percent of the nonsmokers considered passive smoking in the classroom to be dangerous. About 73 percent of the students answered affirmatively to questions of whether smoking was a contributory factor in lung cancer or chronic bronchitis incidence. Of the study subjects, 75 percent found the worldwide antismoking campaign justified. Three-fourths of the students desired more active press participation in the antismoking campaign, while 18 percent of the smokers were opposed to expansion of information on smoking risks.

80-1509. Hay, D. R. **Cigarette Smoking by New Zealand Doctors: Results From the 1976 Population Census.** *New Zealand Medical Journal* 91(658): 285-288, April 23, 1980.

In the 1976 New Zealand population Census, 3,584 male and 505 female doctors aged 25 years and over responded to a question on cigarette smoking. Twenty percent of men and 17 percent of women doctors smoke cigarettes regularly compared with 40 percent of men and 30 percent of women in the general population over 25 years. Cigarette smoking by male doctors has fallen from 37 percent in 1963 to 20 percent in 1976. There has been a minor increase in smoking by women doctors since 1972. Obstetricians smoke more heavily (27 percent) than other specialty groups but have reduced their smoking since 1972. Male physicians have the lowest rate of smoking (16 percent). About one in four of those in other professions is a cigarette smoker compared with about half those in other occupational groups such as laborers, and transport and service workers. (Auth. Abs.)

80-1510. Iammarino, N.; Heit, P.; Kaplan, R. **School Health Curriculum Project: Long Term Effects on Student Cigarette Smoking and Behavior.** *Health Education* 11(2): 29-31, March-April 1980.

The purpose of this study was to assess the attitudes toward cigarette smoking and concomitant behavior in eighth grade students, 3 years after participation in either the School

Health Curriculum Project (SHCP), or the traditional health education instruction (THI), or after no health education instruction (NHI) throughout schooling. The study was conducted in three Ohio cities. The total population was 1,117 students (SHCP, n = 395; THI, n = 562; NHI, n = 160). SHCP students achieved significantly ($p<0.0001$) higher mean scores on smoking attitude scales one (health concern, cost) and three (positive smoke attributes) than either THI or NHI students. Students who participated in the SHCP also achieved significantly higher mean scores on smoking attitude scale two (nonsmokers' rights), scale seven (destiny control, independence), and scale eight (rationalization)—($p<0.0001$, $p<0.01$, and $p<0.0001$, respectively) than did THI, but not NHI students. Of the students who participated in the SHCP, 49 percent indicated that they were nonsmokers, compared to 43.4 percent of THI students, and 43.3 percent of NHI students. On the other hand, only 15.5 percent of the SHCP students claimed to be smokers, while 26.4 percent of the THI students, and 24.8 percent of the NHI students were considered smokers. The percentages of occasional smokers were 35.7 (SHCP group), 30.1 (THI), and 31.8 (NHI). Although the data collected in this study revealed that five of the smoking attitude scales were highly significant in favor of the SHCP, and a significant relationship did exist between smoking behavior and the SHCP, it is not plausible to say that the SHCP alone accounted for the differences. However, it does appear that the SHCP has a positive impact on the smoking attitudes and behavior of eighth grade students and does account for some of the differences.

80–1511. Ippolito, R. A.; Murphy, R. D.; Sant, D. **Staff Report on Consumer Responses to Cigarette Health Information.** Federal Trade Commission, Bureau of Economics, August 1979, 68 pp.

This report provides a detailed statistical examination of the nature and extent of consumer reactions to cigarette health information programs. The study analyzes per capita cigarette and tar and nicotine consumption data for the 50-year period 1925-1975, relying primarily on data collected by the National Center for Disease Control profiling the lifetime smoking histories of 12,000 Americans. The study has been structured to estimate the long-run impact of changes in smoking behavior on cigarette consumption and life expectancy. The report employs a statistical model of consumer smoking behavior to identify the key factors that have influenced smoking decisions over time and among individuals. These possible influences include economic factors, such as cigarette prices and consumer income, a variety of demographic characteristics, and cigarette health disclosures and attendant publicity. Among the results were: (1) health publicity and disclosures have significantly affected per capita consumption since 1964; (2) consumer and producer reactions to health publicity have lowered per capita intake of tar and nicotine by 45 percent between 1964 and 1975; (3) while the decline in per capita consumption observed in 1953 was due to a reduction in the intensity of smokers' habits, after 1964 the proportion of adults smoking declined by 8 percent, but no evidence was found that health publicity affected the average number of cigarettes smoked; (4) about 12 percent of the individuals who started smoking after 1964 quit within 10 years in response to health publicity; and (5) information concerning the dangers of cigarette smoking has added about 0.6 years to the lives of adult Americans.

80–1512. Izard, C. **Neuropsychology and Tobacco.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 44-53.

Neuropsychology and tobacco are discussed in very general terms with affecting imagery encompassing olfactory sensations, anxiety, personality, orality, impulsivity, sexuality, compulsion, and sociality. It is concluded that the fight against tobacco is apparently based on ethical grounds and it is alluded that smokers are a persecuted minority.

80–1513. James, W. G. **Health Education for Adults.** *Preventive Medicine* 9(2): 281-286, March 1980.

This paper briefly reviews current adult education programs for primary prevention of cancer and offers suggestions for more effective programing in the future. Programs now underway include those sponsored by the American Cancer Society, the American Health Foundation, the National Cancer Institute, trade unions, and industries. It is estimated that anticigarette smoking programs account for 90 percent of the present programs in primary cancer prevention, while the remaining programs concern overexposure to the sun and skin cancer, and cancers caused by occupational hazards and other chemicals. Recommendations for the future include (1) the need for a definitive statement separating cancer causes firmly established by research from those suspected cancer causes calling for additional research, and (2) the need for evaluation of smoking cessation clinics so that the relative effectiveness of various approaches can be determined. (Auth. Abs. Mod.)

80–1514. Jamison, R. N. **Cigarette Smoking and Personality in Male and Female Adolescents.** *Psychological Reports* 44(3, Part 1): 842, June 1979.

This study examined the relationship between psychoticism and cigarette smoking and determined differences in correlations between cigarette smoking and personality between male and female adolescents. The subjects were 1,282 schoolchildren between the ages of 13 and 16 years (781 boys and 501 girls). Personality was measured on the Junior Eysenck Personality Questionnaire which includes the Psychoticism, Extraversion, Neuroticism, and Lie scales. The self-report Antisocial Behavior Questionnaire was employed to measure deviancy and smoking behavior. The study showed that psychoticism had the highest correlation with smoking. Extraversion showed a significant positive relationship with smoking, while neuroticism was almost unrelated to smoking behavior. Stronger correlations were noted for females between psychoticism and extraversion and the smoking items. It is concluded that psychoticism might be a better predictor of smoking by adolescents, particularly for girls, than the extraversion or neuroticism scores.

80–1515. Keyl, K. **Rauchgewohnheiten bei Schulern eines Landkreises. [Smoking Habits of Students in a Rural School District.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 153(2): 209-217, May 1979, German.

A survey of 1,502 8th to 12th grade students (14-18 years of age) of the Hagenow Rural School District, East Germany, was conducted to determine the students' smoking habits, and onset and motivation for smoking. The sample included 768 girls and 734 boys from both rural and urban areas. The survey established that 40.8 percent of the students were smokers. The percentage of smokers increased from 26.7 in the 8th grade to 50.7 in the 10th, then decreased to 45.3 in the 11th grade, and increased again in the 12th grade. There were 45.4 percent smokers in purely rural schools vs. 38.1 percent in the small town schools. Over one-half (57.9 percent) of all smokers started smoking at age 14 or 15 and 13.8 percent at the age of 12 or younger. Most of the girls (64.1 percent) and boys (51.6 percent) started smoking at age 14 or 15. Of the smokers, 37.9 percent were motivated by "the desire to keep up with the other smokers," while 30 percent gave "enjoyment" as their reason for smoking. A plurality (46.4 percent) of the nonsmoking students (36.3 percent of the boys and 57 percent of the girls) cited the absence of any desire for smoking as their main motivation. The expense of smoking was the most important reason given for not smoking by 33.8 percent of the boys and 21.0 percent of the girls. Of all the smokers, 19.6 percent (26.7 percent of the boys and 8.4 percent of the girls) smoked over 5 cigarettes/day. Overall, the grades of nonsmokers were better than those of smokers. Only 8.3 percent of the smokers but 16.2 percent of the nonsmokers had the highest average grade and only smokers had the lowest average grade.

80–1516. Lampman, J. H. **Women Hospital Workers Smoke. (Letter).** *New England Journal of Medicine* 299(15): 836-837, October 12, 1978.

Concern over the adverse effects of cigarette smoking in women prompted a survey of the smoking habits of 397 workers in the Cleveland Metropolitan General Hospital. Results indicate that between 36 and 52 percent of employees in the female-dominated occupations of nursing, nurses' aide, social service, and secretarial work smoked cigarettes while in the hospital, compared to 29 percent of all U.S. women. This figure is about 10 times the proportion of smokers among the medical house staff and students ($p<0.001$) and twice the proportion of smokers in the full-time medical staff ($p<0.01$). Of the 20 women among the physicians and students surveyed, none smoked in the hospital. Since the hospital employees were predominantly younger workers, these facts represent an example of the national trend toward increased smoking among young women.

80–1517. Lento, A. G. **Teaching About the Nature and Abuse of Alcohol, Drugs and Tobacco.** Capital Area School Development Association, State University of New York, Albany, 1978, 231 pp.

This publication, written by a professor of education, is designed to provide basic information on drug abuse which may be used in the classroom by both the beginning and the established teacher. Lesson plans, structured conversations, objectives, brief drug use histories, bibliographies of resource organizations, and topical outlines and pertinent information are included. For tobacco education, a "smoking machine" experiment using two bottles, a pump, rubber stoppers, glass tubing, and filter cigarettes is used to illustrate filter ineffectiveness in removing tars and other smoke contaminants.

80–1518. Malcolm, S.; Shephard, R. J. **Personality and Sexual Behavior of the Adolescent Smoker.** *American Journal of Drug and Alcohol Abuse* 5(1): 87-96, 1978.

A study of 38 male and 25 female students (aged 14-17 years) showed that the adolescent smoker differed little from the nonsmoker in terms of scores on the Cattell 16 personality factor inventory. In particular, there was no evidence of tension, extroversion, or reversal of sexually-biased personality characteristics. Students who smoked were more active sexually than their nonsmoking colleagues. The smokers dated members of the opposite sex more frequently than the nonsmokers, this difference being statistically significant for the girls. They also engaged in sexual intercourse more frequently and had a greater number of sexual partners, these differences being statistically significant for the boys. The sexual promiscuity of the adolescent smoker is probably related more to liberal attitudes and a propensity for risk-taking behavior than to uncertainty regarding sexuality. The smoking behavior of the students was equally influenced by the habits of parents and peers. If both parents or the majority of the student's friends smoked, there was a fourfold increase in the risk that the student would also smoke ($p<0.01$ and $p<0.001$, respectively). In this age group the decision to start smoking regularly may depend more on the influence of parents and friends than on inherent personality characteristics. (Auth. Abs. Mod.)

80–1519. Mello, N. K.; Mendelson, J. H.; Sellers, M. L.; Kuehnle, J. C. **Effect of Alcohol and Marihuana on Tobacco Smoking.** *Clinical Pharmacology and Therapeutics* 27(2): 202-209, February 1980.

Tobacco smoking covaried with alcohol consumption in male social drinkers over 15 days of unrestricted alcohol availability. Increased tobacco smoking was associated with alcohol consumption in occasional, moderate, and heavy smokers. Tobacco smoking was not systematically related to marihuana smoking even though both drugs were often smoked at the same time. During 10 days of concurrent access to tobacco, alcohol, and marihuana, tobacco smoking continued to covary with alcohol consumption rather than with marihuana smoking. Marihuana smoking appeared to be independent of alcohol consumption patterns. (Auth. Abs.)

80–1520. Moody, P. M. **The Relationships of Quantified Human Smoking Behavior and Demographic Variables.** *Social Science and Medicine* 14A(1): 49-54, January 1980.

This study, based on a sample (n=517) of adult medical patients at a southeastern medical center who were current users of cigarettes, reports the quantified aspects of smoking behavior and their relationship to sex, race, age, education, income, and socioeconomic status of these patients. Patients from lower socioeconomic groupings had a greater number of puffs, shorter intervals between puffs, longer puff duration, shorter cigarette butts, and more daily tar and nicotine intake than the subjects from upper socioeconomic groupings. Males and older patients had longer puff duration, greater puff volume, and more daily tar and nicotine intake than their counterparts. Implications of this and related studies upon the development of a social policy toward smoking and health are discussed. (Auth. Abs.)

80–1521. Mueller, R. **Soziokulturelle Voraussetzungen von Alkohol- und Tabakerziehungsmassnahmen. [Sociocultural Requirements for Alcohol and Tobacco Education Measures.]** *Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften* 35(1/3): 237-244, March 1979, German.

The effectiveness of a drug (alcohol and tobacco) education program is evaluated by its ability to modify behavior, i.e., to reduce or prevent drug consumption. The few reliable evaluations of such programs are not encouraging primarily because (1) most drug education programs are based on a too narrow concept of education, and (2) they fail to take into account social and cultural requirements. Solid empirical evidence exists of the effects of cultural background, social expectations, and emotions on the reaction to drug consumption. Contradictions between educational aims and the significance attributed to alcohol and tobacco by a pupil account for the failure of drug education. These contradictions are schematically presented at three levels of behavior: recognition, evaluation, and decision. Alcohol and tobacco education programs that cannot solve these basic contradictions are doomed to failure. To overcome the contradictions, a number of theses are proposed. First, education must be clearly related to the pupil's expectations, e.g., show a relationship between smoking and reduced vitality. Second, education must indicate that any drug abuse inhibits development and self-realization of an individual.

80–1522. National Clearinghouse for Smoking and Health. **Adult Use of Tobacco—1975.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, June 1976, 134 pp.

The purpose of the 1975 smoking survey conducted by the National Clearinghouse for Smoking and Health was to evaluate possible changes in knowledge, attitudes, beliefs, and behavior concerning smoking that have taken place since 1970 and to provide indepth information so that smoking programs could become maximally effective. Approximately 12,000 personal or telephone interviews were conducted to obtain survey data which included (1) demographic data; (2) survey of attitudes; (3) survey of perception; (4) smoking history; (5) dosage; (6) cigars, pipes, snuff, and chewing tobacco; and (7) changes in smoking behavior. The survey showed that the proportion of smokers was smaller than that predicted from the 1970 survey data. Men and women divorced or separated were more likely to smoke than individuals from other marital categories. Smoking rates are lowest for individuals who never went to high school, but at other educational levels there was a decrease in the proportion of smokers at higher educational levels. Male white collar workers are less likely to be smokers, but in women an opposite relationship was found. Men in relatively affluent families are less likely to smoke while women in this group are more likely to be current smokers. Awareness of the relationship between smoking and health has greatly increased over the past decade. A current smoker is more apt to discount the health hazards of cigarette smoking, to agree that no further action should be taken about smoking, to disagree with statements that certain groups should be exemplary by refraining from smoking, and to believe what happens to them mostly depends on luck or fate. More nonsmokers than smokers believe being afraid of gaining weight keeps people from quitting cigarettes. Two of three smokers are concerned about possible health effects. Smokers are more likely than nonsmokers to believe that some cigarettes are more hazardous than others. Two-thirds say they have never been advised to quit smoking or cut down. Since 1970 there has been a decrease in smoking prevalence with a corresponding decrease in the perception of prevalence. There is a decrease in the proportion of individuals who think their doctor is a smoker, and it was found that smokers are more likely than nonsmokers to perceive that their doctor is a smoker. Smokers are less likely to report knowing someone whose health has been affected by smoking. Men have smoked for more years than have women. Fifty-seven percent of smokers are expected to be smoking 5 years from now. Many current smokers smoke their cigarettes in such a way as to maximize their hazards. Men smoke in a more hazardous way than do women and smoke more cigarettes/day than do women. Most current smokers use filter-tipped king size cigarettes. There was very little change since 1970 in the use of snuff and chewing tobacco. There was a decrease in pipe smoking since 1970. About 18 percent of the men surveyed smoke cigars. Twelve percent of male current smokers have at some time changed to or smoked more cigars, and 18 percent changed to pipe or smoked a pipe more often.

80–1523. National Institute on Drug Abuse. **Marihuana and Health. Seventh Annual Report to the U.S. Congress From the Secretary of Health, Education, and Welfare, 1977.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institute on Drug Abuse, DHEW Publication No. (ADM) 79-700, 1979, 56 pp.

The nature and extent of marihuana use in the United States and current attitudes and beliefs about marihuana are discussed. In addition, some comparisons are made between marihuana use and the use of licit drugs such as alcohol and tobacco. For instance, slightly more than one in five of the 12- to 17-year-old group (22.3 percent) were current cigarette smokers. Among high school seniors, however, the percentage who had used cigarettes in the month preceding the survey only slightly exceeded the percentage who had used marihuana in a like period (38.4 percent reported cigarette use vs. 35.4 percent who reported marihuana use). It should, however, be emphasized that cigarettes were used much more frequently than was marihuana. Daily use of cigarettes (28.8 percent of 1977 seniors) was more than three times that of marihuana. Among high school seniors, two-thirds of the Class of 1977 disapprove of smoking marihuana regularly, about the same number who disapprove of daily use of either ≥ 1 pack of cigarettes or of daily alcohol use. Various human effects (e.g., cardiovascular, pulmonary, immunological, genetic, psychomotor), therapeutic aspects, and future marihuana research directions are also discussed.

80–1524. Oddoy, A.; Hieronymi, U. **Beziehungen zwischen sozialen Merkmalen und Verhaltensweisen in der Schwangerschaft. 1. Mitteilung: Einfluss des Kinderwunsches auf einige Verhaltensweisen und Gewohnheiten. [Relationship Between Social Characteristics and Behavior in Pregnancy. 1. The Effect of Planned Pregnancy on Certain Behaviors and Habits.]** *Zentralblatt fuer Gynaekologie* 100(13): 877-884, 1978, German.

In order to determine whether unplanned pregnancy is a health risk to the fetus or child in terms of maternal behavior before and during pregnancy, 500 East Berlin women were interviewed just after childbirth and prior to release from the clinic. Questions were asked about nutrition, drug use, alcohol consumption, and smoking, as well as demographic and social characteristics. Of those interviewed, 59.6 percent planned their pregnancy. Significant differences were found between the two groups of women with respect to three out of eight behavior factors, one being smoking. Before becoming pregnant, 41 percent of the women who wanted a child were smokers compared to 46 percent of those who did not want a child. During pregnancy, nearly one-third of the women in both groups quit smoking. However, there were 15 percent more women who continued to smoke >7 cigarettes/day in the group with unplanned pregnancy than in that with planned pregnancy. In the group of planned pregnancy, 32 percent of the mothers smoked <7 cigarettes/day vs. 24 percent in the group of unplanned pregnancy. Multipara smoked more frequently than primipara, e.g., 46 percent vs. 21 percent smoked >7 cigarettes/day in the group of women with unplanned pregnancy. Within each social group, more heavy smokers were found among the women with unplanned pregnancy. However, the differences between the groups are small so that unplanned pregnancy cannot be considered as a significant risk factor for the fetus and newborn child.

80–1525. Powell, G. E.; Stewart, R. A.; Grylls, D. G. **The Personality of Young Smokers.** *British Journal of Addiction* 74(3): 311-315, September 1979.

In a sample of 808 middle-class children more than half of the boys and girls had tried smoking cigarettes by the age of 15 years. These children were found to be particularly extreme on the Psychoticism (positive), Neuroticism (positive), and Lie scale (negative) dimensions of personality, and also somewhat high on the Extraversion dimension. This pattern is identical to that of children who misbehave and do not conform in a general sense. The implications for an antismoking campaign were briefly discussed. (Auth. Abs.)

80–1526. Purcell, I.; Lloyd, D.; Hardes, G.; Alexander, H.; Leeder, S. R. **Children and Smoking.** *Australian Family Physician* 8(12): 1284-1286, December 1979.

An experimental educational program to reduce childhood cigarette smoking in the Hunter health region (New South Wales) is described. It is an example of the efforts currently being made to tackle childhood cigarette smoking as an increasing problem. The program involves 6,300 children, aged 10-12 years, and is designed to modify their knowledge, attitudes, and behavior relating to smoking. Nine different strategies are used in the program, ranging from the presentation of elementary physiological facts about smoking and their health effects, to using role play to simulate behaviors concerned with making decisions on whether or not to use cigarettes. The effectiveness of the program is currently being evaluated. It is hoped that the prevalence of smoking will be lower among children who have received the antismoking education.

80–1527. Rawbone, R. G.; Keeling, C. A.; Jenkins, A.; Guz, A. **Cigarette Smoking Among Secondary School Children in 1975: Its Prevalence and Some of the Factors That Promote Smoking.** *Health Education Journal* 38(3): 92-99, 1979.

A questionnaire relating to smoking habits, respiratory symptoms, and health attitudes was administered to 10,498 secondary schoolchildren ages 11-17 in an outer London borough in 1975. The results reported in this paper suggest that the prevalence of smoking in boys, when compared with previous studies, has fallen, while in girls it has remained steady. Despite this, boys continue to smoke more heavily and at an earlier age than girls. At age 11, 51 percent of boys and 34 percent of girls admitted to having smoked a cigarette. Factors which appear to influence smoking rates are discussed, including parental, sibling, and peer smoking habits, amount of income from both pocket money and part-time earnings, immigration, and, to a lesser extent, academic achievement. Relating the children's smoking habit to the number of parents who smoked indicated that where neither parent smoked, 46 percent of children were nonsmokers and 9.6 percent were regular smokers; where one parent smoked, 35.5 percent were nonsmokers and 16.2 percent were regular smokers; and where both parents smoked, 30.3 percent of children were nonsmokers and 20.5 percent were regular smokers. These group differences are highly significant ($p<0.001$). When data were analyzed in terms of parents' and child's sex and smoking category, the results indicated that boys were influenced equally by either parent smoking ($p<0.05$), whereas girls were influenced to a significantly greater extent by their mother smoking than by their father smoking ($p<0.005$). (Auth. Abs. Mod.)

80–1528. Schachter, S. **Pharmacological and Psychological Determinants of Smoking.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 208-228.

The roles of pharmacological and psychological determinants of smoking in justification of the low-nicotine cigarette campaign are discussed. Data from several studies indicated that the variations in smoking rate were an attempt by smokers to regulate nicotine and that by regulating smoking the smoker avoided withdrawal symptoms. Other experiments where urinary pH was manipulated with either placebo or bicarbonate showed that smoking increased with stress and placebo, while with bicarbonate stress had no effect on smoking (interaction $p<0.01$). It was concluded that the smoker under stress smokes to replenish the nicotine supply but not to relieve anxiety. The justification for the low-nicotine cigarette campaign is questioned.

80–1529. Schwarz, W. **Die Erziehung zum Nichtrauchen als Bestandteil der Gesundheitserziehung. [Smoking Education as a Part of Health Education.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 153(1): 163-170, 1979, German.

The need to make nonsmoking a generally recognized norm of behavior is the central point of health education. To achieve this goal, it is necessary to eliminate the conditions which support the tradition of smoking. An important cause for the development of the smoking habit during adolescence is the young person's belief that smoking provides an external expression of adulthood. The socialist society offers a unique opportunity to overcome the smoking tradition. However, a steadily increasing number of young people start smoking at an early age even in the East German society indicating that the problem of developing a healthy self-confidence still exists. Health education of youth must emphasize the attractiveness of nonsmoking and present credible arguments to influence public opinion in favor of nonsmoking. As a rule, health arguments in favor of nonsmoking can be persuasive only if they are derived from the important life aspirations of the target group. Practical suggestions for antismoking programs are provided. The explanations and propaganda can be effective only in the framework of the total social education process.

80–1530. Taylor, D. H.; Blezard, P. N. **The Effects of Smoking and Urinary pH on a Detection Task.** *Quarterly Journal of Experimental Psychology* 31(4): 635-640, November 1979.

Performance was measured in a detection task over a period of 50 min. Subjects (56 males, aged 20-32 years) were either nonsmokers or smoked more than 15 cigarettes per day. No smoking was allowed during the task, but smokers were all asked to smoke a cigarette immediately before doing it. One hour previously, subjects had been given a double-blind dose of a substance which would shift the pH of their urine either upward or downward by small amounts sufficient to affect the rate of renal excretion of nicotine in the smokers. The pH manipulation had no effect on the performance of the nonsmokers, who improved steadily throughout the task. After some 40 min of performance, the deprived smokers showed less improvement than the nonsmokers, and the smokers whose urine had been made acid improved less than those whose urine had been made alkaline. The results are interpreted as evidence for a phasic effect on performance of small degrees of nicotine deprivation, which has consequences for theories of nicotine dependence. (Auth. Abs.)

80–1531. Thornton, R. E. (Editor). **Smoking Behaviour. Physiological and Psychological Influences.** Edinburgh, Churchill Livingstone, 1978, 419 pp.

This book is the result of the International Smoking Behavior Conference held at Chelwood Vachery, Sussex, England, in November 1977. The objectives of the conference were to explore some of the physiological and psychological effects of smoking which may be important in smoking behavior and motivation, and to relate these effects (where possible) to (a) differences between smokers and nonsmokers; and (b) the smoker's intake of tar, nicotine, and carbon monoxide, including the extent to which this intake can be influenced by changes in cigarette design.

80–1532. Warburton, D. M.; Wesnes, K. **Individual Differences in Smoking and Attentional Performance.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 19-43.

Published literature on individual differences in smoking patterns and its effect on attentional performance are reviewed. Results of two experiments designed to clarify the personality trait—smoking relation are described. The degree of extraversion and neuroticism in subjects (12 nonsmokers, 12 light smokers, and 24 heavy smokers) was assessed. Subjects were then tested for vigilance either with cigarettes of varying nicotine content or with nicotine tablets. Baseline vigilance scores and vigilance scores after 12 hours deprivation of cigarettes were calculated and correlated both with neuroticism and extraversion scores, and with results of a Smoking Motive Questionnaire. Subjects who had a high neuroticism score had poorer initial baselines for vigilance performance. However, their performance improved more than that of the subjects with a low neuroticism score in the 20 min following the medium- and high-nicotine cigarette. Analysis of the Smoking Motive Questionnaire replies revealed that subjects who had high neuroticism scores were sedative smokers, i.e., they smoked in situations when they were worried or angry. Comparison of the nicotine intake across cigarettes suggests that the subjects were compensating by puffing relatively harder at the low-nicotine cigarettes and relatively weaker at the high-nicotine cigarettes compared with the medium-nicotine cigarette. This finding is in agreement with other studies that have shown that subjects seek an optimum dose for a task and modify their smoking patterns to obtain this amount of nicotine. Evidence is presented for an interaction between vigilance, neuroticism, and nicotine. Some possible neurochemical bases for the interaction between nicotine and neuroticism are discussed.

80–1533. Williams, C. L. **Primary Prevention of Cancer Beginning in Childhood.** *Preventive Medicine* 9(2): 275-280, March 1980.

There is evidence suggesting that a large proportion of adult morbidity and mortality from major chronic disease, particularly cancer and atherosclerosis, might be prevented through educational strategies designed to motivate adoption of a healthier lifestyle beginning in childhood. The "Know Your Body" program is a behaviorally oriented prototype chronic disease prevention education program based on developmental capabilities and focused on facilitating appropriate personal health decisions concerning dietary and smoking habits at each age. Pilot studies of several approaches to smoking prevention among children at ages 12-14 years, including methods emphasizing mass media influences, parental smoking influences, and dynamic peer group activity and discussion groups have been conducted in the past 4 years. Only the latter approach was effective. This model has been expanded, refined, and renamed "Life Skills Training" (LST). Conducted during 10 weekly classroom sessions, students develop skills in a number of basic life skills relevant to problems confronting adolescents, including the issue of cigarette smoking but also going beyond it. Included are topics on self-image, decisionmaking, advertising techniques (including cigarette ads), coping with anxiety, communication skills, dating skills, and assertiveness training as well as myths and realities of cigarette smoking. Each skill is taught through the use of role modeling and behavior rehearsal. Preliminary data indicate that the LST approach is effective in preventing the onset of cigarette smoking. In a controlled trial of the 10-week LST intervention classes, the experimental group was smoking 3.5 times less (4 percent compared with 14 percent in the control group) than the control students at the end of the sessions. The effectiveness was greatest among younger (grade 8) students and least among older students (grade 10). (Auth. Abs. Mod.)

80–1534. Williams, D. G. **Effects of Cigarette Smoking on Immediate Memory and Performance in Different Kinds of Smoker.** *British Journal of Psychology* 71: 83-90, 1980.

Changes in performance resulting from smoking were assessed in 48 male cigarette smokers who were classified both by daily consumption (i.e., light smokers, ≤ 15 cigarettes/day; medium smokers, 16-25 cigarettes/day; heavy smokers, >25 cigarettes/day) and by relative desire for smoking in situations inducive of high or low arousal. Each took part in four first cigarette of the day conditions involving a sham smoking control and three cigarettes differing in nicotine delivery (i.e., 0.6 mg, 1.3 mg, and 1.8 mg). With increasing cigarette strength gains in letter cancellation speed on smoking increased although an inverted-U relationship was suggested; immediate memory accuracy progressively deteriorated once presmoking performance was controlled for. Smokers with greater desire to smoke in situations inducive of low arousal appeared to react more strongly to cigarettes and showed superior gain in cancellation speed on smoking. High cigarette consumption did not lead to between-day tolerance to cigarette effects. (Auth. Abs. Mod.)

See also, **80–1447, 80–1540**

SMOKING CESSATION METHODS

80–1535. Fagerstrom, K.-O. **Physical Dependence on Nicotine as a Determinant of Success in Smoking Cessation.** *World Smoking and Health* 5(1): 22-23, Spring 1980.

Smoking cessation appears to be greatly influenced by nicotine dependence as evidenced by degree of withdrawal symptoms. In order to reduce such withdrawal symptoms, 30 smokers were given Nicorette, a nicotine chewing gum. These smokers were compared to 30 smokers who received no nicotine substitute. The mean physical response to abstinence score for the Nicorette group was 4.5 and for the group without Nicorette the score was 5.8, a significant difference at the 5 percent level. There also seemed to be a relationship between quantity of Nicorette and individual abstinence symptoms: the more Nicorette, the less physical response to abstinence. The correlation coefficient reached 0.34, significant at the 5 percent level.

80–1536. Hackett, G.; Horan, J. J. **Partial Component Analysis of a Comprehensive Smoking Program.** *Addictive Behaviors* 4(3): 259-262, 1979.

This study evaluated the effects of a comprehensive program (consisting of several counseling strategies with and without focused smoking) for the treatment of cigarette addiction in 30 subjects (19 males and 11 females, averaging 23.87 years of age). All of the subjects had smoked >1 pack/day for an average of 6.70 years. Abstinence levels of 40 percent, verified by expired air carbon monoxide tests, were achieved in a 6- to 9-month followup period. A partial component analysis revealed that the comprehensive program was not significantly more powerful than a principle component—the focused smoking technique. Revisions in the program are suggested. (Auth. Abs. Mod.)

80–1537. Lamontagne, Y.; Annable, L.; Gagnon, M.-A. **Acupuncture for Smokers: Lack of Long-Term Therapeutic Effect in a Controlled Study.** *Canadian Medical Association Journal* 122(7): 787-790, April 5, 1980.

Two types of acupuncture therapy, one aimed specifically at smoking withdrawal and the other aimed at enhancing relaxation, were compared with self-monitoring in 75 healthy men who wished to stop smoking. The subjects were 20-50 years old and had been smoking 15-20 cigarettes/day for at least 3 years. During the 2 weeks following treatment there was no significant difference in the adjusted mean daily smoking rates of the subjects receiving acupuncture therapy of the two types, but their combined rate was significantly ($p<0.01$) lower than the rate of the subjects in the self-monitoring group. However, at 1, 3, and 6 months following treatment, there were no longer statistically significant differences between the three treatment groups in the adjusted mean smoking rates. At no time were there significant differences between the three treatment groups in the proportion of subjects who stopped smoking during the study. Although acupuncture appears to have become a popular treatment for cigarette smokers, its effectiveness remains to be proven in the treatment of tobacco addiction. (Auth. Abs. Mod.)

80–1538. Luban-Plozza, B.; Ammann, A. **Neue Wege der Raucherentwohnung. [New Methods of Smoking Cessation.]** *Psycho* 3(11/12): 561-572, 1977, German.

The article deals with the prevention of health damage from smoking. Previous research findings concerning goals of smoking therapy and methods of attitude and behavior modification in smoking withdrawal are discussed. It has been found that 75 percent of all ex-smokers revert to their former habit after smoking a single cigarette. While many smokers succeed in reducing their smoking to <10 cigarettes/day, tobacco consumption increases again soon after the end of the therapy. The authors' approach to smoking therapy is based on the concept of smoking as learned environment-conditioned behavior. Therapeutic methods should be tailored to the smoker's personality, not the reverse. Treatment of smoking patients during and after a hospital stay is described. The treatment program includes the patients filling out a smoking protocol during the hospital stay and after discharge. Group therapy in groups of 8 to 12 persons is also used. Sports activities, low-calorie diet, and placebos are recommended for ex-smokers. Relapse prophylaxis comprises booster sessions with the physician, and instruction of therapy helpers (mediators) in the family and at the workplace.

80–1539. McFall, R. M. **Smoking-Cessation Research.** *Journal of Consulting and Clinical Psychology* 46(4): 703-712, 1978.

Methodological problems associated with treatment research on cigarette smoking are explored, and possible solutions are discussed. The main problems considered are the selection and retention of subjects, the measurement of smoking (including laboratory and self-report methods, as well as correlates of smoking behavior), the design of treatment studies, and the interpretation and generalizability of experimental results. (Auth. Abs. Mod.)

80–1540. Wax, R. H.; Wax, M. L. **How People Stop Smoking: An Exploratory Study.** *Mid-American Review of Sociology* 3(1): 1-15, 1978.

Interviews were conducted with 32 persons (18 women and 14 men, median age 37 years) who had smoked at least one pack of cigarettes daily for 3 or more years and who had stopped smoking for at least 1 year. Different reasons for quitting smoking were given by the respondents. They included a serious illness which smoking would aggravate, perceptible respiratory problems, cost of cigarettes, conversion to a new religion, reading the Surgeon General's Report on Smoking and Health, self-perceived physical unattractiveness due to smoking, a friend's death from lung cancer, and being in a happier work environment. Few respondents said their decision was not based on a crucial event. Heavy smokers reported a period of acute discomfort after abstinence. Some avoided social situations where there was the temptation of resuming smoking (e.g., with alcoholic beverages), and others used substitute activities (e.g., chewing mints). Most smokers reported their abstinence as the result of their own determination. It was concluded that the clearest strategy for quitting smoking combined this process with changes in lifestyle, social status, and social relations.

See also, **80–1441, 80–1499**

TOBACCO MANUFACTURING AND PROCESSING

80–1541. Campbell, R. G. **Removal of Radioactive Lead and Polonium From Tobacco.** U.S. Patent No. 4,194,514, March 25, 1980, 5 pp.

Studies have shown that radioactive metals are found in cigarette smoke and in bronchial tissue of smokers at higher concentrations than in nonsmokers. Lead-210 (^{210}Pb) and polonium-210 (^{210}Po) can be removed from tobacco by treating uncured tobacco leaves with a dilute acid solution (0.01-1.0 percent by weight). The solution can also contain hydrogen peroxide and/or a wetting agent in amounts ranging from 0.03 to 3.0 and 0.05 to 0.2 percent by weight, respectively. In one experiment, the effect of several treatment solutions on ^{210}Pb removal was examined. Control tobacco (no treatment) leaves contained 0.28±0.04 pCi/g ^{210}Pb. The lowest ^{210}Pb content (0.0±0.06 pCi/g) was found when 1.0 percent nitric acid, 0.6 percent hydrogen peroxide, and a wetting agent were used. After this treatment, however, leaves were limp and brown. Another solution containing 1.0 percent acetic acid and a wetting agent resulted in 0.0±0.07 pCi/g ^{210}Pb with no adverse leaf effects. Treatment of tobacco leaves with similar solutions resulted in a ^{210}Po content of less than 0.03 pCi/g.

80–1542. Kameswara Rao, B. V.; Gopalachari, N. C. **A Note on the Pattern of Distribution of Polyphenols and Nicotine Within the Tobacco Leaf During Curing.** *Tobacco Research* 4(2): 65-67, December 1978.

This study was conducted to elucidate the pattern of distribution of chlorogenic acid, total flavonoids, and nicotine in the tobacco leaf and their variations during flue-curing and in different primings. From a bulk crop of flue-cured tobacco (Virginia Gold), 150 plants harvested in three primings (representing bottom, middle, and top stalk positions) were withdrawn from the barn during curing at six different stages of curing (harvested leaf, half-yellowed, yellowed, half-fixed, fixed, and cured). Chlorogenic acid increased from base to tip along the margins consistently in all the primings. Total flavonoids (such as rutin) increased from bottom to top stalk positions and in half-fixing stage, and decreased in fixed leaf and restoration of original leaves in the cured product. Within the leaf, the margins had higher nicotine levels than the portions near the midrib, and tips had higher nicotine levels than base portions. The middle portion near the midrib contained the least nicotine levels. Nicotine levels decreased from bottom to top leaves along the stalk. During curing, nicotine levels increased up to half-yellowing, decreased during half-fixing, and increased progressively up to the end of curing.

80–1543. Koiwai, A.; Kisaki, T. **Changes in Glycolipids and Phospholipids of Tobacco Leaves During Flue-Curing.** *Agricultural and Biological Chemistry Journal* 43(3): 597-602, March 1979.

Changes in the lipid components of tobacco leaves during flue curing were investigated. The contents of total fatty acid methyl esters and linolenate decreased considerably, though a temporary increase was observed at the early period of the yellowing stage. The amounts of digalactosyl diglyceride (DGDG), monogalactosyl diglyceride (MGDG), sulfoquinovosyl diglyceride, and phosphatidyl glycerol decreased markedly while phosphatidyl inositol, phosphatidyl serine, and phosphatidyl choline were relatively resistant to the degradation process. While most phospholipids had high proportions of palmitate, MGDG and DGDG were found to have high proportions of linolenate. A considerable decrease was observed in the ratio of linolenate in most polar lipids while the ratio of palmitate and stearate showed a significant increase. (Auth. Abs.)

80–1544. Roman, T.; Wierzba, M.; Nagorski, B. **Proby Wprowadzenia Substytutu Tytoniowego "Cytrel" do Krajanek Tytoniowych. [Experimental Blending of Cut Tobacco With the "Cytrel" Substitute.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* (1/4): 65-76, 1976, Polish.

The physical properties of Oriental and American tobacco blends with 10-30 percent Cytrel and of the cigarettes prepared from these blends were determined. In addition, the cigarette smoke was analyzed for harmful constituents. In comparison with pure tobacco cigarettes, the smoke of cigarettes made from Oriental or American tobacco blends with up to 30 percent Cytrel contained 14 or 22 percent less brut condensate, 7 or 10 percent less tar, 17 or 32 percent less total alkaloids, 12 or 34 percent less free nicotine, 10 or 20 percent less acetaldehyde, and 24 or 27 percent less total phenols, respectively. Thus, blending cut tobacco with Cytrel substitute reduces the content of certain harmful substances in the smoke. However, the taste and flavor of smoke from cigarettes containing more than 10 percent Cytrel deteriorate perceptibly. In contrast, the addition of 10 percent Cytrel to both tobacco varieties did not adversely affect the sensory properties of the smoke. Moreover, a beneficial effect of a 10 percent Cytrel addition to Oriental tobacco was perceived with respect to the taste of the cigarettes.

80–1545. Schmidt, O. K. **Lipid Removal From Tobacco.** United States Patent No. 4,200,113, April 29, 1980, 6 pp.

A method of extracting lipids from tobacco by use of water and water-miscible organic solvents, preferably lower alkanols, while substantially retaining residual structural characteristics and organoleptic properties in smoking, is described. In several experiments, a prewash of the tobacco sample with water followed by successive washings with 80 percent isopropyl alcohol plus 20 percent water results in a 60-82 percent reduction in the lipid content of the tobacco. (Auth. Abs. Mod.)

80–1546. Shapovalov, E. N.; Khariton, M. Sh. **Vliianie Zasorennosti Tabachnogo Syr'ia na Fraktsionnyi Sostav i Kuritel'nye Svoistva Rezanogo Tabaka. [Effect of Dustiness of Raw Tobacco Material on the Fiber Size Distribution and Smoking Characteristics of Shredded Tobacco.]** *Izvestiia Vysshikh Uchebnykh Zavedenii Pishchevaia Tekhnologiia* (4): 17-19, 1977, Russian.

Initial and final dustiness (i.e., q_i and q_f, respectively), fiber size distribution, flavor, and taste were determined in two series of nine shredded specimens of raw and processed Peremozhets tobacco variety from Moldavian SSR. Processing of tobacco leaves was done in a vacuum duster and a rotary sieve to remove extraneous admixtures (dust). Both raw and processed leaves were rehumidified to 16.5-17.0 percent moisture. As the initial dustiness q_i and the difference $\Delta q = (q_i - q_f)$ decreased, the difference $\Delta\Psi = (\Psi_f - \Psi_i)$ between the content of large size (0.5 mm) fibers in processed tobacco and that in raw tobacco decreased to vanish at $q_i = 5.0$ percent. The data show that the difference between taste rating of smoking products from raw tobacco and the rating of products from processed tobacco vanishes also at $q_i = 5$ percent. At $q_i = 10.2\text{-}11.8$ percent, the tobacco products' taste and flavor were rated the lowest, at $q_i = 1.5\text{-}2.0$ percent, the highest. In all cases, the flavor of processed tobacco was rated superior than that of the raw tobacco.

See also, **80–1326**

TOBACCO ECONOMICS

80–1547. Check, W. A. **The Strength of Tobacco—In More Ways Than One.** *Journal of the American Medical Association* 242(26): 2832-2833, December 28, 1979.

The commercial benefits of selling tobacco are evident, strangely enough, in health-related businesses. For example, 93 percent of nonclinic-affiliated pharmacies sold cigarettes in San Francisco in 1978, despite a 1973 California Pharmaceutical Association resolution that pharmacists should discourage the sale of tobacco products in pharmacies where they practiced. Hospitals have found cigarette sales good for giftshop business. Banned tobacco broadcast advertising has become sports sponsorship. Perhaps personal antismoking campaigns are equally or more effective than other methods.

80–1548. Clairmonte, F. F. **World Tobacco: Dynamics of Oligopolistic Annexationism.** *Economic and Political Weekly* 14(30-32): 1331-1344, August 1979.

This paper examines the origin, role, and ramification of conglomerate oligopolistic power in the world tobacco economy. The power of the tobacco transnational conglomerates (TTCs) is such that many developed and underdeveloped countries have been totally marginalized in the output and marketing decision processes. Underdeveloped capitalist countries supply 55 percent of world leaf tobacco; but their processed exports are almost nonexistent, they have no influence whatsoever in the design, output, and innovation of tobacco machinery, and their aggregate receipts from tobacco are based almost exclusively on the demand, response, and marketing decisions dictated by the TTCs. Such an evolution of the political economy of tobacco is not unique; its historic trends—global dominance by a handful of TTCs and relegation of peripheral economies to a dependent relationship under TTC hegemony—are common to a majority of the 25 odd commodities which dominate nonpetroleum exports of the underdeveloped countries. Likewise, the TTCs too are not unique; in their effective creation of barriers to new entrants by way of active nonprice competition with each other, generation of massive surplus cash flows, cross-subsidization into new product lines, and the building up of mutual self-reinforcing relations with other transnational conglomerates, the TTCs represent the ultimate stage in the development of oligopolistic capitalism in the last quarter of the 20th century. (Auth. Abs.)

80–1549. Delman, F. **Multilateral Trade: How Negotiations Affect Tobacco.** *Tobacco Reporter* 107(2): 44-45, February 1980.

Reduced maximum tariff rates in the European Community (EC) obtained in the Tokyo Round trade negotiations will play an increasingly significant role in U.S. leaf exports as the dollar value of U.S. leaf increases through the 1980's. It is estimated that the total gain to U.S. tobacco exports will be between 80 and 100 million dollars per year. In addition to the EC, seven other countries (including Canada, the Ivory Coast, and Israel) either reduced their rates or bound them at current levels. The United States, in return, agreed to tariff concessions on six tobacco items.

80–1550. Hoover, D. M.; Perkinson, L. B. **Flue-Cured Tobacco Harvest Labor: Its Characteristics and Vulnerability to Mechanization.** North Carolina State University, Department of Economics and Business, Economic Research Report No. 38, Raleigh, North Carolina, June 1977, 184 pp.

The composition of the 1972 flue-cured tobacco harvest work force in an eight-county area of eastern North Carolina was determined from a survey of a sample of households. Nonharvest employment and earnings were also measured. The worker and household distribution of projected aggregate labor displacement was simulated as were the probable work force adjustments to mechanization. Belt-wide projections were developed from study area analyses. The effect of a reduction in demand for tobacco was also analyzed. Over half of the 1972 hired work force was <18 years old, >two-thirds was black, and >half was female. Own-farm workers were older, about 60 percent were white, and the number of males exceeded females. Earnings of hired workers averaged 340 dollars, and own-farm workers had imputed earnings of 630 dollars. In the aggregate, harvest earnings represented only 6 percent of area earnings. For the average hired-worker vs. own-farm worker household, the harvest accounted for 8 vs. 15 percent of income, respectively. By 1980, with no change in acres in production, about one-third of households engaged in the 1972 harvest will have no harvest employment. The average household loss in earnings is projected at 4 percent for hired households and 7 percent for own-farm households before other adjustments are considered. Black households will generally lose more than white households. Nonfarm employment will probably be sought by only 10-15 percent of displaced workers. Perhaps an additional 5-10 percent will seek retraining. Potential out-migration will be increased by mechanization, but it appears to be no greater than the historical net migration stream of individuals from the area. While Aid for Families with Dependent Children benefits and bonus Food Stamps will be increased by mechanization, these will probably be <10 percent of the aggregate reduction in earnings. (Auth. Abs.)

80–1551. Humberd, D. R.; Mundy, S. D. **Current Issues Affecting the Program for and Marketing of Tobacco.** *Southern Extension Marketing Publication* (1): 1-6, September 1979.

Internal and external forces present major challenges to the tobacco industry. External forces identified are the smoking and health issue, taxation, social costs and benefits, international trade, labor, energy, and the environment. The smoking and health issue has resulted in a reduction in per capita cigarette consumption. Taxation of cigarettes has increased from 3 cents per pack in 1950 to as much as 21 cents per pack in some States. Benefits from tobacco production include smoker satisfaction and income benefits to stockholders and employees, but these benefits are offset by health care costs of users, nonsmoker dissatisfaction, and reduction in air quality. The relative position of the United States in the world tobacco market has eroded due to technological advances in developing countries and the effect of various tariff and nontariff trade barriers to market accessibility. Labor problems in the tobacco industry are caused by worker mobility, seasonal labor requirements, and the lag in technological and mechanization advances in tobacco processing. Tobacco is a very energy-dependent product because of the curing process. In 1978, fuel costs accounted for 17 percent of total expenses to produce an acre of flue-cured tobacco. Environmental pollution through agricultural chemical use is also a subject of concern for the tobacco industry. Forces within the tobacco industry that affect the marketing of the product are discussed under the categories of farm organization and the structure of tobacco production, the impact of the supply-control/price-support policy, marketing procedures, and the structure and economic philosophy of the industry.

80–1552. Maxwell, J. C., Jr. **How the Brands Ranked. Maxwell International Estimates.** *World Tobacco* (66): 57-60, October 1979.

Maxwell international estimates are presented for leading cigarette brands in Italy, France, Spain, Colombia, Venezuela, Malaysia, Algeria, Hong Kong, Finland, Puerto Rico, New Zealand, Singapore, El Salvador, Costa Rica, Sudan, Norway, Panama, and Guam. Market shares in each country for filter and nonfilter types and for leading brands are tabulated for 1976, 1977, and 1978. Total sales in million pieces are also reported.

80–1553. Naguib, M. R. **Tobacco Consumption in Egypt Expected to Continue Rising.** *Tabak Journal International* (4): 270-271, August 1979.

Tobacco consumption in Egypt in 1978 rose by about 8 percent over 1977, and cigarette production was up roughly 9.3 percent. While exports of tobacco products fell sharply from 215,385 kg in 1977 to 37,023 kg in 1978, the volume of imported cigarettes increased from 1.2 to 1.5 billion. The trend is toward smoking high-class cigarettes which represent 94.6 percent of total cigarette sales. In anticipation of an expected 10 percent annual increase in cigarette consumption, construction of a new cigarette factory which will produce 30 billion cigarettes per year is planned by the Eastern Tobacco Company.

80–1554. Tabak Journal International. **Sharp Swings in British Attitudes to North American Leaf.** *Tabak Journal International* (4): 273-274, August 1979.

Great Britain which, by tradition, has always been one of the best customers for U.S. and Canadian Virginia tobacco, has changed its buying habits, particularly since joining the European Economic Community and being compelled to accept the system of cigarette taxation imposed by this body. During the period from 1974 to 1978, there has been a considerable fluctuation in Britain's purchases of leaf tobacco from North America (United States and Canada), whereas there was a constant drop in the volume of purchases between 1974 and 1977. During 1978, Britain purchased three times as much leaf tobacco from the United States as in the previous year, and 50 percent more leaf tobacco from Canada. Price is a decisive factor for British buyers of leaf tobacco. (Auth. Abs.)

80–1555. Tobacco International. **Cigarettes in 1980: Stable Sales, Lower Tar.** *Tobacco International* 182(6): 9-11, 13, March 21, 1980.

A review of 1978 and 1979 cigarette sales trends indicates that in spite of health warnings and other restrictive measures, cigarette sales increased about 1.2 percent in 1979. The region of greatest sales activity remains that of low-tar brands. Low-tar ($\leq$15 mg tar) cigarettes now constitute well over 40 percent of the sales volume in the United States; more than half the now available brands are, in fact, low-tars. The low-tar ranges include 0-5 mg, 6-9 mg, and 10-15 mg, with the 0-5 mg range being introduced in the greatest number of brands.

80–1556. Tobacco Reporter. **Italian Tobacco: Not a Peak Year.** *Tobacco Reporter* 107(4): 45-46, 48, 50, 55, April 1980.

Italy's total tobacco area in 1979 is estimated at 58,880 hectares, 1.3 percent more than in 1978, but practically at the same level as 1977, when it decreased some 8 percent from 1976—the top year for tobacco in the last decade. Earlier estimates of the 1979 crop have recently been reduced due to adverse weather conditions during the harvesting stage. Production is expected to be 111,000 metric tons compared to 109,658 in 1978. A general increase in growers' prices is expected, except for Oriental tobaccos, to cover inflation. Tobacco leaf exports are expected to increase from 15,849 metric tons in the first half of 1978 to 18,811 in the same 1979 period, while imports should almost double (from 7,831 to 14,743 metric tons) for the same respective periods.

BILLS AND LEGISLATION

80–1557. Kornegay, H. R. **Tobacco Action Network is Vital to Prevent Restrictive Legislation.** *United States Tobacco Journal* 208(1): 16-17, January 10, 1980.

Since 1973, the antismoking movement has introduced nearly 800 smoking restriction bills in various States and succeeded in having about 70 enacted into law. If this pressure succeeds in stopping each American smoker from lighting up just one cigarette a day, cigarette consumption would drop by more than 18 billion units, personal spending for cigarettes would decline by more than half a billion dollars, Federal excise tax revenues would be reduced by nearly 70 million dollars, and State excise tax collections would slip by almost 110 million dollars. In this speech claiming that there has been an effective criminalization of smoking and smokers because of smoking restriction bills, the president of the Tobacco Institute urged wholesalers, retailers, and vendors to join the industry-organized program TAN (Tobacco Action Network) to fight against restrictive legislation. TAN is an organization which exists in 18 states and is composed of 15,000 people who come primarily from manufacturers.

80–1558. U.S. Senate, 95th Congress, 2nd Session. **Racketeering in the Sale and Distribution of Cigarettes. (S. 1487).** Committee on the Judiciary, Report No. 95-962 (to accompany S. 1487), June 28, 1978, 23 pp.

A bill to eliminate racketeering in the sale and distribution of cigarettes (S. 1487) was amended in eight sections including definitions (what constitutes a contraband cigarette), unlawful acts, recordkeeping and reporting (the authority given to the Secretary of the Treasury), penalties for violators (maximum fine increased from 10,000 to 100,000 dollars, and maximum imprisonment increased from 2 to 5 years), effect on state law, enforcement and regulations, and cigarette traffic.

80–1559. U.S. Senate, 95th Congress, 1st Session. **Racketeering in the Sale and Distribution of Cigarettes. (S. 1487).** Hearing Before the Subcommittee on Criminal Laws and Procedures of the Committee on the Judiciary, October 21, 1977, 1978, 193 pp.

Hearings were held by the Senate Subcommittee on Criminal Laws and Procedures of the Committee on the Judiciary on proposed legislation (S. 1487) to eliminate racketeering in the sale and distribution of cigarettes. Testimony focused on the organized smuggling of large quantities of bootlegged cigarettes by major crime groups. Bootlegging involves the transportation of cigarettes from low- to high-tax States without payment of tax. Studies show that organized crime now controls most of this illegal activity. Among the witnesses were Federal and State government officials, as well as tobacco industry spokesmen. A report: "Cigarette Bootlegging: A State and Federal Responsibility," produced by the Advisory Commission on Intergovernmental Relations, is included as part of the hearing.

GENERAL

80–1560. Abratt, R. **Smoking and Advertising. (Letter).** *South African Medical Journal* 55(25): 1015, June 16, 1979.

In an addendum to a previously published report on South African cigarettes, health education, and advertising (*South African Medical Journal* 55(19): 743-748, May 5, 1979), the author of this letter discusses cigarette advertising and the various images it portrays (e.g., smoking is sexy, sophisticated, prestigious, wealthy, successful) and proposes to counteract smoking advertising by both curbing advertising and by entering the marketplace with a health education campaign. According to the author, the smoking habit should be seen as filthy with serious health consequences, and as a graveyard not "good life" image. See also 79-0915.

80–1561. Barrett-Connor, E. **Preventive Medicine and Public Health in the People's Republic of China.** *Preventive Medicine* 8(5): 567-572, 1979.

Smoking is one of several public health issues discussed based on an observation tour of the People's Republic of China. A variety of filtered and unfiltered cheap (less than 10 cents a pack) cigarettes are produced in China, and cigarette smoking is widespread. Bronchitis and lung cancer are said to be increasing, the former being very common. Either from habit or chronic bronchitis, spitoons are everywhere. Although there has been some propaganda against smoking, there has been no mass educational effort.

80–1562. Blum, A. **Medicine vs Madison Avenue. Fighting Smoke With Smoke.** *Journal of the American Medical Association* 243(8): 739-740, February 22-29, 1980.

There is much that physicians can do to combat the propaganda barrage in support of smoking. For instance, physicians' offices should not have either ashtrays or magazines containing cigarette advertising. Physicians should use different techniques with patients to encourage them to stop smoking, for example, stress the unattractiveness of stained teeth and bad breath in counseling a high school girl, or the likelihood of more days lost from work in the case of a blue collar worker. Physicians can also participate in health promotion and prevention efforts outside the office, such as joining DOC (Doctors Ought to Care), an organization of health professionals whose aim is to curb unhealthy life styles. Medical science should adopt Madison Avenue techniques to combat smoking. Prosmoking advertising should be met with counteradvertising campaigns. The effectiveness of doing so was demonstrated by the dramatic decline in smoking which occurred between 1968 and 1970 when both prosmoking and antismoking ads appeared on television.

80–1563. Check, W. A. **Doctors, Let's Stop Dragging (Our Feet).** *Journal of the American Medical Association* 242(26): 2831-2832, December 28, 1979.

Direct physician participation in discouraging smoking among children, adolescents, and adults is advocated. The National Cancer Institute's "Helping Smokers Quit" kit and the American Cancer Society kit for physicians involve them either directly (the former) or indirectly (the latter) in helping patients quit smoking. To underline the success of individual counseling, several recent studies of intervention programs (e.g., Seventh Day Adventists' Five-Day Plan, Stanford Heart Disease Prevention Program) are cited.

80–1564. Hoeffler, D. F. **Smoking and Health in the Navy.** *U.S. Navy Medicine* 71(2): 15-19, February 1980.

An estimate of Navy/Marine Corps active duty personnel indicates that 296,883 men and 10,440 women (44 and 25 percent, respectively) are smokers. Among civilians, respective percentages are estimated as 38-39 vs. 30-34. The prevalence of smoking in active duty personnel is a source of concern in the Corps as smoking-related health effects are well-known. In one 5-year study, smoking-related diseases accounted for 20,176 admissions in Navy hospitals and for 13,536 annual man-day losses at a total cost of 4.4 million dollars. However, other estimates indicate that 123,000 (16 percent) of active duty personnel have quit smoking. Further, between 1968 and 1978, cigarette sales in Navy exchanges dropped 28 percent, from 28 to 20 million cartons. Smoking intervention programs are underway and withdrawal clinics have been established on shore as well as aboard ships.

344-691 O - 81 - 23

80–1565. Hoffmann, D.; Tso, T. C.; Gori, G. B. **The Less Harmful Cigarette.** *Preventive Medicine* 9(2): 287-296, March 1980.

Risk assessment shows that the adverse effects of cigarette smoking on human health are clearly related to the total consumption of cigarettes. Such dose—response is evident from data on the incidence of lung and larynx cancer and is also supported by results from bioassays with Syrian golden hamsters and mice. The concept of a less harmful cigarette, therefore, involves primarily a reduction of the quantities of tar and nicotine in cigarette smoke. The identification of tumorigenic and toxic tobacco smoke constituents and the study of their formation during smoking has guided the development of products with 40-60 percent reduction of sales-weighted tar and nicotine levels in most western countries. These changes have been effected mainly through development of efficient filter tips, selection of specific tobacco types, and modifications in tobacco blends, including the technology of reconstituted tobacco preparation. This presentation discusses details of the development of cigarettes with reduced levels of tar, nicotine, and carbon monoxide. It is emphasized that the risk for diseases associated with tobacco usage can be eliminated only by abstention from smoking, but that a risk reduction through product modification has been achieved and may be further improved. (Auth. Abs.)

80–1566. Kelly, N. R.; Cohen, F. L. **Smoking Policies in U.S. Hospitals: Current Status.** *Preventive Medicine* 8(5): 557-561, 1979.

Hospitals can and should restrict cigarette smoking to promote a healthy environment and to exemplify health promoting behavior. Questionnaires to determine policies and programs relating to smoking were sent to the administrative heads of 360 randomly selected hospitals across the United States. Returns were received from 330 hospitals for a response rate of 91.7 percent. Official smoking policies were in effect in 94.0 percent of the hospitals responding, but only 1.2 percent of the hospitals banned smoking. Smoking cessation programs for patients were offered in 12.1 percent of the hospitals, while 18.5 and 23.0 percent had programs for the community and for hospital employees, respectively. Visitors, patients who smoked, and physicians were seen as most resistant to smoking restrictions. The majority of hospitals (56.0 percent) continued to sell cigarettes on the premises. Hospitals in States whose economies are involved with tobacco were more permissive than the others to a significant degree. Since the hospital administration is the primary force in implementing smoking policy, the smoking behavior of the administrator may affect institutional smoking policies. It does affect their attitude about hospital employee smoking and the health professional's responsibility about smoking to a significant degree. Thus, hospitals are not demonstrating leadership in creating safe nonsmoking environments for the patients, nor in offering smoking education programs to the community. (Auth. Abs. Mod.)

80–1567. Khosla, T. **Obesity, Smoking and Health.** *Community Medicine* 1(3): 221-228, 1979.

The validity of the "desirable body weight" standards at present widely used by the medical profession is examined in relation to physical fitness in the age range 20-40 years. These standards are found to agree closely with body weights of American army recruits in 1914-1918, German soldiers in 1935, and Olympic sportsmen at Munich in 1972. They are also found to be valid in relation to physical fitness tests in the civilian population. However, obesity is inversely related to smoking and, although obesity is hazardous, its hazards are dwarfed in comparison with those of smoking. The use of body weight standards for smokers can therefore be misleading and it is concluded that they are of value in physical fitness assessments for nonsmokers only. Further research is needed on morbidity and mortality in relation to obesity within groups of smokers and nonsmokers in middle age groups. (Auth. Abs.)

80–1568. Kozlowski, L. T.; Herman, C. P.; Frecker, R. C. **What Researchers Make of What Cigarette Smokers Say: Filtering Smokers' Hot Air.** *Lancet* 1(8170): 699-700, March 29, 1980.

Because of pervasive attention concerning the smoking habit and its health effects, it is not surprising to find that many smokers lie about their habit. For this very reason, withdrawal clinic researchers often use biological assays to confirm the patient's claim of stopping smoking. In one group of heart patients, for example, self-reports indicated that 63 percent no longer smoked, but urine assays indicated that 16-20 percent of these were still smoking, even 18 months after a heart attack. These misleading self-reports are not limited to smokers. People who are overweight, or who are guilty of other usually correctable behavioral sins are also likely to give similarly misleading self-reports. Apparently both smokers' statements and what researchers discern from these statements should be viewed with skepticism.

80–1569. Kretzschmar, R. M. **Smoking and Health: The Role of the Obstetrician and Gynecologist.** *Obstetrics and Gynecology* 55(4): 403-406, April 1980.

The role of the obstetrician and gynecologist in influencing the health care behavior of women, including their attitudes toward smoking, is discussed. Women are now smoking at about the same rate as men (35-40 percent of the population). In addition, the amount of tobacco smoked by women has increased dramatically. The effects of smoking on pregnancy include lower infant birthweight and a doubled incidence of spontaneous abortion. Research indicates that deaths associated with smoking and pill usage per 100,000 fertile women jump from less than 2 in the 25- to 29-year age range to 58 in the 40- to 44-year age range. The antismoking effort of obstetricians and gynecologists should include (1) self-education, (2) education of other members of the health care team, (3) patient education, and (4) community action.

80–1570. Kunze, M.; Gredler, B.; Herberg, D.; Kunze, M. J.; Schoberberger, R.; Vutuc, C. **Forschungsergebnisse zum Thema "Rauchen und Gesundheit" 1974 bis 1979. [Research Results on "Smoking and Health" 1974 to 1979.]** In: *Smoking and Health in Austria.* Federal Ministry for Health and Environmental Protection, 3rd Edition, 1980, December 1979, 107 pp., German.

A survey is presented of the research on smoking and health performed at the Institute of Hygiene of the University of Vienna. The bulk of the research is in the field of medicine. Results of the most important studies on (1) cancer of the lung, bladder, and pancreas; (2) other smoking-associated diseases; (3) smoking and pregnancy, and (4) passive smoking are reported. Psychology, epidemiology, health economics, and health information dissemination are other fields of research discussed in connection with smoking. The psychologic research included development of a smoker behavior model, and analysis and therapy of smoker behavior. Economic loss due to smoking-induced diseases was the principal research subject in the field of health economics. Dissemination of health information through mass media and interpersonal communication was examined as one of the most important methods of reducing smoking-associated diseases. Other cancer prevention methods such as smoking cessation, smoking withdrawal, cigarette modification, and tobacco products marketing control were explored. Epidemiologic research included surveys of schoolchildren and clients of a smoking counseling center regarding their knowledge of the risks of smoking and their general attitude toward smoking.

80–1571. Lancet. **Smoking and Health.** *Lancet* 1(8172): 835, April 12, 1980.

World Health Day, April 7, 1980, had a theme of "Smoking or Health: The Choice is Yours." The emphasis was on having smokers' rights end where they clash with the rights of nonsmokers. However, the British government's small increase in tobacco duty, imposed March 26, 1980, warrants greater imagination on the part of antismoking activists. On April 7, the Action on Smoking and Health announced new evidence that people in the United Kingdom wanted more nonsmoking areas in public places and that smokers were still seriously unaware of the risks they ran. A public opinion survey at the end of February found that 4 percent of those polled thought there should be a complete ban on smoking in restaurants and cafes, with a further 40 percent in favor of separate nonsmoking areas. Over half those polled thought smoking should not be allowed in post offices and banks (56 and 54 percent, respectively), with a further 8 percent in each case favoring separate areas. Of those polled, 70 percent thought that people should be able to work in a smoke-free environment (24 percent thought smoking should not be allowed at all in offices, factories, and other work places; a further 48 percent thought there should be separate areas).

80–1572. Lancet. **The Government and the Tobacco Lobby.** *Lancet* 1(8172): 833, April 12, 1980.

The talks between the Department of Health and Social Services and the tobacco industry in Great Britain that were to have produced an updated version of the 3-year-old voluntary agreement on advertising by March 31, 1980, continue, to no dismay on the part of the advertisers. The Health Under-Secretary, who is in charge of the negotiations, is interested in progressive advertising reduction, vivid health warnings on cigarette packs, and phased withdrawal from sports sponsorship. However, it is accepted that self-regulation and voluntary agreement are preferable to legislation. The strategy of tackling health problems and not interfering with a profitable industry continues to be discussed.

80–1573. Lenfant, C.; Liu, B. M. **(Passive) Smokers Versus (Voluntary) Smokers.** *New England Journal of Medicine* 302(13): 742-743, March 27, 1980.

Since the 1964 Surgeon General's Report on Smoking and Health, innumerable scientific studies have attempted to determine the effects of smoking. However, it is important to recognize that most of the evidence for deleterious health effects has come from studies of voluntary smokers, despite that inhalation of cigarette smoke may also be involuntary (passive). Further, the evidence that passive smoking in a general environment has health effects is often sparse, incomplete, and sometimes unconvincing. However, one recent study has demonstrated a reduction in measures of small airways of healthy nonsmokers exposed to cigarette smoke in the workplace. Eventually this quantitative measurement of a physical change in passive smokers may stimulate the search for a public smoking policy that is equitable and based on scientific evidence.

80–1574. Lucas, A. O.; Erinoso-Francis, A. O. **Smoking and Health in Nigeria.** WHO Expert Committee on Smoking and Its Effects on Health, Geneva, December 9-14, 1974, 16 pp.

This paper reviews the available information about the use of tobacco in Nigeria and the role of Nigeria as a grower, producer, exporter, and importer of tobacco. The occurrence of the health hazards associated with smoking in Nigeria is also discussed. Total cigarette production amounts to 1,200 million items per month, two-thirds of which are consumed locally. Smoking surveys show that Nigerians are not heavy smokers; with an estimated average consumption of about 10-20 cigarettes/person/month, Nigeria has one of the lowest recorded rates of smoking in Africa (compared to a rate of 265 cigarettes/person/month in the United States). The prevalence of smoking is higher in urban than in rural populations, higher in men than in women, and highest in the third decade of life.

80–1575. MacLean, N. **Smoking and Acclimatisation to Altitude. (Letter).** *British Medical Journal* 2(6193): 799, September 29, 1979.

It is reported that prior subjective conclusions of significantly less altitude sickness during skiing and mountaineering expeditions among smokers who either abstained or smoked less, than among nonsmokers, are apparently confirmed in a study on the effect of stopping smoking for 48 hours on oxygen availability in pregnant women (*British Medical Journal* 2(6186): 355-356, August 11, 1979). It is suggested that the British Olympic Association consider the economic advantages of substituting controlled smoking for altitude training in the pre-Olympic training program. See also 80-0720.

80–1576. Mahfouz, M. **The Egyptian Anti-Smoking Action.** WHO Expert Committee on Smoking Control, Geneva, October 23-28, 1978, 1978, 4 pp.

The Egyptian government has taken steps to implement a National Antismoking Campaign which was started by the Ministry of Health early in 1978. An interdisciplinary committee from various government agencies was formed whose objective was to formulate a program of action for an antismoking campaign. The immediate target was to reduce the number of smokers by 10 percent by the end of 1978, and by 50 percent during the following 5 years. The antismoking program planned included the use of the cinema and television, the press, as well as warning notices in public places, and education.

80–1577. Mecoy, R. **"If I Stop Smoking Will My Chest Get Better?"** *Australian Family Physician* 8(9): 971-973, 975, September 1979.

There is encouraging evidence available to those who want to know if the considerable effort of giving up smoking will be rewarded. Following cessation of smoking, according to many studies, there is a progressive decline in respiratory symptoms, such as chronic cough and sputum production, to a level similar to nonsmokers. An improvement in lung function has also been found for smokers who have chronic bronchitis and ex-smokers who have peripheral airways disease. To derive these benefits of smoking cessation, a smoker must have three main objectives: (1) find a compelling motive, (b) modify smoking behavior, and (3) adopt a positive attitude toward one's self as a nonsmoker. Physicians and smoking cessation programs are other important resources for the smoker who wants to quit.

80–1578. Mettlin, C. **Prerequisites of Successful Lifestyle Intervention.** *Social Science and Medicine* 13A(5): 559-562, 1979.

Evidence suggests links between lifestyle and disease (e.g., smoking and cancer, smoking and heart disease), but not all lifestyle interventions are equally likely to succeed. The four possible prerequisites in lifestyle intervention discussed here include issues of the certainty of our knowledge, the strength of the disease-lifestyle relationship (e.g., smoking, alcohol, and oral cancer; smoking, oral contraceptives, and heart disease; smoking, asbestos exposure, and lung cancer), the availability of outcomes measures (e.g., cessation of smoking and reduction in lung cancer risk), and the accessibility of lifestyle-related institutions (e.g., television, workplace). A variety of lifestyle phenomena are discussed with respect to the presence or absence of prerequisite conditions. (Auth. Abs. Mod.)

80–1579. National Institute of Environmental Health Sciences. **Basic Concepts of Environmental Health.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, DHEW Publication No. (NIH) 77-1254, 1977, 41 pp.

This is a general review of basic environmental health concepts. Among the topics discussed are atmospheric pollutants, multiple pollutant sources (e.g., food, water), problems of special environments (e.g., home, hospital), toxic waste disposal, carcinogenesis, mutagenesis, pollutant detection and measurement, and human development (i.e., placental transmission of substances from mother to embryo and to fetus). Concerning the latter, it is suggested that prenatal exposures may be estimated by examining the placenta for crucial residues or critical enzyme activity. For example, the enzyme activity elicited by cigarette smoking by pregnant women can be qualitatively measured in this way.

80–1580. Neumann, G. **Westeuropaisches Arbeitstreffen zur Bekampfung des Rauchens, London, 1. bis 3. Marz 1978. [Western European Workshop for Smoking Control, London, March 1-3, 1978.]** *Praxis der Pneumologie* 32(7): 491-494, July 1978, German.

Antismoking measures recommended by an international workshop for adoption by the participating countries are reported. The workshop was sponsored by the International Union Against Cancer and included representatives of the WHO and the International Union Against Tuberculosis. The measures include limitation or prohibition of advertisements for tobacco products, health warnings printed on cigarette packs, restrictions on smoking in public places, printing the tar and nicotine contents on each cigarette pack, and taxation of cigarettes. These restrictive measures must be accompanied by an effort to inform the public about the following: (1) there are fewer smokers than nonsmokers in any population; (2) nonsmoking is healthy; (3) smoking cessation is achievable in many cases; and (4) such symptoms as cough, expectoration, and short breath improve shortly after smoking cessation. The information must convey the health risks and the economic disadvantage of smoking vs. the social, psychological, and environmental advantages of nonsmoking. Target groups for an antismoking campaign are indicated. The physicians' responsibility toward their smoking patients is emphasized. Presentation of the smoking problem to children and adolescents in the framework of general health education is recommended.

80-1581. New Zealand Medical Journal. **Alcohol, Smoking and Drugs.** *New Zealand Medical Journal* 90(649): 467-468, December 12, 1979.

Alcohol consumption, smoking, and drug interactions are briefly discussed in this editorial. One important interaction between smoking and drugs concerns theophylline used for acute and chronic asthma or bronchitis. Smokers are prone to both conditions. For a therapeutic effect smokers need one to one-and-a-half times as much theophylline as nonsmokers. Similarly, smokers need up to 40 percent more pentazocine, an analgesic, than nonsmokers. Smoking also interacts with imipramine, glutethimide, frusemide, and propranolol. On the other hand, in normal therapeutic doses smokers are less drowsy than nonsmokers when taking diazepam and chlorpromazine. Such interactions are something the prescriber must be aware of and counsel accordingly.

80-1582. Pharmaceutical Journal. **Pharmacists Warn Against Smoking.** *Pharmaceutical Journal* 224(6072): 552, May 10, 1980.

Pharmacists in Canada are using a label to warn patients that the efficacy of some drugs (e.g., dextropropoxyphene, pentazocine, and imipramine) may be affected by cigarette smoking. The new warning is part of a campaign by the Canadian Pharmaceutical Association to involve pharmacists in community health services.

80-1583. Wake, F. R. **Preventing the Onset of Smoking.** In: Wakefield, J. (Editor). *Public Education About Cancer: Recent Research and Current Programmes.* UICC Technical Report Series, Volume 45, Geneva, 1979, pp. 24-35.

Knowledge of the normal psychological development of children and adolescents can be used to prevent the onset of smoking. The newborn child should be protected from parental cigarette smoking because early unhealthy behavioral patterns are being established. Among the means for preventing smoking in adolescents are identifying the danger of smoking for the teenager, convincing the peer group of the hazards of smoking, demolishing the idea that smoking is a proof of maturity, and using peers to aid the antismoking campaigns. Other issues discussed are the use of idealism in youth (using famous athletes), and urging teenagers to pursue their goals in life without the hazards of smoking.

80-1584. Webster, R. M. **"Smoking" as a Cause of Death. (Letter).** *Southern Medical Journal* 72(6): 770, June 1979.

It is stated that if smoking were accepted by vital statisticians as a cause of death, and were used by physicians on death certificates, these certificates might portray a more accurate cause of death when smoking is a factor (e.g., emphysema, lung cancer). Duration of both the disease causing death and smoking, in years, could be noted to better reflect the impact of smoking on health. It is suggested that questions on smoking be included in the 1981 census.

80–1585. Atkinson, W. O.; Link, L. A.; Nichols, B. C.; Peedin, G. F. **The Effects of Ethephon on Ripening and Certain Quality Components of Burley Tobacco.** *Tobacco International* 182(12): 88-89, June 13, 1980.

Field yellowing of Burley tobacco plants sprayed with ethephon (2-chloroethylphosphonic acid) was not consistent. Tendencies for lower yield and lower nicotine content in ethephon-treated tobacco indicate that growth and/or curing characteristics were altered. Market price and percent usable leaf were not influenced by chemical treatment. (Auth. Abs.)

80–1586. Congleton, W. F. **Effects of Transplanting Date, Variety, Nitrogen Rate and Ethephon on the Yield, Quality, Sugar and Alkaloid Concentrations and Harvest Period of Flue-Cured Tobacco.** Doctoral Dissertation, North Carolina State University, Raleigh, University Microfilms International 79-05501, 1978, 48 pp.

Six experiments were conducted in 1976 and 4 in 1977 at 10 sites in North Carolina to determine the effects of transplanting date, variety, nitrogen rate, and the chemical ripening agent, ethephon, on yield, quality index, sugar, and total alkaloid concentrations, and harvest initiation and completion of flue-cured tobacco. Tobacco transplanted 31 to 54 days later than normal had a significantly lower yield, quality index, and sugar concentration than tobacco transplanted at the normal time. Total alkaloid concentration was not affected by transplanting date. The yield and quality index of C-347 and G-28 varieties were not significantly different when transplanted at the normal date. However, C-347 produced a significantly higher yield but lower quality than G-28 when transplanted late. G-28 had a significantly lower total alkaloid concentration than C-347 in both transplantings, and a significantly lower sugar level when transplanted at the normal time. There were no varietal differences for the time to end of harvest in the normal transplanting nor the time to beginning of harvest in the late transplanting. Reducing the nitrogen rate to a level 15 lbs below the recommended rate had no effects on any of the characters measured. Increasing the nitrogen rate to a level 30 lbs above the recommended rate significantly increased the yield and decreased the sugar concentration of late-transplanted tobacco but had no effect on the quality index, total alkaloid concentration, or the time to beginning of harvest. Ethephon significantly reduced yield, increased the quality index, and reduced the harvest period of C-347 transplanted at the normal time without affecting the sugar and total alkaloid concentrations. Delaying transplanting as a means of extending the total harvest period of flue-cured tobacco and using ethephon to avoid overlaps in harvesting the two crops seems possible. If yield and quality losses associated with late transplanting of nonirrigated tobacco can be overcome by better management or with irrigation, extending the harvest period to reduce fixed production costs may become an accepted management practice. (Auth. Abs. Mod.)

80–1587. Lamarre, M. **Les Effets du pH et des Engrais sur la Composition Chimique du Tabac a Cigarette. [The Effects of pH and Fertilizer on the Chemical Composition of Cigarette Tobacco.]** *Canadian Journal of Soil Science* 59(3): 265-270, August 1979, French.

A 3-year study was made of the effect of pH and fertilizer on the chemical composition of cigarette tobacco. The experiment comprised three pH treatments ranging from pH 4.5 to 7.0, and three rates of fertilizer application (673; 1,346; and 2,018 kg/hectare of 2-12-12T) and was carried out at Lavaltrie on an Uplands series sand. The leaf samples were analyzed to determine these 12 parameters: ash, silica, nicotine, total nitrogen, reducing sugars, phosphorus, potassium, sodium, calcium, magnesium, manganese, and iron. In general, when the pH of the soil was high (6.5-7.0), the leaves contained heavier concentrations of the above-mentioned elements. Manganese and iron levels were lower, however, for plants on neutral soils than those on acid soils. The application of fertilizer raised the percentage of ash, nicotine, total nitrogen, phosphorus, potassium, magnesium, and manganese. Concentrations of the other elements declined when fertilizer was applied. (Auth. Abs.)

80–1588. Lamarre, M. **L'Influence du Stade d'Ecimage et de la Distance de Plantation sur les Caracteristiques Agronomiques et Chimiques du Tabac a Cigarette. [The Influence of Topping Stage and Planting Distance on the Agronomic and Chemical Characteristics of Cigarette Tobacco.]** *Canadian Journal of Plant Science* 59(4): 1109-1112, October 1979, French.

The influence of topping stage and planting distance on the agronomic and chemical characteristics of cigarette tobacco was studied from 1974 to 1976. The plants were spaced 41, 51, and 61 cm in the row, 122 cm between rows. The tobacco was topped at the early button stage, the button stage, and at 25 percent flowering. A planting distance of 41 cm with early topping produced the best results, taking into consideration both agronomic and chemical factors. Early topping had no significant effect on yield, grade index, maturity index, and percentage of reducing sugars. Late topping reduced revenue, percentage of nicotine, total alkaloids, and total nitrogen. Close planting in the row increased yield and revenue while it decreased the level of total alkaloids and total nitrogen, and had no significant effect on the other parameters studied. (Auth. Abs.)

80–1589. Lamprecht, M. P.; Botha, A. H.; Pretorius, C.J.H.; De Beer, M. C. **The Effect of Spacing and Topping of Plants on the Growth, Yield and Value of Low Profile Flue-Cured Tobacco.** *Agroplantae* 11(2): 29-33, 1979.

A previous study indicated that low profile flue-cured tobacco plants spaced 50 x 25 cm, topped to seven (T7) or eight (T8) leaves, produced a high yield and income of good quality tobacco. The present study was undertaken to find more convenient combinations of spacing and topping. Five spacings and four topping treatments were combined in an experiment at Rustenburg over four seasons from 1973-74. Growth, yield, and income were studied. The 75/25 x 25 row spacing (twin rows, 25 cm apart, separated by 75 cm inter-row space) combined with topping to eight leaves per plant compared very favorably in yield stability, price, and income with the recommended 50 x 25 T8 combination. Since this treatment also resulted in relatively small, early maturing (±80 days) plants, and because of its more convenient spacing, it should be considered an obvious alternative to the 50 x 25 T8 combination. (Auth. Abs.)

80–1590. Lamprecht, M. P.; Pretorius, C.J.H.; de Beer, M. C.; Shawe, F. J. **The Effect of Leaf Removal by Priming and Topping on the Growth, Yield and Quality of Flue-Cured Tobacco.** *Agroplantae* 11(3): 55-61, 1979.

Two cultivars of flue-cured tobacco (*Nicotiana tabacum* L.) were subjected to combinations of three priming and three topping treatments in 1976-77 and 1977-78. In 1977-78, plots were split to compare treated and untreated (untopped and unprimed) tobacco. There was no significant cultivar x treatment interaction. Leaf number per plant and per hectare (ha), and removal of dry matter and nitrogen, phosphorus, potassium, and chlorine were significantly affected by leaf numbers removed through priming and topping. Removal of the inflorescence at the early flowering stage was the most significant factor influencing the results of the experiment. High yield and income per ha were obtained from plants topped without leaf removal, plants of which four leaves were removed by topping, and plants from which four leaves were taken both by topping and priming. Priming had a significant positive effect on price per kg and total alkaloid concentration, whereas nitrogen and sugar concentrations were affected to a lesser extent. Likewise, an increase in the number of leaves per plant removed by topping had a positive effect on alkaloid concentration. Therefore, leaf removal by priming and topping could be a useful practice for maintaining acceptable quality levels. (Auth. Abs. Mod.)

See also, **80–1827, 80–1846**

80–1591. Lamprecht, M. P.; Shawe, F. J.; Pretorius, C.J.H.; Botha, A. H.; de Beer, M. C.; Nel, J. G. **The Effect of Spacing and Topping on Some Quality Components of Low-Profile Flue-Cured Tobacco.** *Agroplantae* 11(2): 35-39, 1979.

The effects of five combinations of row and plant spacings and four topping treatments on both physical and chemical quality components of low-profile flue-cured tobacco were studied in field trials at Rustenburg and Nelspruit in South Africa. Nitrogen and total alkaloid levels were higher and sugar levels lower at Nelspruit than at Rustenburg. At both localities the nitrogen:total alkaloids and sugar:total alkaloids ratios, protein N, and filling value increased with increased leaf number per hectare, but total alkaloids showed the opposite trend. The 50 x 50 cm and 75/25 x 50 cm spacings, i.e., plants topped lightly, were low in total alkaloids and high in sugars. Because of the inadequate alkaloid levels and the high nitrogen:total alkaloids ratio, these spacings cannot be recommended for high quality tobacco. The quality of the 50 x 25 cm, 100 x 12.5 cm, and 75/25 x 25 cm spacings (plants topped deeply), on the other hand, was acceptable. However, the 75/25 x 25 cm spaced plants topped to eight leaves/plant seemed to be the best choice, having an adequate interrow space, and a previously demonstrated satisfactory yield and value. (Auth. Abs.)

80–1592. Rosa, N. **Changes in Quality Parameters of Tobacco With Time.** *Lighter* 49(3): 10-13, 1979.

This study compares the performance of plots of untreated tobacco over a 9-year interval to observe changes in tobacco quality parameters. The agronomic parameters evaluated were yield, grade index, return per hectare, number of suckers, average dry weight per sucker, and leaf length and width. Filling value, lamina weight and leaf shatterability, and the proportion of midrib to lamina were also used to establish similarities among physical parameters. Total alkaloids and reducing sugars were the only chemical quality parameters evaluated. Account was also taken of some weather conditions (e.g., temperature, sunshine, and rainfall) which are believed to influence tobacco growth and productivity. Correlations among the various parameters over the 9-year period showed that the proportions of leaf length to width changed very little. Reducing sugars were negatively correlated with all parameters studied. Yield was positively correlated with all parameters except reducing sugars, and the highest positive association occurred with total alkaloids. Lamina weight showed the highest positive association with filling value.

80–1593. Albo, J.-P.; Dumery, B. **Estimation par Pyrolyse du Potentiel en Condensat des Tabacs. [Evaluation of Tobacco Condensate Potential Through Pyrolysis.]** *Annales du Tabac. Section 2* (14): 205-216, 1977, French.

The following simple method for evaluating tobacco condensate potential using macropyrolysis has been developed: (1) a sample of tobacco powder of uniform-sized particles weighing not more than 350 mg and occupying a constant volume is subjected to pyrolysis at 560°C in an airflow of 4 ml/sec; and (2) after pyrolysis (13 min), the increase in weight of a filtering disk (Cambridge) is measured. This method already permits a very satisfactory grading of "puros" tobaccos according to their condensate potential, but could be improved by automation of some of the operations. (Auth. Abs.)

80–1594. Bluethgen, A.; Nijhuis, H.; Hamann, J.; Heeschen, W. **Gaschromatographische Methode zur Bestimmung von Nikotin in Milch und Blut. [Gas Chromatographic Method for the Determination of Nicotine in Milk and Blood.]** *Milchwissenschaft* 33(3): 137-141, March 1978, German.

As substitutes for chlorinated hydrocarbons, insecticides of plant origin (including nicotine and pyrethrines) are of special interest in the control of ectoparasites on farm animals. It therefore seemed necessary to develop a determination method for nicotine residues in milk and blood. The milk of the treated animal is defatted, the protein is precipitated with trichloroacetic acid and, after adding caustic solution, the supernatant is treated with organic solvent. An intermediate cleanup with diluted hydrochloric acid, transferring the nicotine into the hydrophilic phase, removes the main part of co-extracted interfering substances. Afterwards the nicotine is transformed into a free base, extracted with organic solvent and determined by gas chromatography, thin-layer chromatography, and mass spectrometry. The method was tested in an experiment with four lactating cows with a registered, commercial preparation containing 0.04 percent nicotine. The results show that the application of 50 to 225 mg nicotine per animal (0.1 to 0.45 mg/kg body weight) causes residues in milk of up to 0.085 mg/l. (Auth. Abs. Mod.)

80–1595. Brooks, R. R.; Trow, J. M. **Lead and Cadmium Content of Some New Zealand and Overseas Cigarettes.** *New Zealand Journal of Science* 22(3): 289-291, 1979.

Lead and cadmium contents were determined in tobaccos and papers from 49 New Zealand and 13 overseas brands of cigarettes. The mean cadmium content of various types of New Zealand brands was 0.23-0.56 $\mu g/g$ (mass at 60 percent relative humidity). The cadmium content of cigarette papers was considerably lower than that of the tobacco. Lead levels in New Zealand brands were fairly uniform at 0.48-0.55 $\mu g/g$ for the various types of cigarettes, but were appreciably higher in the papers. Higher lead levels in papers were not considered to be of much importance because of the much lower mass of the paper compared with the tobacco. Both cadmium and lead concentrations in New Zealand brands were low and within the range of levels reported for overseas tobaccos, but it is suggested that even lower levels are desirable. (Auth. Abs.)

80–1596. Chakarvarti, S. K.; Dhiman, J.; Nagpaul, K. K. **Determination of the Uranium Content in Some Indian Cigarettes.** *Health Physics* 36(5): 638-640, May 1979.

Twenty popular brands of Indian cigarettes were collected and the tobacco from each was ground into a fine powder for analysis. It was necessary to calculate an average value for total uranium content as its distribution in the samples was not uniform, as evidenced by the large number of track clusters (sunbursts) formed in the detectors. Results showed a variation between 0.04 and 0.1 $\mu g/g$. Further studies of the uranium content of cigarette tobacco may be useful in determining whether some of the uranium content accompanies the particulate material of cigarette smoke.

80–1597. Cohen, B. S.; Eisenbud, M.; Harley, N. H. **Alpha Radioactivity in Cigarette Smoke.** *Radiation Research* 83(1): 190-196, July 1980.

The alpha (α) activity of cigarette smoke tar deposited onto membrane filters was found to be associated with the relatively insoluble fraction. Perfusion of the tar with physiological saline resulted in no change in the mean measured activity, but there was more variability in the measured values for the perfused tar than for the initial tar samples. Analysis of cigarette smoke condensate showed that radium and thorium were present, but over 99 percent of the α activity resulted from polonium-210 (^{210}Po). Repeat measurements after a time lapse of 2.5 years indicate that the initial lead-210 content of the tar is roughly 30-40 percent of the original ^{210}Po content for both unprocessed and perfused samples. An increase in the α activity concentration of smoke deposited in lung tissue may result from the lack of solubility of the radioactive material compared with other smoke constituents. (Auth. Abs.)

80–1598. Elmenhorst, H. **Sauren und Phenole in der "Semi-Volatile"-Fraktion des Rauches von Blend-, Virginia-, Burley- und Orient-Cigaretten. [Acids and Phenols in the Semi-Volatile Fraction of Smoke From Blend, Virginia, Burley and**

Oriental Cigarettes.] *Beitraege zur Tabakforschung* 6(4): 182-188, August 1972, German.

The purpose of this work was to compare the composition of the semivolatile fraction of the smoke of different tobacco varieties. A German blend cigarette without any additive and three cigarettes made from Virginia, Burley, and Oriental tobaccos were compared. The composition of the acid fraction of the "semivolatile" cigarette smoke was analyzed. Twenty-two substances including the newly identified octadeca-9,12,15-trien-1-ol were identified by gas chromatography (GC) and mass spectrometry and quantitatively determined by GC-methods. Although the smoke samples were qualitatively similar in volatile acid and phenol composition, there were, quantitatively, remarkable differences between the different tobacco types. In the semivolatile fraction of the smoke of the Oriental cigarette, the level of 3-methylvaleric acid and isovaleric acid was highly increased. The semivolatile fraction of the smoke of the Virginia cigarette showed an overall increased level of the rest of the aliphatic acids, especially palmitic acid. Phenol, cresol, catechol, guajacol, and benzoic acid were also increased compared with Burley and Oriental cigarettes. In the semivolatile fraction of Burley smoke all the analyzed substances were diminished. (Auth. Abs.)

80–1599. Joigny, C. **Report on the Collaborative Experiment on the Determination of Carbon Monoxide and Nitric Oxide.** *CORESTA Information Bulletin* (3/4): 67-76, 1978.

In 1978, 18 laboratories participated in three experiments to determine carbon monoxide (CO) and nitric oxide (NO) yields of the same type of cigarette (85 mm cigarette made of dark tobacco with an acetate filter). The first two experiments determined the influence of conditioning room humidity and the puff duration and volume on CO yield. The third experiment compared NO yields by laboratories using their own method. Results indicated that an increase in relative humidity from 60-65 percent resulted in an increase of 2.7 percent in CO yield per cigarette. A change in puff duration from 2 to 2.5 sec increased CO yield per cigarette by 0.05 mg. An increase in puff volume from 30 to 40 ml resulted in negligible error. Since all participating laboratories used chemiluminescence for NO determination, no comparison between techniques could be made. However, a considerable difference (17 percent) in reported values could not be explained.

80–1600. Kaul, K.; Lester, R. L. **Isolation of Six Novel Phosphoinositol-Containing Sphingolipids From Tobacco Leaves.** *Biochemistry* 17(17): 3569-3575, August 22, 1978.

A concentrate of phosphosphingolipids from tobacco leaves has been resolved into two groups by chromatography on diethylaminoethylcellulose. The first group eluted contains no acetyl residues, whereas the second group contains one *N*-acetyl/mol of phosphorus. Three lipids from each group have been purified by chromatography on Porasil columns. The chemical composition of these six novel lipids shows them to be related to the previously characterized phosphosphingolipids PSL-I, *N*-acetylglucosamine (glucuronic acid, inositol phosphate) ceramide and PSL-II, glucosamine (glucuronic acid, inositol phosphate) ceramide. The novel lipids are: PSL-IA, PSL-I-($\mathrm{Ara_2Gal_2}$); PSL-IB, PSL-I-($\mathrm{Ara_3Gal_2}$); PSL-IC, PSL-I-($\mathrm{Ara_4Gal_2}$); PSL-IIA, PSL-II-($\mathrm{Ara_3Gal}$); PSL-IIB, PSL-II-($\mathrm{Ara_{2\ or\ 3}Gal_2}$); PSL-IIC, PSL-II-($\mathrm{Ara_2Gal_2Man}$). The unusual finding of unacetylated hexosamine in the PSL-II group of lipids appears not to be an artifact, as suggested by experiments involving rapid harvesting and processing of the tobacco leaves. Some of the fractions obtained by chromatography on diethylaminoethylcellulose appear to contain phosphosphingolipids with 16 or more sugar residues. (Auth. Abs.)

80–1601. Miyano, M.; Matsushita, H.; Yasumatsu, N.; Nishida, K. **New Minor Alkaloids in Burley Tobacco (*Nicotiana tabacum*).** *Agricultural and Biological Chemistry* 43(7): 1607-1608, 1979.

Two new minor alkaloids were identified in aged Burley tobacco leaves: *N'*-formylanabasine and *N'*-formylanatabine. Their content in tobacco leaves was determined by quantitative gas liquid chromatography and was found to be 0.0002 percent and 0.0001 percent respectively.

80–1602. Neri, M.; Carugno, N.; Rossi, S. **Nouvelle Methode Automatique Pour la Mesure de la Reaction Entre les Composes de Fumee de Cigarette. [New Automatic Method for Measuring the Reaction Between Cigarette Smoke Compounds.]** *Annales du Tabac. Section 1* (16): 89-95, 1978, French.

The importance of thiols as protective agents against ionizing radiation and oxidizing and alkylating substances is well known. Since these groups are present in cigarette smoke, the reduction of thiols is considered, by some researchers, as an index of smoke quality. For this reason, the reactivity of thiols with cigarette smoke was the subject of this investigation. This paper describes a new analytical method to determine the products of these reactions. The reactions of certain compounds such as nitric oxides, aldehydes, ketones, and free radicals with thiols is also described. It was found that the reactions are affected by pH. The preferred pH is 7 because it matches the pH of the smoker's body fluids. This newly developed analytical method facilitated the determination of the amounts of products and reactants. It was found that certain thiols react to the extent of 95 percent and that major reaction products are cystine (5 percent) and thiazolidine (35 percent). (Auth. Abs. Mod.)

80–1603. Rickert, W. S.; Robinson, J. C.; Young, J. C. **Estimating the Hazards of "Less Hazardous" Cigarettes. I. Tar, Nicotine, Carbon Monoxide, Acrolein, Hydrogen Cyanide, and Total Aldehyde Deliveries of Canadian Cigarettes.** *Journal* *of Toxicology and Environmental Health* 6(2): 351-365, March 1980.

The tar, nicotine, carbon monoxide (CO), hydrogen cyanide, total aldehyde, and acrolein deliveries of 102 brands of Canadian cigarettes have been determined. On the basis of these deliveries, 15 brands (9 percent of sales) were categorized as low hazard, and 9 brands (10 percent of sales) as high hazard. All six constituents were used for the classification as tar was a poor indicator of gas phase deliveries for most brands. As a result, smokers who indiscriminately switch to a brand with a lower tar delivery may inadvertently increase their exposure to gas phase constituents such as CO and increase their risk of smoking-related pathology. With respect to Canadian cigarettes, the choice of a 30-mm standard butt length may result in omission of some of the available tobacco from the testing procedure, reducing the value of tar level as a guide to choosing a less harmful cigarette. (Auth. Abs.)

80–1604. Sekin, S. **Enzymatic Determination of Glucose, Fructose, and Sucrose in Tobacco.** *Tobacco International* 181: 27-29, July 13, 1979.

A modification of the Boehringer enzymatic method for a rapid and accurate micro determination of glucose, fructose, and sucrose in tobacco leaves is described. This method of analysis needs no sophisticated laboratory equipment except a photometer, and it enables one to work with very small sample quantities. For determination of the accuracy of the method, known amounts of glucose, fructose, and sucrose were added to tobacco samples which were known to differ in sugar content before extraction and analyses. Results indicate that the recoveries were satisfactory. The average recovery is 100 percent for glucose, 101 percent for fructose, and 101 percent for sucrose with standard deviations of ±0.3609, ±0.1990, and ±0.2377, respectively. (Auth. Abs. Mod.)

80–1605. Takagi, Y.; Fujimori, T.; Kaneko, H.; Kato, K. **Phytuberol, From Japanese Domestic Tobacco, *Nicotiana tabacum* cv. Sulfu.** *Agricultural and Biological Chemistry* 43(11): 2395-2396, 1979.

The isolation and identification of phytuberol and its derivatives from Suifu tobacco are reported. A methanol eluate, designated as fraction 11, from a silicic acid column chromatography of neutral volatiles of Suifu tobacco, was rechromatographed using preparative gas liquid chromatography. Mass spectral, infrared, and nuclear magnetic resonance analyses identified a constituent compound as phytuberol.

80–1606. Thomas, C. E. **Automated Method for FTC Tar of Low Delivery Cigarettes Based Upon Fluorescence of TPM Extracts.** *Tobacco International* 182(11): 67-71, May 30, 1980.

An automated method for the determination of Federal Trade Commission (FTC) tar of low delivery cigarette brands using 2-propanol extracts of total particulate matter (TPM) pads has been developed. The method is based on the measurement of the fluorescence emission intensity of the TPM extracts at 520 nm when excited at 475 nm. A linear correlation was shown between fluorescence and FTC tar. The detection limit of the method is approximately 0.5μg tar/ml of extract. The average relative standard deviation is 14 percent at the 2-s level. The automation consists of an AutoAnalyzer I system and a UNC microcomputer for data acquisition. (Auth. Abs.)

80–1607. Uchimiya, H.; Chen, K.; Wildman, S. G. **A Micro Electrofocusing Method for Determining the Large and Small Subunit Polypeptide Composition of Fraction 1 Proteins.** *Plant Science Letters* 14(4): 387-394, 1979.

A method is described whereby Fraction 1 protein contained in 300-500 mg of leaves is precipitated by antiserum, the antibody-protein complex carboxymethylated and subjected to isoelectric focusing in 8 M urea. The number of resolved polypeptides composing the large and small subunits agrees with previous determinations made with crystalline Fraction 1 proteins obtained from *Nicotiana excelsior, N. glauca, N. tabacum, and N. glutinosa* as well as noncrystalline proteins purified from other genera of plants. (Auth. Abs.)

80–1608. Walker, E. K.; Zilkey, B. F. **Effect of Cigarette Pressure Drop and Weight and Tobacco Stalk Position on Smoke Tar and Nicotine Yields.** *Tobacco International* 181(16): 30-33, August 10, 1979.

A number of bulk lots (grades) of flue-cured tobacco from different stalk positions were made into cigarettes and selected within relatively narrow ranges of weight and pressure drop to provide a large number of subsamples for smoking. A large proportion of the subsamples within each grade provided comparisons of the effects of variable pressure drop at constant weight or of variable weight at constant pressure drop on smoke parameters. Based on such comparisons, appropriate adjustments were tabulated for each smoke parameter in order to minimize the influence of variable cigarette weight and pressure drop. Increasing weight and pressure drop affected both tar and nicotine levels; nicotine was affected to a greater extent than tar. Adjusted values for the smoke parameters were less divergent from the means than the original values, except for tar and nicotine per cigarette where adjusted and unadjusted values were similar. All parameters of the smoke increased with ascending stalk position, but nicotine increased proportionately more than tar. (Auth. Abs.)

80–1609. Abel, E. L.; Dintcheff, B. A.; Day, N. **Effects of In Utero Exposure to Alcohol, Nicotine, and Alcohol Plus Nicotine, on Growth and Development in Rats.** *Neurobehavioral Toxicology* 1(2): 153-159, Summer 1979.

Compared with exposure of pregnant Long Evans rats to alcohol (5 g/kg/day) alone, the combination of alcohol plus nicotine (1.5 mg/kg/day, subcutaneously) reduced maternal food intake and liver/body weight ratio in female offspring at 12 weeks, but did not affect fecundity, length of gestation, maternal weight gain, mortality, litter size or pup weight at birth, postnatal mortality, or weight at 11 weeks to a significantly greater extent. Relative to pair-fed controls, alcohol and the combination of alcohol plus nicotine reduced fecundity, increased gestation length, and reduced pup weight at birth and at 11 weeks (male offspring only). Relative to pair-fed controls, alcohol, but not the combination of alcohol plus nicotine, reduced litter size at birth and body weight at 11 weeks (among female offspring), increased the liver and spleen body weight ratios, and decreased the heart/body weight ratio at 12 weeks. Relative to pair-fed controls, alcohol plus nicotine, but not alcohol alone, increased mortality at birth and increased the adrenal/body weight ratio (among male offspring) at 12 weeks of age. Relative to pair-fed controls, female nicotine-treated offspring had higher lung/body weight ratios at 12 weeks of age. (Auth. Abs.)

80–1610. Ashton, H.; Marsh, V. R.; Millman, J. E.; Rawlins, M. D.; Telford, R.; Thompson, J. W. **The Use of Event-Related Slow Potentials of the Brain as a Means to Analyse the Effects of Cigarette Smoking and Nicotine in Humans.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 54-68.

The effect of some centrally active drugs (e.g., caffeine, nitrazepam, diazepam) and of cigarette smoking on the electroencephalographic phenomenon known as the contingent negative variation (CNV) is discussed in this chapter. A prediction that smoking has a dual effect on the CNV: stimulant effects of nicotine increasing CNV magnitude and depressant effects decreasing it, was tested and confirmed in 22 subjects. The conclusion that CNV effects were due to a central action by nicotine was tested in the following experiments: (1) the relationship between personality, nicotine (in cigarette smoke) intake, and CNV magnitude; (2) the effects of nicotine administered intravenously on the CNV; and (3) the dose—response relationship of nicotine and the CNV. The various data indicated that personality factors (e.g., extraversion or introversion) appeared to interact with nicotine dose-dependence. More extraverted subjects took a smaller dose of nicotine and showed a mean increase in CNV magnitude, whereas the converse was found for introverts. In the second experiment, it was determined that intravenous nicotine and cigarette smoking had similar effects on the CNV. Data from the third experiment indicated that nicotine caused biphasic, dose-related CNV effects.

80–1611. Bartelik, S.; Mycinska, M. **Poziom Immunoglobulin w Slinie i Surowicy Nalogowych Palaczy Papierosow. [Immunoglobulin Levels in the Serum and Saliva of Habitual Cigarette Smokers.]** *Wiadomosci Lekarskie* 32(15): 1059-1060, 1979, Polish.

The effect of cigarette smoking on the immunoglobulin levels in the serum and mixed saliva was investigated. It was found that in smokers the IgG level was markedly lowered (mean serum level (mg/dl) for smokers was 3.39 as compared to 7.46 for nonsmokers, $p = 0.058$; corresponding mean saliva levels (mg/dl) were 1.238 and 1.650, $p = 0.004$). The levels of IgA and IgM were also low but without statistical significance. (Auth. Abs. Mod.)

80–1612. Brandange, S.; Lindblom, L. **The Enzyme "Aldehyde Oxidase" is an Iminium Oxidase. Reaction With Nicotine $\Delta^{1'(5')}$Iminium Ion.** *Biochemical and Biophysical Research Communications* 91(3): 991-996, December 14, 1979.

The second of the two reaction steps involved in the metabolic transformation of (-)-nicotine to (-)-cotinine (i.e., the oxidation of the intermediate is mediated mainly, if not solely, by the enzyme aldehyde oxidase. Of the molecular species that constitute the intermediate, nicotine $\Delta^{1'(5')}$ iminium ion appears to serve the substrate. The enzyme has a strong affinity for the iminium ion as shown in a study on the inhibition of the oxidation of 3-(aminocarbonyl)-1-methylpyridinium chloride. This study gave a value of $K_i = 6\mu M$; $K_m = 2\mu M$ (pH 7.4). Mainly in view of this finding, iminium oxidase seems to be a more adequate name than aldehyde oxidase for this enzyme. (Auth. Abs.)

80–1613. Castro, A.; Monji, N.; Ali, H.; Yi, J. M.; Bowman, E. R.; McKennis, H., Jr. **Nicotine Antibodies: Comparison of Ligand Specificities of Antibodies Produced Against Two Nicotine Conjugates.** *European Journal of Biochemistry* 104(2): 331-340, March 3, 1980.

Conjugates between bovine serum albumin and (*R,S*)-2-aminonicotine were produced, and these conjugates were employed in rabbits and goats for the production of nicotine antibodies. In the assay of nicotine, an ^{125}I-tyrosine methyl ester derivative of (*R,S*)-6-aminonicotine was employed as radioligand. The antibody-bound derivative was separated from the free derivative by charcoal adsorption (0.5 percent charcoal, 0.1 percent dextran T-70, 0.1 percent bovine serum albumin, pH 7.3). Among the 25 nicotine derivatives and metabolites examined, (*R,S*)-6-aminonicotine gave the highest cross-reaction. Cross-reaction with cotinine, a major mammalian metabolite of nicotine, was less than 0.1 percent for both the rabbit-derived and the goat-derived antisera. Cross-reaction by other metabolites, such as (*S*)-nicotine-*N*′-oxide, (*S*)-nornicotine, and *N*-methylpyrrolidine was less than 1 percent. The antibodies produced were thus highly specific to nicotine. The radioimmunoassay for nicotine showed a maximum sensitivity of 10 ng/ml in 50-μl plasma samples for both antisera. After the smoking of a single cigarette (1.2 mg nicotine content in mainstream), the peak blood plasma level of nicotine in the subjects varied from 20 to 104 ng/ml, and high levels of nicotine were not necessarily found in heavy smokers. (Auth. Abs.)

80–1614. Claude, J. R.; Lellouch, J.; Thevenin, M. **Air Pollution and Carboxyhaemoglobin in Man.** In: Benarie, M. M. (Editor). *Studies in Environmental Science.* Volume I. Amsterdam, Elsevier Scientific Publishing Company, 1978, pp. 113-115.

Smoking habits and exposure to air pollution by car exhausts were investigated in 1,418 traffic policemen in relation to morning carboxyhemoglobin (COHb) levels. Results indicate that COHb is raised in smokers and remains higher than in nonsmokers even after 8 to 10 hours of tobacco abstinence, both in inhaling as well as in noninhaling smokers. The exposure of traffic policemen to air carbon monoxide does not increase the COHb level in nonsmokers, but has a minor effect in smokers. (Auth. Abs. Mod.)

80–1615. Cooksley, W.G.E.; Farrell, G. C.; Cash, G. A.; Powell, L. W. **The Interaction of Cigarette Smoking and Chronic Drug Ingestion on Human Drug Metabolism.** *Clinical and Experimental Pharmacology and Physiology* 6(5): 527-533, September-October 1979.

Drug metabolism was assessed in 20 subjects by the disappearance rate of antipyrine as measured in saliva. Results were expressed in terms of both clearance and half-life. Basal measurements of the antipyrine disappearance rate were performed in seven nonsmokers and six smokers. After administration of glutethimide, 250 mg daily for 7 days, a second measurement of the antipyrine disappearance rate was made. Glutethimide enhanced antipyrine metabolism in both smokers and nonsmokers. The degree of change in both groups was similar. A similar study was carried out with a smaller dose (125 mg) of glutethimide. It revealed no significant difference between smokers and nonsmokers. The data are consistent with smoking and chronic drug ingestion having additive effects on enhancement of hepatic drug metabolism. (Auth. Abs.)

80–1616. Crossen, P. E.; Morgan, W. F. **Sister Chromatid Exchange in Cigarette Smokers.** *Human Genetics* 53(3): 425-426, March 1980.

The incidence of sister chromatid exchanges (SCE) in peripheral blood lymphocyte chromosomes in smokers and nonsmokers was investigated in 120 subjects. There was no difference in the SCE rate between smokers and nonsmokers, nor was there any difference between heavy (>10 cigarettes/day) and light (<10 cigarettes/day) smokers. (Auth. Abs.)

80–1617. Cusack, B.; Kelly, J. G.; Lavan, J.; Noel, J.; O'Malley, K. **The Effect of Age and Smoking on Theophylline Kinetics.** *British Journal of Clinical Pharmacology* 8(4): 384P-385P, 1979.

The effect of smoking and age on theophylline metabolism was studied in five young (age 21-30 years) and six elderly (age 67-86 years) nonsmokers and eight young (age 20-33 years) and six elderly (age 67-79 years) cigarette smokers. Nonsmokers received theophylline 125 mg orally and intravenously on separate occasions while smokers were given 125 mg theophylline intravenously. The rate of metabolism and extent of absorption for nonsmokers were similar in both age groups as were values for volume of distribution, elimination half-life, and plasma clearance. The comparison between nonsmokers and smokers in respective age groups shows that clearance was significantly greater ($p<0.02$) and half-life significantly shorter ($p<0.01$) in young smokers. Although half-life values were significantly shorter in elderly smokers ($p<0.001$), the increase in clearance did not reach significance because of the scatter of values in nonsmokers.

80–1618. Davies, P.; Sornberger, G. C.; Huber, G. L. **Microperoxisomes in the Rat Alveolar Macrophage: A Cytochemical and Morphometric Study of the Effects of Tobacco Smoke Inhalation.** *Journal of the Reticuloendothelial Society* 26(6): 815-825, December 1979.

The effects of experimental tobacco smoke inhalation on the morphology of microperoxisomes (MP) in alveolar macrophages (AM) were examined. Alveolar macrophages were obtained by bronchopulmonary lavage of rats exposed for 30 consecutive days to tobacco smoke. Microperoxisomes were identified within the cells by the cytochemical localization of catalase. In AM from nonexposed, control animals MP profiles were situated close to cisternae of the smooth endoplasmic reticulum, while in AM from exposed animals MP were often close to, or surrounded by, smooth-membraned tubules containing a central, osmiophilic band. Morphometric techniques applied to cell profiles in randomly sampled electron micrographs demonstrated a decrease in the numerical density of MP but an increase in volume density and in their mean volume. Size frequency curves confirmed a shift in the exposed group to a population of larger MP, but shape (eccentricity) frequency curves were similar in control and exposed groups. The significance of these results in terms of the reported functions of peroxisomal granules is discussed. (Auth. Abs.)

80–1619. Denis, P.; Pasquis, P.; Duval, C.; Lefrancois, R. **P_{50} Values of Non Smoker and Smoker Pregnant Women.** *Biomedicine* 33(2): 49-51, April 1980.

The influence of tobacco smoke during pregnancy on hemoglobin concentration and oxygen affinity in smoking women has been studied. Fifty-nine nonsmokers and 45 smokers were investigated during pregnancy and early puerperium. Results have shown (1) a slight nonsignificant increase in oxygen affinity in smokers, (2) a significant decrease in oxygen affinity in smokers at term and early puerperium, the same as in nonsmokers, and (3) a significant increase in hemoglobin concentration in smokers at term as compared to nonsmokers ($p<0.001$). The small modifications of oxygen affinity in smokers and the increase in hemoglobin concentration at term suggest that the oxygen delivery from the maternal to the fetal blood are probably unaffected by these factors. (Auth. Abs.)

80–1620. Garrett, R.J.B.; Jackson, M. A.; Filio, A. K.; Garrett, N. E. **Effect of Cigarette Smoke on Drug Metabolism *In Vitro.*** *Life Sciences* 25(9): 755-758, August 27, 1979.

N-demethylation of aminopyrine and C-hydroxylation of aniline by hepatic microsomal enzymes prepared from rat livers were measured during in vitro exposure to cigarette smoke. Metabolism of aminopyrine during smoke exposure was not significantly altered. Metabolism of aniline during smoke exposure was inhibited 70-80 percent ($p<0.001$) indicating that an initial effect of exposure to cigarette smoke is a decreased rate of biotransformation via C-hydroxylation. This finding, coupled with the findings of other investigators who have shown that the delayed effect of exposure to cigarette smoke is induction of hydroxylase activity, suggests that cigarette smoke produces a biphasic alteration in certain hepatic biotransformation processes. (Auth. Abs.)

80–1621. Gilli, G.; Corrao, G.; Scursatone, E. **Valori Relativi Alla Carbossiemoglobina in un Gruppo di Popolazione. Analisi di Alcune Variabili. [Carboxyhemoglobin Values in a Group of People. Analysis of Certain Variables.]** *LAB. Giornale di Ricerca e Diagnostica di Laboratorio* 6(4): 389-396, 1979, Italian.

The purpose of this work is to evaluate carboxyhemoglobin (COHb) values in a sample of people in connection with chronic subliminal exposure to carbon monoxide (CO) (collective and individual pollution). A group of 377 people was chosen and divided into many subgroups according to the exposure source to CO. The following variables were considered: cigarette smoking and exposure to urban air pollution. Analysis of the results has shown that cigarette smoke affects COHb levels much more than atmospheric pollution; in fact, significant differences among nonsmokers divided by residence (city and country) were not found. (Auth. Abs. Mod.)

80–1622. Gilli, G.; Scursatone, E.; Vanini, G. C.; Vercellotti, E. **La Misura del Tiocianato nel Siero Come Elemento Discriminante Nella Valutazione dell'Esposizione a Ossido di Carbonio. [Serum Thiocyanate Values as a Discriminating Element in Assessment of Exposure to Carbon Monoxide.]** *Minerva Medica* 70(55): 3803-3807, December 8, 1979, Italian.

Blood carbon monoxide (CO) concentration levels in two groups of subjects with (i.e., mechanics) or without occupational CO exposure are examined in light of the subjects' declared smoking habits (in the exposed group, there were 10 smokers and 23 nonsmokers; in the nonexposed group, there were 31 smokers and 50 nonsmokers) and serum thiocyanate levels. The latter parameter proved an effective indicator of an individual's exposure to CO due to tobacco smoking. Assessment of the results showed that serum thiocyanate levels could be proposed as a discriminating test of habitual exposure to CO, so as to enable expired CO and carboxyhemoglobin values to be interpreted in occupationally exposed subjects. (Auth. Abs. Mod.)

80–1623. Gilli, G.; Silva, C.; Scursatone, E. **Esposizione all'Ossido di Carbonio: Determinazione Della Carbossiemoglobina e del Tiocianato nel Siero Quale Misura per la Esposizione Ambientale o a Fumo di Sigarette. [Exposure to Carbon Monoxide: Determination of Carboxyhemoglobin and Thiocyanate in the Serum as a Measure of the Environmental and Cigarette Smoke Exposure.]** *LAB. Giornale di Ricerca e Diagnostica di Laboratorio* 6(4): 361-368, 1979, Italian.

The purpose of this work is to evaluate the carboxyhemoglobin (COHb) and thiocyanate (SCN) concentrations in the serum to test the influence of carbon monoxide (CO) in both the environment and cigarette smoke on these two biochemical measurements. During this study, the blood of 350 people was divided into classes on the basis of residence and smoking habits. Smokers were also subdivided by average daily cigarette consumption. Subjects included 191 city and 159 rural residents, and 173 smokers (48 smoked ≤ 10 cigarettes/day, 93 smoked ≤ 20 cigarettes/day, and 32 smoked >20 cigarettes/day), of whom 100 were city and 73 were country residents. The results have shown that the percentage of saturation of CO is closely dependent on the amount of inhaled smoke and therefore on the number of smoked cigarettes. The environmental pollution (CO in the atmosphere) doesn't significantly affect COHb concentration; the finding that the value of COHb is the same in nonsmokers divided by residence (e.g., city, country) supports this. Cigarette smoke increases the SCN concentration in the serum. By using these two tests together one can get more precise information than by using a single parameter. (Auth. Abs. Mod.)

80-1624. Goldbourt, U.; Medalie, J. H.; Herman, J. B.; Neufeld, H. N. **Serum Uric Acid: Correlation With Biochemical, Anthropometric, Clinical and Behavioral Parameters in 10,000 Israeli Men.** *Journal of Chronic Diseases* 33(7): 435-443, 1980.

Serum uric acid (SUA) was measured in a group of 10,000 middle-aged male civil servants and municipal employees (of whom 5,138 were smokers and 1,652 ex-smokers) in Israel. Serum uric acid levels did not vary appreciably by age or country of birth. There were small significant correlations between SUA and weight, systolic blood pressure, diastolic blood pressure, and serum cholesterol. Serum uric acid was elevated in men with coronary heart disease and rose with increased level of education. It was negatively correlated with high-density lipoprotein cholesterol and total fat intake, and was significantly reduced among smokers of more than 10 cigarettes/day (compared to light smokers, nonsmokers, and ex-smokers) and in men with diabetes mellitus or peptic ulcer. Multiple regression analysis revealed that 11 variables were significantly related to SUA, together accounting for about 16 percent of SUA variance. The Quetelet index (weight/height^2) made most of the contribution. Among the other variables, only blood pressure and the presence of diabetes mellitus accounted for more than 1 percent of the variance. (Auth. Abs. Mod.)

80-1625. Guillerm, R.; Radziszewski, E.; Caille, J. E. **Effects of Carbon Monoxide on Performance in a Vigilance Task (Automobile Driving).** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 148-155.

The purpose of this study was to determine at which level in the blood carbon monoxide is capable of affecting vigilance and brain electrogenesis. The vigilance test involved nocturnal, long-duration (5 hours) driving of an automatic car on a circuit. The driving precision, visual response time (VRT), electrocardiogram (ECG), and electroencephalogram (EEG) of eight nonsmoking subjects were measured. Results showed that carboxyhemoglobin levels of 7 and 11 percent caused no significant changes in driving precision, VRT, EEG, or heart rate. Under all experimental conditions, however, a gradual deterioration of operational behavior and of cortical activity was observed, denoting a gradual building up of tiredness.

80-1626. Huber, G. L.; Pochay, V. E.; Shea, J. W.; Hinds, W. C.; Weker, R. R.; First, M. W.; Sornberger, G. C. **An Experimental Animal Model for Quantifying the Biologic Effects of Marijuana on the Defense System of the Lung.** *Advances in the Biosciences* 22/23: 301-328, July 22-23, 1978.

An animal model was developed as an inhalation bioassay for acute and chronic delivery of tobacco and marihuana smoke in physiologic doses to the lungs of experimental animals. Although generating identical aerosols, the pyrolyzation characteristics of marihuana and tobacco differed considerably. Both products impaired host intrapulmonary antibacterial defenses, with marihuana appearing in comparable dosages to be significantly more toxic to the alveolar bactericidal activity of the lung than tobacco smoke. The effect of both smoking products on mucociliary clearance by the airways, in this model system, appeared to be negligible. Direct biologic comparisons between the effects of the two smoking products must be guarded, however, as dosimetry cannot be precisely equated because of their opposing physiologic effects on the lung. (Auth. Abs.)

80-1627. Hussain, M. Z.; Lee, S. D.; Bhatnagar, R. S. **Increased Aryl Hydrocarbon Hydroxylase and Prolyl Hydroxylase Activities in Lung Organ Cultures Exposed to Benzo(a)pyrene.** *Toxicology* 12(3): 267-271, March-April 1979.

Exposure of neonatal rat lungs in organ culture to 10-25 μM benzo(*a*)pyrene (BaP) elevated the activities of aryl hydrocarbon hydroxylase (AHH) and prolyl hydroxylase (PH). Pyrene, a noncarcinogenic hydrocarbon did not elicit this response. Prolyl hydroxylase is an indicator of collagen synthesis, and increased PH activity in the lungs reflects increased collagen synthesis. These studies suggest that the earliest events in BaP-induced lung injury may include altered collagen metabolism. (Auth. Abs.)

80-1628. Izard, C.; Valadaud-Barrieu, D.; Fayeulle, J.-P.; Testa, A. **Influence des Parametres de Fumage sur l'Activite Genotoxique de la Phase Gazeuse de Fumee de Cigarette, Mesuree sur le Lymphocyte Humain et la Levure. [Effect of Machine-Smoking Parameters on the Genetoxic Activity of Gas Phase Cigarette Smoke Toward Human Lymphocytes and Yeast.]** *Mutation Research* 77(4): 341-344, April 1980, French.

Toxicity of the gas phase of cigarette smoke, the number of chromatid exchanges induced in human lymphocytes by cigarette smoke, and the rates of mitotic recombinants and respiratory mutants induced in yeast were determined using a smoking machine adjusted either to international standards or to the new experimental conditions. The new conditions were set up based on observations of the smoking habits of some black tobacco smokers. Accordingly, the international standards A for puff volume (35 ml), puff duration (2 sec), and puff frequency (58 sec) were modified to 12 ml, 1.5 sec, and 34.5 sec, respectively (standard B). Chromatographic analysis of the major organic components of the gaseous smoke phase showed a 63-82 percent decrease in the components' content, when standard B was used instead of A. Simultaneously, tar and nicotine contents decreased by 55 and 65 percent, respectively. The bioassays showed that the number of sister chromatid exchanges per lymphocyte decreased from 21.38 with standard A to 10.47 with standard B. Also, the rate of mitotic recombinations induced by the equivalent of 1/2 or 1 cigarette/ml of yeast culture decreased significantly ($p<0.001$) from 58.4 to 18.0 or from 82.7 to 43.4/10^4 survivors, when standard B is substituted for A. The rate of respiratory mutations induced in yeast culture under the same conditions decreased significantly ($p<0.01$) from 4.2 to 0.6 or from 13.9 to 4.3/100 survivors.

80–1629. Janzon, L.; Trell, E.; Larme, P.; Lindell, S.-E.; Lindgarde, F.; Berntorp, K.; Norden, G.; Hood, B. **Smoking and Oral Glucose Tolerance Tests.** *IRCS Journal of Medical Science* 7(10): 514, October 1979.

The effect of smoking on glucose metabolism was studied in an ongoing population screening in Malmo, Sweden. After overnight fasting and smoking abstinence, male smokers were asked to ingest a glucose solution within 5 min. Subsequent blood glucose levels were compared with those of nonsmokers. Fingertip capillary blood glucose was determined by a standard glucose oxidase method. The fasting blood glucose level was identical in heavy smokers (>20 g/day) and nonsmokers matched for body weight, sex, and age. The area of the glucose curve was larger in smokers than in nonsmokers; however, the 120 min blood glucose value was 10 percent lower ($p<0.005$) in smokers than in nonsmokers matched for age. These conflicting results reflect either the chronic effect of smoking on glucose metabolism or the effect of an overnight tobacco abstinence.

80–1630. Jaubert, F.; Barbey, S.; Grune, M.; Danel, C.; Chretien, J.; Nezelof, C. **Human Alveolar Macrophages Spontaneous Reduction of BSPT Salt.** *Histochemistry* 64(2): 163-169, 1979.

Alveolar macrophages of nonsmoking and smoking human adults reduce 2-(2′-benzothiazolyl)-5-styryl-3-(4′-phthalhydrazidyl) tetrazolium chloride (BSPT) salt spontaneously. The staining obtained is located on three cell membrane systems: the endoplasmic reticulum, the Golgi apparatus, and the nuclear envelope. Methylene blue inhibits BSPT reduction. The smokers' alveolar macrophages have less positivity than those of the nonsmokers. The endogenous cell substrate revealed in this work is the initial common pathway of two different oxidative chains bound to the microsome. One acts with cytochrome P-450 for chemical detoxification by hydroxylation, the other acts with cytochrome B_5 for lipid oxidation or peroxidation, and both may be connected with the cell bactericidal system. (Auth. Abs.)

80–1631. Jedrychowski, W.; Adamczyk, B.; Jaskolka, D. **IgG and IgA in Saliva and Blood Serum in Relation to Smoking and Respiratory Symptoms.** *Revue Francaise des Maladies Respiratoires* 7(5): 471-474, 1979.

The main purpose of the paper was to find a possible relationship between smoking and immunological disorders, which are to be the common ground for chronic respiratory diseases. Measurements of IgG and IgA in saliva and blood serum were performed in 87 persons (61 smokers and 26 nonsmokers) during medical checkup before admission to work in a sulphur mine. Persons with cough and/or sputum production were defined as having positive symptoms. From the study, different immunoglobulin patterns have emerged in saliva and serum in relation to smoking. IgG content in saliva of smokers was lower and IgA was higher than in nonsmokers. Levels of both immunoglobulins in serum were lower among smokers. In persons with respiratory symptoms, IgG content in saliva was markedly lower, especially in smokers. (Auth. Abs. Mod.)

80–1632. Knott, V. J. **Smoking, EEG and Input Regulation in Smokers and Non-Smokers.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 115-130.

Two studies which attempted to investigate whether smokers and nonsmokers have different electroencephalogram (EEG) characteristics and related studies are briefly reviewed. The first study centered on tonic (basal) resting levels of EEG activity, with specific reference to alpha activity between 8 and 12 Hz. Nonsmokers (NS) were compared with both tobacco-deprived smokers (DS) who were required to abstain from smoking for a period of 12 to 15 hours, and nondeprived smokers (NDS) who were required to smoke in their normal manner up until the experimental session. In order to examine the immediate effects of smoking on EEG alpha activity, a random sample of the DS and NDS groups was required to smoke two cigarettes (each cigarette 100 mm long, yielding 1.6 mg nicotine and 21 mg tar) within a 10-min period, inhaling at 30-sec intervals. For control, the remaining DS, NDS, and NS subjects were required to inhale air through an unlit cigarette for the same duration and in the same manner. Analysis of presmoking activity indicated significant differences between DS and both NDS and NS groups in that DS exhibited slower dominant alpha frequencies. No significant differences in EEG activity were observed between NDS and NS groups. The immediate effect of smoking two cigarettes relative to sham smoking was to produce a significant increase in dominant alpha frequency in the DS group. There was no significant time effect. The second study centered on phasic (stimulus-induced changes) EEG activity in response to visual stimulation, with specific reference to latency and amplitude components of the averaged evoked response. Based on the findings that tobacco tended to normalize cortical activity, this investigation examined the hypothesis that DS would exhibit differential responsivity than NS and that smoking, as exhibited in the NDS group, would show evidence of response normalization. No significant differences were observed between NS and NDS. Analysis of mean peak latency values indicated that DS showed faster latencies than both NS and NDS. The chapter concludes with a discussion of models for EEG effects of smoking behavior.

80–1633. Knott, V. J.; Venables, P. H. **EEG Alpha Correlates of Alcohol Consumption in Smokers and Nonsmokers. Effects of Smoking and Smoking Deprivation.** *Journal of Studies on Alcohol* 40(3): 247-257, March 1979.

This investigation examined electroencephalogram (EEG) alpha correlates of smokers and nonsmokers under smoking and smoking deprivation conditions. Subjects were 35 male university students (26 smokers and 9 nonsmokers). Deprived (DS) and nondeprived smokers (NDS) participated in nontobacco (NT) and tobacco (T) conditions. There were no statistically significant differences between groups in age, weight, personality inventory scores, number of years of smoking, number of cigarettes/day, nicotine and tar yield of cigarettes, and liters of beer consumed/day. In each experimental session, electrode application was followed by a 5-min eyes-closed relaxation period during which a baseline EEG record was obtained. The baseline period was immediately followed by a 10-min period during which subjects drank alcoholic or nonalcoholic beverages. All subjects were required to drink the beverages slowly over a 10-min period. Tobacco smoking, for both DS-T and NDS-T subgroups (in alcohol and nonalcohol sessions), consisted of smoking at a constant inhalation rate of once every 30 sec during four 5-min periods spaced at 0-5, 10-15, 20-25, and 30-35 min (four cigarettes) from the beginning of ingestion of alcoholic and nonalcoholic beverages. Subjects in the nontobacco subgroups (NS-NT, DS-NT, NDS-NT) were required to inhale an unlighted cigarette at the same inhalation rate for the same 5-min periods. Filtered Benson and Hedges deluxe length were used, each cigarette containing 1.6 mg of nicotine and 21 mg of tar (manufacturer's estimates). Despite an apparent marked reduction in alpha amplitude of the DS-T group in the nonalcohol session, the analysis did not yield significant Group, Time, or Group x Time interaction effects. Analysis of dominant alpha frequency data yielded a significant Group effect ($p<0.05$), while a planned comparison of DS-NT and DS-T subgroups indicated a significant increase in dominant alpha frequency in the DS-T group ($p<0.005$, 1-tailed), indicating a stimulant effect of tobacco smoking. In addition, a comparison of NDS-NT and NDS-T subgroups yielded a significant effect ($p<0.005$, 1-tailed), the NDS-T group showing increased dominant alpha frequency after smoking. Comparison of alcohol and nonalcohol sessions within each group yielded significant reductions only in the NS-NT ($p<0.05$, 1-tailed) and DS-NT groups ($p<0.005$, 1-tailed). No significant differences were observed between NS-NT and DS-NT groups in the alcohol session.

80–1634. Kowal, N. E.; Johnson, D. E.; Kraemer, D. F.; Pahren, H. R. **Normal Levels of Cadmium in Diet, Urine, Blood, and Tissues of Inhabitants of the United States.** *Journal of Toxicology and Environmental Health* 5(6): 995-1014, 1979.

Cadmium (Cd) was measured in the feces, urine, blood, and hair of U.S. inhabitants without known high Cd exposure in Chicago, Illinois, and Dallas, Texas, and in autopsy tissues of accident victims in Dallas. The average intake of Cd in food was estimated to be 13-16µg/d and was higher for males than females. The average levels of Cd were 0.59-0.77µg/l urine, 0.09-0.11µg/100 ml blood, 0.83-1.10µg/g hair, 21µg/g kidney cortex, 1.2µg/g liver, 0.067µg/g muscle, 0.58µg/g pancreas, and 0.040µg/g fat. Hair Cd was higher for males than females. Cd levels increased with age in urine and all tissues and were higher in cigarette smokers than nonsmokers in urine, blood, and all tissues. (Auth. Abs.)

80–1635. Kraal, J. H.; Kenney, E. B. **The Response of Polymorphonuclear Leukocytes to Chemotactic Stimulation for Smokers and Non-Smokers.** *Journal of Periodontal Research* 14: 383-389, 1979.

Polymorphonuclear leukocyte (PMN) function may be an important determinant in resistance against periodontitis. Since tobacco smoke may interfere with PMN function, the chemotactic response of PMN to serum and saliva was studied in 10 smokers and 10 nonsmokers and again in the same subjects after all smoked one cigarette. The assay used in this investigation appeared to be sensitive enough to detect some differences between polymorphonuclear leukocyte chemotaxis in some individuals. However, no effect of smoking on the ability of the cells to migrate or the ability of serum or saliva to stimulate such migration was detected. (Auth. Abs.)

80–1636. Lehrer, S. B.; Wilson, M. R.; Salvaggio, J. E. **Immunogenicity of Tobacco Smoke Components in Rabbits and Mice.** *International Archives of Allergy and Applied Immunology* 62(1): 16-22, 1980.

Several extracts of tobacco leaf and tobacco smoke components were tested for their ability to stimulate and react with antibodies. Two experimental animal models, mice producing reaginic antibody and rabbits producing precipitating antibody, were used in this study. Results indicate that tobacco smoke extracts can stimulate immune responses to tobacco leaf antigens in rabbits and mice. The immunogen is apparently not a product of incineration because air passed through unlit cigarettes clearly extracted the antigenic component. (Auth. Abs.)

80–1637. Leuchtenberger, C.; Leuchtenberger, R.; Chapuis, L. **Difference in Response to Vitamin C Between Marihuana and Tobacco Smoke Exposed Human Cell Cultures.** *Advances in the Biosciences* 22/23: 209-218, July 22-23, 1978.

Marihuana smoke evoked in normal human lung and human breast cancer cultures (SK-Br-3) a significantly higher frequency of mitotic abnormalities than did tobacco smoke, regardless of whether the cultures were grown in the absence or the presence of vitamin C. The response to vitamin C of marihuana smoke-exposed breast cancer cultures was completely different from those exposed to tobacco smoke. While vitamin C caused a reduction of mitotic abnormalities in the tobacco smoke-exposed cultures and led to differentiation, these features were completely absent in marihuana smoke-exposed cultures. In the latter cultures, vitamin C accelerated not only the abnormal growth, but also the dedifferentiation of the breast cancer cells. (Auth. Abs.)

80–1638. Lewis, D. J.; Braybrook, K. J.; Prentice, D. E. **The Measurement of Ultrastructural Changes Induced by Tobacco Smoke in Rat Alveolar Macrophages. A Comparison of High and Low Tar Cigarettes.** *Toxicology Letters* 4: 175-181, September 1979.

Rats were exposed to tobacco smoke from high- and low-tar cigarettes for 6 weeks at two dose levels. Ultrastructural features of alveolar macrophages from the tobacco smoke-exposed and control rats were measured with an image-analyzing computer. Compared with control rats, there were statistically significant ($p<0.01$) increases in macrophage area, area to perimeter ratio, and the area of cytoplasm occupied by lysosomes in rats from both high-tar groups. There were no statistically significant changes in any parameter when rats from the low-tar groups were compared with control rats. (Auth. Abs.)

80–1639. Mackenzie, J. S.; Flower, R.L.P. **The Effect of Long-Term Exposure to Cigarette Smoke on the Susceptibility of Mice to Combined Infection With Influenza Virus and Bacteria.** *FEMS-Microbiology Letters* 5(2): 77-79, 1979.

The effect of immunosuppression by long-term exposure to cigarette smoke was studied in an experimental murine system. Female mice were exposed to fresh cigarette smoke equivalent to a human consumption of 20 to 30 cigarettes/day. Exposed mice and nonexposed age-matched controls were inoculated intranasally with influenza virus strain A/MEL (HONI) and the bacterial strains *H. influenzae* and *K. pneumoniae* and the susceptibility of smoke-exposed mice was compared to controls. Both bacterial strains were found to be avirulent at a dose of 2×10^2 colony-forming units if given alone, but if either bacterial strain was administered 4 days after a prior infection with 0.05 LD_{50} of influenza A/MEL, the smoke-exposed animals were found to be significantly more susceptible than unexposed, age-matched control animals, with death resulting from secondary bacterial pneumonia 7-14 days later. Results suggest that incidence of chronic and acute respiratory tract diseases associated with cigarette smoking in man may also be due to secondary bacterial invasion following respiratory virus infections.

80–1640. Maher, F. A.; Bing, O.H.L.; Huber, G. L.; Arenella, L.; Abelmann, W. H. **The Direct Effect of Tobacco Smoke on the Intrinsic Mechanical Properties of Cardiac Muscle.** *Environmental Research* 20: 282-288, 1979.

The direct effects of tobacco smoke from Kentucky University Reference cigarettes on the mechanical performance of isolated rat left ventricular muscle preparations were examined. Experiments were carried out with preparations contracting isometrically 12 times per minute at 28°C while at the apex of the length tension curve. Under oxygenated conditions, tobacco smoke bubbled through the bath induced an initial negative inotropic response. With larger volumes of smoke, initial depression was followed by recovery toward control values. Late recovery was inhibited by first passing the tobacco smoke through a Cambridge filter. Tobacco smoke added prior to 60 min of hypoxia, followed by 30 min of reoxygenation, had a dose—dependent deleterious effect on muscle performance during hypoxia and subsequent reoxygenation. The deleterious effect of smoke during hypoxia could largely be prevented by first passing the tobacco smoke through a Cambridge filter. The nicotine present in tobacco smoke could not by itself account for the changes under oxygenated conditions or those seen during hypoxia and reoxygenation. It is concluded that the gaseous phase of tobacco smoke is responsible for the negative inotropic effect seen under oxygenated conditions. The action of the particulate phase of the smoke (which is blocked at least in part by the Cambridge filter) appears to be responsible for the partial recovery of mechanical performance under oxygenated conditions and the potent deleterious effects seen during hypoxia and reoxygenation. (Auth. Abs.)

80–1641. Maksud, M. G.; Baron, A. **Physiological Responses to Exercise in Chronic Cigarette and Marijuana Users.** *European Journal of Applied Physiology* 43(2): 127-134, 1980.

Several physiologic responses to graded exercise were determined in four groups of young adult blue-collar workers identified as users of (1) marihuana, (2) cigarettes, (3) both marihuana and cigarettes, or (4) neither substance. Using covariate analysis of variance to control for race, age, height, and weight, the groups did not differ in estimated percent fat, lean body mass, hematocrit levels, or blood hemoglobin values. Heart rate and oxygen consumption responses also were similar at all levels of exercise. The mean minute ventilation and perceived exertion were found to be higher ($p<0.05$) in cigarette smokers than nonsmokers of cigarettes, but aerobic power and the physical work capacity did not differ. The differences in pulmonary function observed suggest the need for further study on older populations of chronic cigarette and marihuana users. (Auth. Abs.)

80–1642. Marcus, A. H.; Czajkowski, S., Jr. **Moments of the Exit-Time Distribution for Carboxyhemoglobin Due to Cigarette Smoking.** *Journal of Applied Probability* 15(1): 13-25, March 1978.

Linear (i.e., shot noise) and nonlinear (i.e., Coburn-Forster-Kane equation) secondary Poisson process models are developed for the level of carboxyhemoglobin due to carbon monoxide from cigarette smoke. The moments of the time of first exit above a critical threshold are expanded as power series in the initial state of the process. Recurrence relations for the coefficients in the moment series are solved numerically. An explicit formula involving Poisson probability functions is obtained for the mean exit time of shot noise. (Auth. Abs. Mod.)

80–1643. Merrill, W. W.; Goodenberger, D.; Strober, W.; Matthay, R. A.; Naegel, G. P.; Reynolds, H. Y. **Free Secretory Component and Other Proteins in Human Lung Lavage.** *American Review of Respiratory Disease* 122(1): 156-161, July 1980.

Subsegmental bronchoalveolar lavage of the lingula and right middle lobe was performed in a group of normal subjects (32 cigarette smokers and 24 nonsmokers) to analyze the recoverable fluid for its content of respiratory cells, several proteins including IgG and IgA, and free secretory component, a glycoprotein elaborated by serous bronchial epithelial cells. The lavage fluid obtained from the smokers consistently demonstrated an increased ($p<0.001$) cell yield, reflecting primarily an increase in alveolar macrophages. In addition, the number of polymorphonuclear granulocytes was tenfold greater in smokers, and the total number of lymphocytes recovered was approximately twice that of nonsmokers. As in previous studies, the analysis of proteins contained in bronchoalveolar lavage (BAL) revealed an increase in the ratio of IgA to albumin in both smokers and nonsmokers, as compared with serum, but an increase in the IgG/albumin ratio in BAL was found only in smokers. The total amount of secretory component recovered in lung lavage was significantly ($p<0.015$) decreased in 20 percent of the cigarette smokers. Surprisingly, this decrease did not correlate directly with pack-years smoked. It is possible that this glycoprotein may be an indicator of biochemical dysfunction of epithelial cells after inhalant exposure to cigarette smoke, which occurs relatively early after exposure, and is perhaps independent of smoking history. (Auth. Abs.)

80–1644. Nicolov, I. G.; Chernozemsky, I. N. **Tumors and Hyperplastic Lesions in Syrian Hamsters Following Transplacental and Neonatal Treatment With Cigarette Smoke Condensate.** *Journal of Cancer Research and Clinical Oncology* 94(12): 249-256, 1979.

Cigarette smoke condensate (CSC) in olive oil was injected into outbred Syrian hamsters: in adults intraperitoneally on the 10th-14th day of gestation, total dose 1.5-2.5 mg/g body weight; in 12- to 14-day-old animals subcutaneously, total dose 0.5-1.5 mg/animal. Following 15-25 months of observation, benign and malignant neoplasms in various locations were found in 2/58 (3.4 percent) females treated during pregnancy; in 17/51 (33.3 percent) of their transplacentally exposed offsprings; in 5/53 (9.4 percent) of neonatally treated hamsters. In the last two groups, females were more affected than males. Tumors of the adrenal glands, pancreas, female sex organs, and liver were most frequent. No tumors appeared in controls, either untreated or injected with olive oil. In addition, hyperplastic lesions, in particular multiple liver cysts and cholangiomatosis, were also observed, mainly in animals exposed transplacentally and as neonates. (Auth. Abs.)

80–1645. Oldendorf, W.; Braun, L.; Cornford, E. **pH Dependence of Blood-Brain Barrier Permeability to Lactate and Nicotine.** *Stroke* 10(5): 577-581, September-October 1979.

Brain uptake of radiolabeled D- and L-lactate, D-glucose, and nicotine, as measured by the intracarotid bolus method, was examined in rats over a range of pH of the injected solution. The uptake of L-lactate was highest at pH 6.1, and lowered significantly at pH 7.2, 7.5, and 8.4. In contrast, the uptake of the D-enantiomer was not as dramatically affected. Glucose uptake was not affected by alterations in pH. Nicotine uptake decreased with pH reduction through a range of 8.3-4.2. These data suggest that it is the uncharged molecule which penetrates the blood-brain barrier by both carrier and lipid mediation. A mechanism relating to these observations is postulated and possible relevance to lactate washout from ischemic brain is discussed. (Auth. Abs.)

80–1646. Paxton, R.; Bernacca, G. **Urinary Nicotine Concentration as a Function of Time Since Last Cigarette: Implications for Detecting Faking in Smoking Clinics.** *Behavior Therapy* 10: 523-528, 1979.

Nicotine concentration and the ratio of nicotine to creatinine were assessed in urine samples collected from 26 smokers and 14 nonsmokers. The smokers were asked to provide serial samples after their last cigarette and to refrain from smoking for up to 24 hours. Nicotine concentration correlated inversely with time since the last cigarette, and this measure discriminated between smokers who had smoked within the past 15 hours and nonsmokers. The reliability of the procedure was adequate, and a level of nicotine concentration which gives low error probabilities was suggested as a cutoff point for detecting smoking. The relative merits of different biochemical procedures for detecting smoking are briefly discussed. (Auth. Abs.)

80–1647. Pfaffinger, I.; Marshall, M.; Hess, H.; Staubesand, J. **Die akute Wirkung von Zigarettenrauch- oder Kohlenmonoxidinhalation auf die Arterienwand beim Miniaturschwein. [Acute Effect of Cigarette Smoke or Carbon Monoxide Inhalation on the Arterial Wall of Miniature Pigs.]** *Verhandlungen der Deutschen Gesellschaft fuer innere Medizin* 22: 887-890, 1979, German.

In a series of experiments in a smoke inhalation chamber, 26 miniature pigs, on the average 6 months old, were exposed to cigarette smoke, carbon monoxide (CO), or compressed air for 4 to 64 hours. In a second series, 35 male miniature pigs, 3-4 months old, were made to inhale cigarette smoke, 200 ppm CO in synthetic atmosphere, or air, for 4 hours through a tight mask. After a short exposure to cigarette smoke or CO, the scanning electron micrographs of the arterial wall showed typical thrombocyte adhesions to an apparently intact endothelium. After a 2-week experiment with cigarette smoke and in 4-day experiments with CO in concentrations up to 500 ppm, heavy stratified deposits of thrombocytes and fibrin, i.e., parietal thrombi, were found. Blind evaluation of the micrographs failed to detect any adherent thrombocytes after air exposure. In addition, increased endothelial changes, fibrin deposits, and larger mononuclear cells on the endothelium were detected in cigarette smoke experiments. Thrombocyte adhesion and, more often, mononuclear cells were observed after a single 4-hour CO or cigarette smoke inhalation. Almost identical results obtained by exposure to 500 ppm CO and by a 0.05 mg/kg intravenous injection of nicotine show an apparent reaction to stress. The experiments with CO concentrations show changes in humoral parameters (hematocrit, blood viscosity, plasma viscosity, platelet aggregation) at CO concentrations above 200 ppm. Apparently, CO unfavorably affects the interaction between thrombocytes and the arterial wall.

80–1648. Reznik, G.; Borgmeyer, L. **Deposition and Clearance of Labeled Cigarette Smoke Particles in Rat Respiratory and Digestive Tract.** *Journal of Toxicology and Environmental Health* 6(2): 433-444, March 1980.

In this investigation, 150 8-week-old Sprague-Dawley rats inhaled smoke from ^{14}C-labeled cigarettes in a Hamburg II smoking machine. The research cigarettes were labeled with (16,17-^{14}C)dotriacontane. Eighty of the rats were acclimatized to smoke from nonlabeled research cigarettes for 21 days before inhaling labeled cigarette smoke. The investigation was divided into four experiments. In experiment I, all rats were killed immediately after exposure to the ^{14}C-labeled smoke from 30 cigarettes (10μCi/cigarette) and the organs of the respiratory and digestive tracts were measured for ^{14}C activity. These animals showed high activity in the apex nasi, fundus nasi, and larynx. The highest activity was found in the lobus dexter caudalis and the lobus sinister of the lung. The lungs contained four-fifths of the condensate inhaled into the respiratory tract. In experiment II, three animals were killed immediately after smoking, three after 6 hours, and four after 24 hours. No activity was found after 6 hours in apex, fundus nasi, larynx, and trachea. The lungs showed no clearance at all. The activity in the digestive tract was highest in the stomach and the esophagus immediately after cessation of smoking. These values decreased rapidly after 6 hours. In experiment III, the animals were killed after 7, 21, and 42 days. Clearance from the upper respiratory tract was fast, whereas clearance from the lungs was slow. Clearance from all lung lobes was faster in the nonacclimatized animals. In experiment IV, the rats inhaled the smoke from ^{14}C-labeled cigarettes (2μCi/cigarette) on 5 successive days. The animals were killed 7, 14, and 21 days after the last smoke inhalation. Clearance from the lungs was slower in acclimatized and nonacclimatized animals than in experiment III. (Auth. Abs.)

80–1649. Rogers, W. R.; Bass, R. L., III.; Johnson, D. E.; Kruski, A. W.; McMahan, C. A.; Montiel, M. M.; Mott, G. E.; Wilbur, R. L.; McGill, H. C., Jr. **Atherosclerosis-Related Responses to Cigarette Smoking in the Baboon.** *Circulation* 61(6): 1188-1193, June 1980.

Thirty-six young adult male baboons (*Papio cynocephalus*) were fed an atherogenic diet (40 percent calories from lard, 1.5 mg cholesterol/kcal) and taught to puff by operant conditioning with water rewards. Eighteen baboons (smokers) were randomly assigned to smoke 43 cigarettes/day, and 18 baboons (shams) were randomly assigned to puff air under conditions equivalent to those of the experimental group. During 14-19 months of smoking, the baboons had significantly higher carbon monoxide and thiocyanate concentrations in blood, and cotinine concentrations in urine. (The mean cotinine concentration in the urine was 0.55μg/ml.) There were no significant differences in serum total cholesterol, very low density lipoprotein and low density lipoprotein cholesterol, high density lipoprotein cholesterol, or triglyceride concentrations of smokers and shams. Smoking baboons had significantly higher fasting blood glucose concentrations and lymphocyte counts ($p<0.05$). Platelet count, platelet aggregation, red blood cell count, hematocrit, food and water intake, and body weight were not significantly different in the two groups. (Auth. Abs. Mod.)

80–1650. Rogowsky, M.; van der Putten, M.; Baudoux, A. **Etude Critique de la Formation de Carboxyhemoglobine (HbCO) Chez les Soudeurs Travaillant en Atmosphere a Faible Concentration d'Oxyde de Carbone (CO). [Critical Study of the Formation of Carboxyhemoglobin Among Welders Working in a Low-Level Carbon Monoxide Environment.]** *Archives des Maladies Professionelles, de Medecine du Travail et de Securite Sociale* 39(3): 151-161, March 1978, French.

Carboxyhemoglobin (HbCO) levels were measured in 35 welders after 6 hours work in a low-level carbon monoxide environment. High levels of HbCO were found only in workers who were cigarette smokers (5.25 percent in light smokers (1-20 cigarettes/day) and 8.43 percent in heavy smokers (>20 cigarettes/day)). The slight increase in HbCO levels in nonsmokers should be considered the result of an augmentation of the CO level in the air by the reaction $C—CO_2 = 2\,CO$. (Auth. Abs. Mod.)

80–1651. Rosenkrantz, H.; Fleischman, R. W. **Effects of Cannabis on Lungs.** *Advances in the Biosciences* 22/23: 279-299, July 22-23, 1978.

Because of reported changes in lung function and morphology in cannabis users and animals exposed to marihuana smoke, a rat model was developed for standardized automatic chronic exposure to marihuana smoke. The smoking apparatus was regulated to present a 50-ml puff volume simultaneously from each of three National Institute on Drug Abuse cigarettes in a 2-sec puff for a 30-sec exposure interval followed by 30 sec of fresh air each min. Estimated Δ^9-tetrahydrocannabinol (THC) doses in marihuana smoke were 0.4-5 mg/kg and were relevant to those used by man. The doses were attained by varying the number of puffs from 4 to 16 which correlated with carboxyhemoglobin levels between 15 and 48 percent and plasma Δ^9-THC levels of 34-313 ng/ml.

The latter were similar to those found in human cannabis consumers (20-300 ng/ml). Behavioral signs of central nervous system (CNS)-inhibition and CNS-stimulation were in accord with dosages, and development of tolerance was observed. No pulmonary pathology could be attributed to marihuana smoke when exposure was for 14-57 days. In contrast, exposures for 87-360 days induced a dose-related focal alveolitis or pneumonitis which progressed from extensive mobilization of alveolar macrophages and foreign body cell inflammation to pronounced focal proliferative aberrations associated with focal granulomatous and cholesterol clefts. In all chronic studies, the effects of marihuana smoke were distinguished from those of placebo and tobacco smoke. The lung pathology was due to a combination of nonspecific smoke particulates and cannabinoids. (Auth. Abs.)

80–1652. Rotenberg, K. S. **The Pharmacokinetics of Nicotine Following Intravenous Administration and Cigarette Smoke Inhalation in the Rat.** Doctoral Dissertation, University of Maryland, Baltimore, University Microfilms International 78-10,862, 1978, 203 pp.

The pharmacokinetics of nicotine (^{14}C-methyl) were evaluated in male rats following either cigarette smoke inhalation or intravenous injection of 0.08, 0.40, or 0.80 mg/kg nicotine. Following intravenous administration and independent of dose, the plasma concentration of nicotine declined biexponentially, with a mean half-life ranging from 0.92 to 1.10 hours. However, the total plasma clearance of nicotine increased ($p<0.05$) from a mean value of 2.53 l/hr/kg at the 0.08 mg/kg dose to a mean value of 3.88 and 4.04 at the 0.40 and 0.80 mg/kg doses, respectively. There were no dose-related differences in either the formation half-life of the major metabolite, cotinine (range 0.33 to 0.46 hours) or the elimination half-life (range 5.39 to 6.63 hours). However, the plasma concentration vs. time curves (AUC) of cotinine did not increase proportionately with the dose of nicotine, indicating that the dose-related increase in the total plasma clearance of nicotine is not due to an increase in its biotransformation to cotinine. The urinary excretion of nicotine and its metabolites were dose independent. The dose of nicotine absorbed ranged from 0.03 to 0.22 mg/kg. Plasma nicotine concentration following cigarette smoke inhalation declined biexponentially similar to the decline of nicotine after intravenous administration. There were no significant differences in the pharmacokinetic parameters of nicotine, cotinine, and origin activity between the cigarette smoke inhalation study and the intravenous study. (Auth. Abs. Mod.)

80–1653. Rowe, J. W.; Kilgore, A.; Robertson, G. L. **Evidence in Man That Cigarette Smoking Induces Vasopressin Release Via an Airway-Specific Mechanism.** *Journal of Clinical Endocrinology and Metabolism* 51(1): 170-172, July 1980.

The plasma vasopressin response to intravenous nicotine (2 mg) and smoking cigarettes of high- (1.2 mg) and low- (0.6 mg) nicotine content was studied in healthy young subjects with a history of cigarette smoking. Nicotine administered intravenously had no effect on plasma vasopressin. There was a statistically significant increase in plasma vasopressin after both the low- and high-nicotine content cigarettes, with the high-nicotine content cigarettes resulting in a significantly greater increase. All three interventions resulted in transient increases in blood pressure and pulse of similar magnitude. These studies indicate that cigarette smoking-induced vasopressin release is not mediated via an action of circulating nicotine, and suggest the presence of an airway-specific mechanism for vasopressin release. (Auth. Abs.)

80–1654. Ryan, L. J.; Barr, J. E.; Sanders, B.; Sharpless, S. K. **Electrophysiological Responses to Ethanol, Pentobarbital, and Nicotine in Mice Genetically Selected for Differential Sensitivity to Ethanol.** *Journal of Comparative and Physiological Psychology* 93(6): 1035-1052, December 1979.

Cortical electroencephalographic (EEG) changes induced by ethanol (4.3 and 1.4 g/kg), pentobarbital (50 and 16 mg/kg), and nicotine (1.0 g/kg) were examined in long-sleep (LS) and short-sleep (SS) mice that were genetically selected for differential sleep times induced by a hypnotic dosage of ethanol. Ethanol (4.3 g/kg) caused EEG changes that paralleled the behavioral differences, whereas no differences between selected lines were observed following the activating dose (1.4 g/kg). Data support the notion that the known difference in ethanol sleep times is due not to greater SS sensitivity to ethanol activation but rather to greater LS sensitivity to ethanol hypnosis. No differences between selected lines were observed following 50 mg/kg pentobarbital, which again parallels previous behavioral data. The SS mice were more responsive to pentobarbital activation (16 mg/kg). Nicotine more severely reduced EEG power ($p<0.01$) and heart rate ($p<0.01$) in LS mice; a continuous infusion of nicotine elicited a distinct pattern of behavioral stereotypy for each selected line, with more profound motor and reflex depression in LS mice. The lines do not differ in rate of nicotine metabolism, hence they must differ in central nervous system sensitivity to nicotine. Thus, lines of mice selectively bred for differential sensitivity to ethanol also display marked differences in electrophysiological and behavioral responses to nicotine. (Auth. Abs.)

80–1655. Scharfman, A.; Lafitte, J. J.; Tonnel, A. B.; Aerts, C.; Sablonniere, B.; Roussel, P. **Glycosidases and Proteases of Alveolar Macrophages Obtained by Bronchoalveolar Lavage from Smokers and Non-Smokers.** *Lung* 157(3): 135-142, 1980.

Glycosidase (i.e., N-acetyl-β-D-glucosaminidase, N-acetyl-β-D-galactosaminidase, β-D-galactosidase, α-L-galactosidase, α-D-mannosidase, α-L-fucosidase, β-D-glucuronidase) and protease (i.e., elastolytic; chymotrypsin; cathepsin B, C, and D; collagenase) activities were characterized in alveolar macrophages obtained by bronchoalveolar lavage from two nonsmokers, two light (5-10 cigarettes/day) smokers, and two heavy (>20 cigarettes/day for 10 years) smokers. These enzymatic activities were lower in nonsmokers than in smokers. The glycosidase activities for the light smokers were intermediate between those of nonsmokers and heavy smokers. (Auth. Abs. Mod.)

80–1656. Schievelbein, H.; Heinemann, G.; Loschenkohl, K.; Troll, C.; Schlegel, J. **Metabolic Aspects of Smoking Behaviour.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 371-379.

Parameters indicative of metabolism in smokers (e.g., erythrocyte count, mean cell hemoglobin, glucose-6-phosphate dehydrogenase, 2,3-diphosphoglycerate, nicotine, cotinine, carboxyhemoglobin) were measured in two experiments. Subjects included 30 smokers (15 males, 15 females) and 10 nonsmokers (5 males, 5 females). Smokers were evaluated after smoking their usual cigarette brand and four other brands. Almost all metabolic parameters were higher for smokers than for nonsmokers. The data also indicated a significant sex difference in nicotine and cotinine excretion; male smokers excreted more of these two compounds than female smokers.

80–1657. Schoenfisch, W. H.; Hoop, K. A.; Struelens, B. S. **Carbon Monoxide Absorption Through the Oral and Nasal Mucosae of Cynomolgus Monkeys.** *Archives of Environmental Health* 35(3): 152-154, May-June 1980.

Previous studies have shown that blood levels of carbon monoxide increase during cigarette smoking. It has generally been assumed that increases in blood levels of carbon monoxide could be interpreted as evidence that deep lung penetration of cigarette smoke had occurred. This study was designed to examine whether increased blood levels of carbon monoxide could result from absorption in the nasal and oral cavities. The nasal and oral cavities of cynomolgus monkeys were exposed, independently of the lungs, to cigarette smoke under rigorous smoking conditions. Pre- and post-exposure blood levels of carbon monoxide were measured. As a positive control, similar volumes of cigarette smoke were passed directly into the lungs, thus bypassing the oral and nasal cavities, and blood levels of carbon monoxide were again measured. The results indicate that absorption of carbon monoxide in the oral and nasal cavities is negligible under the heavy smoking regimen employed here, and hence, would be negligible under normal smoking conditions. (Auth. Abs.)

80–1658. Shors, E. C.; Fu, P. C.; Matsumura, K.; Cohen, A. H.; Benfield, J. R. **Sustained Release of Benzo(*a*)pyrene From Silicone Polymer Into the Tracheobronchial Tree of Hamsters and Dogs.** *Cancer Research* 40(7): 2288-2294, July 1980.

A method was developed to expose specific sites of the hamster and canine tracheobronchial tree to benzo(*a*)pyrene (BP) at quantitatively sustained rates. Implants for sustained release were formed by incorporating BP in a silicone rubber matrix at concentrations of 9.05 to 12 percent. Forty-nine hamsters and 12 dogs had a total of 86 implants surgically adhered to the tracheobronchial mucosa for up to 200 days. BP was released from the implants in hamsters and dogs as a first-order exponential function with a half-time of 54.8 and 44.5 days, respectively. Pathogenesis was progressively time- and dose-dependent. Squamous metaplasia with atypia regularly occurred in dogs within 150 days or after 7.17 mg BP, and in hamsters after 50 days or 288 μg BP. Bronchogenic cancers occurred in 93 percent of our hamsters after 100 days and 467 μg BP. This method has applicability potentially as a bioassay for evaluating carcinogens in hamsters and currently as a means of producing a model of lung cancer in which neoplasia is induced at precise, selected sites. (Auth. Abs.)

80–1659. Stenback, F.; Rowland, J. **Experimental Respiratory Carcinogenesis in Hamsters: Environmental, Physicochemical and Biological Aspects.** *Oncology* 36(2): 63-71, 1979.

The respiratory tract of Syrian golden hamsters was exposed to polycyclic hydrocarbons alone and in combination with dusts. These included benzo(*a*)pyrene (BaP), silicon dioxide (SiO_2), manganese dioxide (MnO_2), and agar gelatin (gel), as well as dibenz(*a,j*)acridine (DBA) in combination with the carrier dust ferric oxide (Fe_2O_3). The purpose of the study was to evaluate the possible environmental hazards of these compounds and the physicochemical characteristics determining their carcinogenic activity. The prognostic significance of premalignant alterations in relation to treatment by carcinogenic and noncarcinogenic agents, and the applicability of respiratory tumor classification in this system were also studied. When instilled alone, the dusts induced bronchial nonspecific epithelial alterations, interstitial cell proliferation, and a few granulomatous changes in the lung. No respiratory tract tumors were seen. BaP alone induced only tracheal epithelial alterations, desquamation and metaplasia, as well as five papillomas and one squamous cell carcinoma. BaP in a suspension of gelatin-saline induced a few papillomas and squamous cell carcinomas of the trachea and lungs. SiO_2 with BaP was highly effective and induced papillomas, squamous cell carcinomas, adenomas, and adenocarcinomas of the larynx, trachea, and lungs. They were preceded by epithelial proliferative dysplastic alterations, not seen in animals treated with BaP and MnO_2, or DBA and Fe_2O_3. These groups also failed to exhibit a significant neoplastic response. The tumors could be divided into benign and malignant, and glandular or squamous cell tumors. The trachea, main bronchi, bronchioli, and lungs showed the efficacy and applicability of a classification similar to that used in man. It is also concluded that preneoplastic alterations sufficient in degree and extent are indicative of subsequent tumor formation. (Auth. Abs.)

80–1660. Topping, D. C.; Griesemer, R. A.; Nettesheim, P. **Development and Fate of Focal Epithelial Lesions in Tracheal Mucosa Following Exposure to 7,12-Dimethylbenz(*a*)anthracene.** *Cancer Research* 39(12): 4829-4837, December 1979.

Rat tracheal transplants were exposed to 7,12-dimethylbenz(*a*)anthracene delivered continuously over a 4-week period from intraluminal pellets. Groups of tracheas were collected from 2 to 12 months after cessation of carcinogen exposure, and the number and type of focal lesions were scored. These focal lesions had previously been shown to arise well after cessation of carcinogen exposure. The highest incidence of focal lesions (all types) was found at 4 months. Over the subsequent 8 months, the incidence of tracheas with lesions declined from 72 to 44 percent, and the average number of lesions per exposed trachea declined from 2.5 to 1.3. Metaplasias with no or minimal atypia were the most common, and the drop in overall lesion incidence between 4 and 12 months was due primarily to a decrease in the observed frequency of these types of lesions, a finding which indicates that they are reversible. Significant numbers of more advanced (dysplastic) lesions occurred only from 4 months on. The incidence of the "advanced" lesions did not decline between 4 and 12 months. Invasive carcinomas first occurred at 4 months with an incidence of 5 percent. This did not change significantly during the subsequent time intervals. A tumor induction study with a total of 86 tracheas was carried out over a 28-month period. The same carcinogen exposure was used as in the serial sampling study. The cumulative incidence of invasive carcinomas was 9 percent (that of all carcinomas, including carcinoma in situ, was 15 percent), with the first carcinoma appearing at 10 months and a mean induction time of 14 months. The cumulative incidence of advanced lesions in this study was 10 percent, but none of the tracheas collected more than 2 years after exposure contained carcinomas or advanced lesions. The data from these two experiments clearly indicate that in tracheal epithelium exposed to a low dose of 7,12-dimthylbenz(*a*)anthracene: (a) a large number of carcinogen-induced lesions occur well after the acute toxic effects of 7,12-dimethylbenz(*a*)anthracene have passed; (b) the morphologically more advanced lesions develop with latent periods of 4 or more months; (c) many of these advanced lesions appear to persist for long periods of time without progressing further, while some may evolve into invasive neoplasms; and (d) most of the metaplastic lesions with little or no atypia regress, and some of the advanced lesions also appear to ultimately regress. (Auth. Abs.)

80–1661. Topping, D. C.; Griesemer, R. A.; Nettesheim, P. **Quantitative Assessment of Generalized Epithelial Changes in Tracheal Mucosa Following Exposure to 7,12-Dimethylbenz(*a*)anthracene.** *Cancer Research* 39(12): 4823-4828, December 1979.

Sequential morphological changes occurring after brief carcinogen exposures of heterotopic tracheal transplants in rats were semiquantitatively studied. Tracheas were exposed to 7,12-dimethylbenz(*a*)anthracene for 1, 2, or 4 weeks, during which time means of 138, 152, and 160μg 7,12-dimethylbenz(*a*)anthracene, respectively, were delivered. The first two types of exposures resulted only in generalized epithelial changes; these included hyperplasia and early metaplasia, both of which regressed rapidly, and persistent atrophic alterations. No focal epithelial lesions or tumors developed. The third type of exposure (160μg 7,12-dimethylbenz(*a*)anthracene delivered in 4 weeks) resulted in the appearance of generalized mucosal changes with long-lasting, severe inhibition of mucus production. In addition, focal metaplastic lesions reappeared at 4-8 months after exposure, and invasive carcinomas developed after 1 year with an incidence of 9 percent. Overall carcinoma incidence, including carcinoma in situ, was 15 percent. The studies emphasize the importance of the duration of carcinogen exposure, and they demonstrate the emergence of focal lesions when effective carcinogenic exposures are being used. The possible significance of epithelial atrophy in the pathogenesis of cancer in this experimental model is discussed. (Auth. Abs.)

80–1662. Werner, C. **A Comparison of the Acute and Chronic Effects of Nicotine and Amphetamine on the Regulation of Food Intake, Water Intake, and Body Weight in Rats.** Doctoral Dissertation, Swiss Federal Institute of Technology, Zurich, 1978, 157 pp.

Nicotine and amphetamine were chronically and acutely administered in male rats (Roman Low Avoidance and High Avoidance lines), and the effects of these drugs on the regulation of food intake, water intake, and body weight were evaluated. When rats that had free access to food and water received two daily injections of nicotine (0.2 and 0.4 mg/kg) during the dark phase of the 12:12 hour day-night cycle for 6 weeks, food and water intake were reduced but eventually returned to control levels, whereas body weight was chronically reduced for the duration of treatment. Upon drug withdrawal, food and water consumption were increased until the animals reached the weight level of saline-control rats. These changes in food and water intake and body weight were dose-dependent and unaffected by age and weight, but differed with respect to the magnitude of the effect, but not the temporal pattern of occurrence in the two rat lines. Twice daily injections of 2.0 mg/kg d,l-amphetamine induced similar effects on food and water intake and body weight. In contrast to nicotine-treated rats that showed a gradual development of anorexia over a 3-day period and a protracted action upon drug withdrawal, amphetamine anorexia was maximal on the first day of drug treatment and the effects dissipated rapidly. Rats that underwent weight reduction to the expected drug-dependent level of weight regulation prior to chronic drug treatment did not show the anorexia normally observed during nicotine and amphetamine administration and no further weight loss was seen. Rats maintained on an ad libitum feeding and drinking regimen that were tolerant to the anorectic effects of nicotine and had stabilized body weight were switched to chronic amphetamine treatment and vice versa. Upon both drug substitutions no changes in food intake and body weight occurred. Rats with free access to water that were only permitted to eat for the first 4 hours of the 12-hour dark period showed the same pattern of anorexia, hypodipsia, and chronically reduced body weight when treated with nicotine as did rats with free food access. No cross-tolerance to the effects on food and water intake was seen in rats maintained on the 4-hour feeding regimen when nicotine was substituted for amphetamine or vice versa. Drug-naive and drug-tolerant rats that were maintained on the 4-hour feeding regimen with free access to water were pretreated with different receptor blocking agents 15 min before drug treatment and food access. In drug-naive rats nicotine-induced anorexia seemed to be mediated through cholinergic and dopaminergic mechanisms, whereas amphetamine-anorexia was probably dopaminergically mediated. In drug-tolerant rats pretreatment with the various receptor blockers had different effects than those observed in drug-naive rats. The present investigation demonstrated that chronically injected nicotine and amphetamine induced comparable effects on food intake and body weight in free-feeding rats but different effects in rats that had only 4 hours daily access to food. (Auth. Abs. Mod.)

80–1663. Wilson, A. J.; Schaefer, K. E. **Effect of Prolonged Exposure to Elevated Carbon Monoxide and Carbon Dioxide Levels on Red Blood Cell Parameters During Submarine Patrols.** *Undersea Biomedical Research* 6(Submarine Supplement): S49-S56, 1979.

Twenty volunteers were chosen at random from the crew of a Polaris submarine to ascertain the effect of prolonged exposure to elevated levels of carbon monoxide (CO) and carbon dioxide (CO_2) on red blood cell parameters during a routine patrol. Measurements were made of hemoglobin (Hb) concentrations, hematocrit (Hct) levels, and red blood cell (RBC) and reticulocyte counts. The average CO concentrations on red blood cell parameters in submarine patrolmen during patrol remained below 20 ppm (range 15-20); the average CO_2 concentration was 0.9 percent. A baseline study was performed prior to patrol while the subjects were breathing atmospheric air. Three determinations were performed at different times during patrol. These studies revealed a statistically significant rise in Hb, Hct, and RBC counts during the first 32 days of patrol and a subsequent decline almost to baseline levels on the 52nd day of patrol. Chronic exposure to low levels of CO (caused by smoking) is known to produce a rise in Hb, Hct, and red cells, to compensate for the anoxic stress induced by higher carboxyhemoglobin levels. Inhalation of CO_2 in higher concentrations (5-8 percent CO_2) causes an increase in ventilation and higher oxygen tensions, which are known to inhibit the erythropoietic response caused by acute or chronic hypoxia. However, the CO_2 concentration of 0.9 percent found in submarines is apparently too low to compensate for the slight anoxic stress produced by increased levels of CO. Both smokers and nonsmokers showed this response; however, only smokers exhibited a rise in reticulocyte counts. (Auth. Abs.)

80–1664. Wysocki, J.; Bakaj, J. **Bialka Surowicy Krwi u Palaczy Papierosow. [Serum Proteins in Cigarette Smokers.]** *Polski Tygodnik Lekarski* 34(47): 1829-1831, November 19, 1979, Polish.

In 108 blood donors (27 nonsmokers and 81 cigarette smokers), the serum concentrations of total protein, protein fractions, and main immunoelectrophoretic fractions of alpha$_1$-antitrypsin, alpha$_2$-macroglobulin, alpha$_2$-haptoglobin, alpha$_2$-coeruloplasmin, beta-transferrin, components C3 and C4 of complement, and properdin were determined. Numerous significant changes were found in serum proteins in habitual smokers. These changes are related to subclinical bronchitis and also to possible lowering of protein synthesis in cigarette smokers. (Auth. Abs.)

80–1665. Yoshida, K.; Imura, H. **Nicotinic Cholinergic Receptors in Brain Synaptosomes.** *Brain Research* 172(3): 453-459, August 31, 1979.

In order to elucidate pharmacological characteristics of nicotinic receptors in the brain, competitive binding of nicotine analogues or cholinergic agents to brain particles was studied utilizing (^{3}H) nicotine. The binding of (^{3}H)nicotine to rat brain crude mitochondrial or synaptosomal fraction was progressively inhibited by the addition of increasing amounts of native nicotine or nornicotine, but cotinine had little effect. Of the myelin synaptosomal, and mitochondrial subfractions of the crude mitochondrial fraction, (^{3}H)nicotine binding was almost exclusively confined to synaptosomes. In this binding, the affinity constant was 1.97×10^9/M and the maximum binding capacity was 7.30×10^{-8} M/g of protein. This binding was reduced by 50 percent in the presence of 4×10^{-7} M D-tubocurarine and 10^{-6} M carbamylcholine, while atropine was the least effective of the drugs tested. These findings suggest the possible existence of nicotinic cholinergic receptors in the synaptic region. A study of regional differences in nicotinic cholinergic receptors showed that higher specific bindings of nicotine to synaptosomes occurred in the hypothalamus, hippocampus, and thalamus. These findings suggest that the nicotinic cholinergic mechanism plays an important role in these regions of the central nervous system. (Auth. Abs.)

See also, **80–1675, 80–1760, 80–1772, 80–1777, 80–1785, 80–1793**

80–1666. Asp, S.; Hernberg, S.; Collan, Y. **Mortality Among Finnish Doctors, 1953-1972.** *Scandinavian Journal of Social Medicine* 7(2): 55-62, 1979.

The mortality with respect to the total population of Finnish physicians during the period 1953-1972 has been analyzed and compared with the corresponding statistics for the general Finnish population, for Finnish foundry workers, and for American physicians. It was found that the overall mortality was lower for male physicians than for the general population or for foundry workers, but was clearly higher than that for American physicians. Male physicians did not exhibit any major differences from the general population with regard to cardiovascular diseases and suicide, but had a lower mortality from malignant neoplasms, accidents, and "other diseases" (including infectious diseases). The explanation for cancer mortality being lower than expected among male physicians was mainly found in a deficit in lung cancer. Although female physicians had a higher life expectancy than both male physicians and the general female Finnish population, they did not show any clear deficit for cancer. With respect to all specialists, surgical specialists had the lowest mortality and general practitioners the highest. Most of these variations were attributable to differences in coronary mortality, but mortality from lung cancer was also remarkably low among surgeons. Differences of a similar type were also found between occupational sub-categories; private practitioners had the highest, and research workers and central hospital physicians the lowest mortality figures. The lower cancer mortality among male physicians, as contrasted with the general population, is probably attributable to differences in smoking habits; about 22 percent of male physicians smoked in 1973, whereas earlier studies have indicated that the corresponding proportion was about 50 percent in the general population. In contrast, the differences in mortality between different specialist categories probably arise from other factors, since Finnish physicians reportedly display a relatively homogeneous smoking pattern. (Auth. Abs.)

80–1667. Hackett, B. J. **The Influence of Alcohol and Cigarettes on Invalidity Retirement.** *Australian Family Physician* 9(2): 115-118, February 1980.

A survey of disability retirements (n = 1,071) under the Commonwealth Government superannuation scheme showed that the most common medical disabilities were anxiety/depressive neurosis (35 percent) and ischemic heart disease (21 percent). At the time of retirement, an adverse effect on their health was noted from alcohol in 11 percent and from cigarette smoking in 16 percent of the group. Cigarette consumption patterns were as follows: 62.4 percent were nonsmokers, 8.5 percent 1-10 cigarettes/day smokers, 14.5 percent 11-20 cigarettes/day smokers, 10.5 percent 21-40 cigarettes/day smokers, and 4.1 percent >40 cigarettes/day smokers. Alcohol consumption was as follows: none, 32.6 percent; occasional, 42.6 percent; most days, 13.4 percent; and everyday, 11.4 percent. (Auth. Abs. Mod.)

80–1668. McDonald, J. C.; Liddell, F.D.K.; Gibbs, G. W.; Eyssen, G. E.; McDonald, A. D. **Dust Exposure and Mortality in Chrysotile Mining, 1910-75.** *British Journal of Industrial Medicine* 37(1): 11-24, 1980.

A further followup of a birth cohort of 11,379 workers exposed to chrysotile is reported. The cohort consisted of all 10,939 men and 440 women, born 1891-1920, who had worked for at least a month in the mines and mills of Asbestos and Thetford Mines in Quebec. For all subjects, length of service and estimates of accumulated dust exposure were obtained, with a smoking history for the vast majority. Three methods of analysis, two based on the "man-years" method, the other a "case-and-multiple-controls" approach, gave results consistent with one another and with previous analyses. By the end of 1975, 4,463 men and 84 women had died. Among men, the overall excess mortality, 1926-1975, was 2 percent at Asbestos and 10 percent at Thetford Mines, much the dustier region. The women, mostly employed at Asbestos, had a standardized mortality ratio (SMR) (all causes, 1936-1975) of 0.90. Analysis of deaths 20 years or more after first employment showed that in men with short service (less than 5 years) there was no discernible correlation with dust exposure. Among men employed at least 20 years, there were clear excesses in those exposed to the heaviest dust concentrations. Reanalysis in terms of exposure to age 45 showed definite and consistent trends for SMRs for total mortality, for lung cancer, and for pneumoconiosis to be higher the heavier the exposure. The response to increasing dose was effectively linear for lung cancer and for pneumoconiosis. Lung cancer deaths occurred in nonsmokers, and showed a greater increase of incidence with increasing exposure than did lung cancer deaths in smokers, but there was insufficient evidence to distinguish between multiplicative and additive risk models. There were no excess deaths from laryngeal cancer, but a clear association with smoking (the SMR was 0.46 for nonsmokers compared to 4.85 for heavy smokers). Ten men and one woman died from pleural mesothelioma. If the subjects studied had been the 1,904 men with at least 20 years' employment in the lower dust concentrations, averaging 6.6 million particles per cubic foot (or about 20 fibers/cc), excess mortality would not have been considered statistically significant, except for pneumoconiosis. The inability of such a large epidemiological survey to detect increased risk at what, today, are considered unacceptable dust concentrations, and the consequent importance of exposure—response models are therefore emphasized. (Auth. Abs. Mod.)

MORTALITY AND MORBIDITY

80–1669. Sabroe, S.; Olsen, J. **Health Complaints and Work Conditions Among Lacquerers in the Danish Furniture Industry.** *Scandinavian Journal of Social Medicine* 7(3): 97-104, 1979.

In this study, morbidity rates were based upon 418 replies to postal inquiries sent to 478 members of the carpenters/cabinetmakers trade union in the Arhus area of Denmark. Lacquerers, when compared with former lacquerers and nonlacquerers, had an excess of neurophysiological symptoms (e.g., headache, dizziness, day-dreaming, tremor) and respiratory symptoms. Smokers constituted between 59.1 and 62.5 percent of the workers in each group. A significant association was found between lung symptoms and lacquering, smoking habits, and dust exposure ($p<0.001$, $p<0.001$, and $p<0.05$, respectively). The statistical association between exposure to lacquers and lung symptoms remained significant after correction for age, smoking habits, and exposure to dust. (Auth. Abs. Mod.)

See also, **80–1710, 80–1717, 80–1719, 80–1721, 80–1728, 80–1741**

NEOPLASTIC DISEASES

80–1670. Colbourne, M. J.; Ho, J. H-C. **Bronchial Cancer in Hong Kong, 1976-77: A Preliminary Report.** In: *Second Symposium on Epidemiology and Cancer Registries in the Pacific Basin.* National Cancer Institute Monograph 53, U.S. Department of Health, Education, and Welfare, NIH Publication No. 79-1864, November 1979, pp. 49-51.

A case—control study of the relationship of bronchial cancer to smoking was conducted during 1976-77 in Hong Kong, in which particular note was taken of the histologic type of the tumor. A total of 132 male and 111 female patients were interviewed. The association with smoking was more evident in males than it was in females, and squamous and small-cell type carcinomas, as a group, appeared to be more closely associated with smoking than did adenocarcinomas and large cell tumors. Of the women with bronchial carcinoma, 44 percent were nonsmokers; 79 percent of their tumors were adenocarcinomas or large cell carcinomas. For the women in this group, no association could be detected with place of residence, occupation, or use of kerosene for cooking. (Auth. Abs.)

80–1671. Foster, F. **New Zealand Cancer Registry Report.** In: *Second Symposium on Epidemiology and Cancer Registries in the Pacific Basin.* National Cancer Institute Monograph 53, U.S. Department of Health, Education, and Welfare, NIH Publication No. 79-1864, November 1979, pp. 77-80.

In 1976, a question regarding cigarette smoking was included in the New Zealand national census. Preliminary results from a 10 percent sample show that of the 2.2 million persons surveyed aged 15 years and over, 97.2 percent answered the question. Of each 100 males, 38 reported they had never smoked regularly, 22 were former smokers, and 40 were current smokers. In each 100 females, 57 had never smoked regularly, 12 were former smokers, and 31 were current smokers. The age and sex of all respondents, their cigarette smoking status, and the number of cigarettes smoked by current smokers on the day before the census were determined. A cigarette smoking question has now been incorporated in the New Zealand standard cancer notification form. Standardized rates for cancer of selected sites were determined for Maoris and non-Maoris. During the 25-year period from 1949 to 1973, the incidence of malignant melanoma of the skin in non-Maoris increased markedly. In females aged 45-54 and in males aged 55-64, the age-specific rates increased almost sixfold. The increases for other age groups, although substantial, have not been as large. Between 1958 and 1974, threefold increases in standardized incidence rates have been reported for cancer of the lung in Maori males and females and in non-Maori females, but for non-Maori males the rates have less than doubled. (Auth. Abs.)

80–1672. Hirohata, T.; Fukuda, K. **Incidence and Etiology of Lung Cancer in the Pacific Basin.** In: *Second Symposium on Epidemiology and Cancer Registries in the Pacific Basin.* National Cancer Institute Monograph 53, U.S. Department of Health, Education, and Welfare, NIH Publication No. 79-1864, November 1979, pp. 133-137.

Incidence of lung cancer in the Pacific Basin was either compiled from published reports or computed by the authors. The results showed a great variation in age-standardized annual incidence rates of lung cancer among 10 countries and 17 areas in the Pacific Basin where tumor registry statistics are available. For males, the incidence rates ranged from 10 to over 70 per 100,000 population, and for females from less than 5 to over 30 per 100,000 population. The reason(s) for the great variation is unclear. Ionizing radiation, carcinogenic chemical substances (e.g., chromium, arsenic compounds, asbestos, etc.), or air pollution is unlikely to be responsible. Because cigarette smoking is known to be a major cause of lung cancer, it is suggested that surveys on cigarette smoking be conducted among various populations in the Pacific Basin so that the etiologic significance of cigarette smoking can be assessed. In Hawaii such a survey is under way, and a preliminary analysis was made to examine the association between lung cancer and cigarette smoking among five races. The association between estimated risks for lung cancer and the observed incidence rates of the disease was not a clear straight-line relationship. On the whole, however, Caucasians and Hawaiians smoke more and have higher lung cancer incidence rates than do Orientals. It is believed that these results are compatible with the hypothesis that different levels of smoking among the five races surveyed are associated with their risks for lung cancer. Smoking patterns in New Zealand, based on a census survey, have also recently become available. Maoris of both sexes are indeed heavy tobacco users; for example, 68.5 percent of the Maori young women 20-24 years of age smoke. Women of all ages smoke twice as much as non-Maori women. Thus the unusually high rates of lung cancer for Maoris are probably a reflection of their heavy smoking habits. (Auth. Abs. Mod.)

80–1673. Lemoine, J. M. **Les Cancers Bronchogeniques. Leur Frequence chez les Hommes de Moins de 50 Ans. [Bronchogenic Cancers. Their Incidence in Men Below 50.]** *Broncho-Pneumologie* 29(5): 416-419, 1979, French.

The incidence of bronchogenic cancer in men of different ages was evaluated statistically, using bronchial biopsy data collected in the Paris metropolitan area during the first 3 years of each of the last 4 decades. The male population below age 50 was the target group of the evaluation because of the possible correlation of the increased cigarette consumption during the 1914-1918 years of the First World War with an increase in bronchogenic cancer incidence from 1943-1944. Statistical comparison of the bronchoscopic data shows that bronchogenic cancers were rare in men below 40. The volume of data for the first 3-year period (1941-1943) was insufficient for statistical comparisons. The highest incidence of bronchogenic cancer was in the 50-59 year age bracket and the incidence was even greater in men above 60. The cancer incidence in the 40-49 year bracket decreased from 7 and 10.5 percent in the first two 3-year periods, respectively, to 4.1 and 5.7 percent in the last two periods (61-63 and 71-73). Only six out of nine statistical comparisons by age bracket were significant, and only one out of possible nine comparisons between the three periods was significant. Therefore, the incidence of bronchogenic cancers in men below 50 in the Paris area did not increase in the last 3 decades. Most of the examined men started smoking at the age of 20. It is anticipated that in the next 1 or 2 decades bronchogenic cancer symptoms would appear 5 to 10 years earlier than at the present time because teenagers not only start smoking more frequently, but are expected to reach the cancer risk threshold of 150,000 cigarettes earlier.

80–1674. Lin, T. M.; Yang, C. S.; Tu, S. M.; Chen, C. J.; Kuo, K. C.; Hirayama, T. **Interaction of Factors Associated With Cancer of the Nasopharynx.** *Cancer* 44(4): 1419-1423, October 1979.

A retrospective study of nasopharyngeal carcinoma (NPC) revealed that smoking, working under poor ventilation, use of nasal balms or oil for nasal and throat troubles, use of herbal drugs, and anti-Epstein-Barr virus antibody titer were found statistically associated. The dual interactions of these factors to the risk of NPC were presented. Except in work conditions with poor ventilation and when herbal drugs were used, all the combinations were synergistic. The synergistic actions were especially remarkable with smoking and use of herbal drugs (relative risk = 3.63), and smoking and working under poor ventilation (relative risk = 3.29). The possible etiological mechanisms of NPC are discussed. (Auth. Abs. Mod.)

80–1675. McLemore, T. L. **Aryl Hydrocarbon Hydroxylase Induction in Human Lung Tissue, Pulmonary Macrophages, and Blood Lymphocytes: Effects of Cigarette Smoking.** Doctoral Dissertation, University of Texas Health Science Center, Houston, University Microfilms International 78-7209, June 1977, 235 pp.

Aryl hydrocarbon hydroxylase activity was fluorometrically measured in fresh or cultured pulmonary alveolar macrophages, cultured, mitogen-stimulated lymphocytes, and fresh, surgically-excised lung tissue from nonsmokers and cigarette smokers with and without lung cancer. There were 36 smokers and 11 nonsmokers among the lung cancer patients, and 36 smokers and 20 nonsmokers among the noncancer patients. When enzyme levels for fresh pulmonary macrophages and cultured lymphocytes from smokers and nonsmokers were compared, smokers' cells exhibited higher levels of aryl hydrocarbon hydroxylase activity regardless of whether lung cancer was present. Also, the in vitro induction of macrophage aryl hydrocarbon hydroxylase by benzanthracene was similar to the induction of the enzyme in situ in the lungs of cigarette smokers whether or not the patient had lung cancer. When fresh macrophage enzyme activity or cultured lymphocyte enzyme values were compared for noncancer and lung cancer patients, higher enzyme levels were observed when either type of cells were obtained from patients with lung cancer. It is stated that comparison of enzyme levels in different tissues of the same individual could be useful in lung cancer diagnosis, particularly when bronchoscopy is an inconclusive diagnostic procedure. (Auth. Abs. Mod.)

80–1676. Mitchell, D. M.; Morgan, P.G.M.; Ball, J. B. **Prognostic Features of Large Cell Anaplastic Carcinoma of the Bronchus.** *Thorax* 35(2): 118-122, February 1980.

The clinical features of 208 patients with large cell anaplastic carcinoma of the bronchus are reviewed. Of the sample, 47.6 percent had disseminated disease at presentation, and 95 percent were cigarette smokers. Of the smokers, 9.4 percent smoked <10 cigarettes/day, 17.4 percent smoked 10-20/day, 38.5 percent smoked 20-30/day, 13.9 percent smoked 30-40/day, and 20.8 percent smoked >40/day. The mean number of cigarettes smoked was 24.5/day. At diagnosis, 159 had not stopped smoking, 26 stopped smoking over 1 year before diagnosis, and 16 stopped smoking during the year before diagnosis. The median survival was 6.0 months and 5-year survival was 5.9 percent. Twenty-seven percent of the patients had surgical resection of the tumor. The median survival of resected patients was 13.0 months and 5-year survival was 21 percent. Radiotherapy was ineffective in controlling the disease in this series. (Auth. Abs. Mod.)

80–1677. Shanmugaratnam, K.; Tye, C. Y.; Goh, E. H.; Chia, K. B. **Etiological Factors in Nasopharyngeal Carcinoma: A Hospital-Based, Retrospective, Case—Control, Questionnaire Study.** In: de-The, G.; Ito, Y. (Editors). *Nasopharyngeal Carcinoma: Etiology and Control.* IARC Scientific Publications No. 20, Lyon, International Agency for Research on Cancer, 1978, pp. 199-212.

A total of 379 Singapore Chinese patients with nasopharyngeal carcinoma (NPC) were interviewed by use of a questionnaire including the following items: socioeconomic status; personal and family history of nasal illness; types of medicine used; use of Chinese medicine for the nose and throat; use of soya sauce, Chinese tea, cooling drinks, and alcohol; cigarette smoking (number and duration); cooking fuels; and use of incense (frequency and duration) and of anti-mosquito coils. The same questionnaire was given to two groups of controls: 595 patients with diseases of the ear, nose, and throat other than NPC (Ear, Nose, and Throat Department Controls (ENTC)) and 1,044 patients with diseases other than cancer or otorhinolaryngeal disease (Other Hospital Controls (OHC)). NPC patients differed significantly from both groups of controls in that they showed stronger associations with personal history of nasal illness, family history of nasal illness, use of Chinese medicine for the nose and throat, and exposure to smoke from anti-mosquito coils. Persons who had smoked cigarettes for ≥ 10 years had a higher risk for NPC when compared with ENTC ($p<0.001$), but there was no significant difference when compared with OHC. The pattern of distribution suggests, however, that the risk differential with ENTC, although highly significant, arose from the presence of more nonsmokers and fewer smokers of long duration in the ENTC group rather than from any positive relationship between NPC and cigarette smoking. (Auth. Abs. Mod.)

80–1678. Smith, E. M. **Epidemiology of Oral and Pharyngeal Cancers in the United States: Review of Recent Literature.** *Journal of the National Cancer Institute* 63(5): 1189-1198, November 1979.

See also, **80–1668**

Recent literature on morbidity and mortality patterns, known risk factors, and related sociobehavioral characteristics of oral cancer have been examined. Despite variation in populations and methodologies, alcohol and tobacco were seen as the major independent etiologic agents. One study found that the relative risks of oral cancer for light and heavy smokers as compared to nonsmokers were 4.4 and 5.6 respectively. Another study of tobacco users found the risk of pharyngeal cancer greatest in cigarette smokers. The effects of alcohol and tobacco were associated with age, sex, and religion-ethnicity. Other factors were suggested, but their correlation was less consistent: geographic location, race, socioeconomic status, nutrition, dental conditions, and concurrent diseases. Social and behavioral components may alter risk, stage of disease at diagnosis, treatment, or survival from oral cancer. The impact of sociobehavioral elements on the reduction of the incidence of, and the mortality from, the disease is an important area needing further investigation. (Auth. Abs. Mod.)

80–1679. Tirmarche, M.; Flamant, R. **Carcinomes Bronchiques Primitifs (Cancers du Poumon).Epidemiologie Dimensions du Probleme en France et a l'Etranger. [Primary Bronchial Carcinomas (Lung Cancers). Epidemiology Dimensions of the Problem in France and Abroad.]** *Revue Francaise des Maladies Respiratoires* 5(Supplement 2): 9-18, 1977, French.

During the last 25 years, epidemiological inquiries studying the relation between cigarette smoking and the risk of lung cancer have shown that this risk grows according to the number of cigarettes smoked, the duration of cigarette smoking, and the inhalation of the smoke. On the other hand, the stopping of cigarette smoking helps, to a certain extent, reduce the bronchial cancer risk. In England and in the United States, the mortality rate due to lung cancer in men less than 60 years old reached a plateau which could be explained by the appearance, 20 years ago, of filter cigarettes containing less tar and nicotine. Among the female population, the mortality rate due to bronchial carcinoma increased regularly. The experimental studies of the action of the smoke components from different types of cigarettes on the bronchial mucous membrane, the appearance of artificial tobacco substitutes, and the genetic studies which show populations particularly sensitive to polycyclic aromatic hydrocarbons, will bring new elements to the understanding of bronchial carcinogenesis in man. (Auth. Abs.)

80–1680. Agrawal, K. P.; Kumar, A. **Fall in Specific Airway Conductance at Residual Volume in Small Airway Obstruction.** *Respiration Physiology* 40(1): 65-78, April 1980.

A technique of body plethysmography has been developed for measuring specific airway conductance (SGaw) at residual volume (RV). Box pressure variations were recorded as the difference between chest volume change and air volume respired at atmospheric pressure $\Delta(V_1\text{-}V_2)$. They could be easily related to alveolar pressure change per liter of lung volume and SGaw could be directly calculated without breathing against a closed shutter. To eliminate temperature-humidity artifact, calculations were made near end-tidal points where air movement in and out of lungs is minimal. For that purpose airflow changes were related to box pressure changes over a period of 0.2 sec from the beginning of inspiratory effort. SGaw was measured at functional residual capacity as well as at RV. Excessive air trapping at RV in some cases resulted in some chest expansion unaccompanied by any airflow at mouth. This has been termed pre-flow work (PFW). Any abnormal fall in SGaw at RV or the presence of PFW could be used as an indicator of small airway obstruction. Using this method, obstruction in small airways was found in all smokers with smoking history of 5 to 30 pack years (mean 13.1 pack years) and in four patients who had a history of cough and/or dyspnea or exertion. Simultaneous measurements of SGaw at FRC and SGaw 0.2 sec and PFW at RV could be of great help in determining the degree and site of airflow obstruction. (Auth. Abs.)

80–1681. Akbarkhanzadeh, F. **Long-Term Effects of Welding Fumes Upon Respiratory Symptoms and Pulmonary Function.** *Journal of Occupational Medicine* 22(5): 337-341, May 1980.

To evaluate the long-term influence of welding fumes and of cigarette smoke on the function of the bronchopulmonary system, a comparative study of spirometric measurements in 209 welders and 109 nonwelder controls was performed in a shipyard. The two groups were matched for age, height, smoking habits, residence, and social class. Fifty-one percent of the welders had one or more of the respiratory symptoms, while only 26 percent of the controls had any of the symptoms. Chronic bronchitis was found to be confined to welders who smoked or had smoked. The welders appeared to show significantly increased impairment of lung function and, with advancing years, a deterioration in lung function greater than that of the controls. In general, however, welders did not show serious pulmonary insufficiency. (Auth. Abs.)

80–1682. Dosman, J. A.; Cotton, D. J.; Graham, B. L.; Li, K.Y.R.; Froh, F.; Barnett, G. D. **Chronic Bronchitis and Decreased Forced Expiratory Flow Rates in Lifetime Nonsmoking Grain Workers.** *American Review of Respiratory Disease* 121(1): 11-16, January 1980.

The purpose of this study was to determine whether clinical symptoms and pulmonary function abnormalities were more common in lifetime nonsmoking grain elevator workers exposed to grain dust than in lifetime nonsmoking community control subjects who were not occupationally exposed to dust. An occupational health questionnaire, spirometric variables, and flow-volume curves were obtained from 90 lifetime nonsmoking grain workers and 90 lifetime nonsmoking community control subjects. For these groups the prevalence of chronic bronchitis, defined as daily production of phlegm for 3 months/year for at least 2 years was calculated. The prevalence of chronic bronchitis was higher in nonsmoking workers (23.1 percent) than in nonsmoking control subjects (3.3 percent) ($p<0.01$). Nonsmoking grain workers had lower mean values than did nonsmoking control subjects for maximal mid-expiratory flow rate ($p<0.01$) and for maximal expiratory flow at 50 percent of vital capacity ($p<0.01$). It is concluded that exposure to grain dust in lifetime nonsmoking grain workers is associated with an increased prevalence of chronic bronchitis and evidence of airflow obstruction. (Auth. Abs.)

80–1683. Durda, M.; Ziemecka, E.; Szafranski, W. **Pollution Atmospherique Dans une Cimenterie, Habitude de Fumer le Tabac et Apparition de la Bronchite Chronique et de l'Emphyseme Pulmonaire. [Atmospheric Pollution in a Cement Plant, Tobacco Smoking Habit and Occurrence of Chronic Bronchitis and Pulmonary Emphysema.]** *Bulletin of the International Union Against Tuberculosis* 51: 671-674, 1976, French.

A sample of 2,027 workers (17-65 years of age) in a cement plant was examined for symptoms of nonspecific chronic respiratory diseases and was surveyed about their smoking habits by means of a questionnaire. The sample included patients with and without symptoms of chronic bronchitis but with symptoms of pulmonary emphysema. In a sample of 1,978 workers (about 80 percent male) who did not have any changes in the lung tissue, the chronic respiratory disease incidence increased with age for both sexes. These workers have been exposed to an average 250t/km²/year of cement dust. Among the nonsmoking men in this sample, the incidence of chronic bronchitis increased to 7.1 percent with an increase in the dust exposure index to over 200. The incidence of nonspecific chronic respiratory diseases at the identical dust exposure in slightly or moderately smoking men was 16.5 percent vs. 7.1 percent in nonsmoking men. Among all smokers this incidence was 3.1 percent for men and 2.1 percent for women, a statistically insignificant difference. Among women, symptoms were found in 2.1 percent of the nonsmokers and 8.8 percent of the regular smokers.

80–1684. Dutu, S.; Jienescu, Z.; Baicoianu, S. **Etude Epidemiologique de la Bronchite Chronique Dans une Population Agee de 60 a 85 Ans. Contributions a l'Etude de l'Histoire Naturelle de la Bronchite Chronique. [Epidemiological Study of Chronic Bronchitis in a Population Sample Aged 60-85 Years. Contributions to the Study of the Natural History of Chronic Bronchitis.]** *Respiration* 39(1): 49-59, 1980, French.

The spontaneous development of chronic bronchitis was studied in a population sample of elderly people, aged 60-85 years, and compared to a middle-aged population (40-59 years old) which was studied previously. The population sample in the present epidemiological study was comprised of 242 male smokers and 132 male nonsmokers, 49 female smokers and 328 female nonsmokers. Respiratory symptoms and smoking habits were determined from a questionnaire. Ventilatory function of the lungs was studied, using measurements of vital capacity (VC) and forced expiratory volume in 1 sec (FEV$_1$), which had been recorded over a 10-year period. The VC and FEV$_1$ values of these subjects were used for regression estimates of the VC and FEV$_1$ reference values for the age bracket studied. Chronic productive cough was determined in 30.6 percent of male smokers vs. 11.4 percent of male nonsmokers and in 28.6 percent of female smokers vs. 11.6 percent of female nonsmokers. Among those with chronic cough, 39.2 percent of the male smokers had normal spirographic values vs. 66.7 percent of the male nonsmokers. The respective percentages of female smokers and nonsmokers were 57.1 and 73.7. The spirographic values characteristic of obstructive ventilatory failure were present in only 40.5 percent of the male smokers and in smaller percentages in the other subject categories. The normal and obstructive spirographic values of male smokers in the 40-59 year bracket differed significantly ($p<0.001$) from the corresponding values of male smokers in the 60-85 year bracket. In the total population sample, obstructive disorders were more frequent in male smokers (24.4 percent) than in nonsmokers (6.1 percent) or in female smokers (8.2 percent) and nonsmokers (2.1 percent). Frequency of obstructive syndrome increased with the age of male smokers.

80–1685. Fainstein, V.; Musher, D. M. **Bacterial Adherence to Pharyngeal Cells in Smokers, Nonsmokers, and Chronic Bronchitics.** *Infection and Immunity* 26(1): 178-182, October 1979.

Selective adherence to host mucosal surfaces is probably a requirement for colonization and infection by bacteria. Since pharyngeal colonization may be an important determinant in the pathogenesis of pneumonia, the adherence of 10 different bacteria to pharyngeal cells obtained from nonsmokers, smokers, and chronic bronchitics was studied. Smokers were defined as those who smoked ≥ 40 cigarettes/day currently, and for at least 3 years, but who were otherwise healthy and asymptomatic. Various patterns of adherence among the different groups of subjects were found. Adherence of *Staphylococcus pneumoniae* type I was strikingly greater for smokers than for nonsmokers (21.0 ± 3.1 in smokers vs. 1.4 ± 1.2 in nonsmokers, $p<0.001$). Adherence of *S. pneumoniae* type III and *S. aureus* was increased in smokers, albeit to a lesser degree (3.2 ± 1.5 vs. 1.3 ± 1.2, $p<0.01$; 3.0 ± 2.8 vs. 1.2 ± 1.0, $p<0.05$; respectively). There was less adherence of *Klebsiella* and *Pseudomonas* to pharyngeal cells of smokers than of nonsmokers ($p<0.01$). Middle-aged smokers with a long history of chronic bronchitis had significantly increased ($p<0.001$) adherence only of nontypable *Haemophilus influenzae* when compared with age-matched nonsmokers. The acquisition of pneumococcal pneumonia by smokers and the role of nontypable *Haemophilus* species in chronic bronchitis may be determined, in part, by bacterial adherence to pharyngeal cells. (Auth. Abs. Mod.)

80–1686. Federspiel, C. F.; Layne, J. T.; Auer, C.; Bruce, J. **Lung Function Among Employees of a Copper Mine Smelter: Lack of Effect of Chronic Sulfur Dioxide Exposure.** *Journal of Occupational Medicine* 22(7): 438-444, July 1980.

Lung function among 599 white male employees of a southeastern Tennessee copper mine/smelter operation was compared according to smoking history and occupational experience. Subjects included never-smokers, ex-smokers, and current smokers. For smokers, data were converted to an index of pack-years of exposure. Analyses of variance for the average lung function indices of those workers who had never smoked, those who formerly smoked, and those who currently smoked revealed statistically significant differences among mean values for percent predicted FEV$_1$ ($p<0.001$) but not for FVC, strongly suggesting a deleterious effect of smoking on timed expiratory volume. The rates of subjects with evidence of obstructive impairment were 18.2, 11.4, and 8.5 percent among current smokers, former smokers, and never-smokers, respectively. The prevalence rates of bronchitis vary significantly according to smoking habits ($p<0.001$), revealing bronchitis among 23.0, 9.6, and 5.5 percent of current smokers, former smokers, and never-smokers, respectively. The job categories compared included employees with work histories in low sulfur dioxide (SO_2) environments (both underground mining and nonmining), in high SO_2 exposure work areas, and in those with transient exposure to SO_2. Miners with low SO_2 exposure were found to have lower lung function indices (both forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV$_1$)) than did employees in other job categories. After adjusting for smoking history, cumulative long-term exposure to SO_2 was not demonstrated to contribute to decreased lung function. (Auth. Abs. Mod.)

80–1687. Higenbottam, T.; Feyeraband, C.; Clark, T.J.H. **Cigarette Smoke Inhalation and the Acute Airway Response.** *Thorax* 35(4): 246-254, April 1980.

The acute airway response to smoking varying numbers (1-4) of identical cigarettes in rapid succession and smoking single cigarettes of differing tar (low to high)/nicotine (0.70 to 3.39 mg) yields was assessed repeatedly in 13 healthy smokers. The airway response was variable, indicating airway narrowing consistently in only three subjects. There appeared no difference between forced spirometry and measurement of airway resistance in detecting the airway response. No relationship was observed between the airway response and amount of smoke inhaled into the lungs as measured either by changes in venous blood nicotine or percentage carboxyhemoglobin. When five smokers inhaled smoke directly from a cigarette, acute airway narrowing was consistently observed. A normal smoking pattern consisting of an initial drag of smoke into the mouth, followed after a pause by inhalation of smoke diluted with air, did not consistently cause airway narrowing, although similar amounts of smoke as the direct drag were inhaled as assessed by changes in venous blood nicotine. The manner of smoke inhalation affects the relative concentrations of the different constituents of smoke reaching the lungs and also appears to be the main determinant of the acute airway response to smoking, which was unrelated to the number of cigarettes smoked or the tar content of the smoke. This suggests that patterns of smoke inhalation may influence the pathogenesis of bronchial disease associated with smoking. (Auth. Abs.)

80-1688. MacArthur, C.G.C.; Hunter, D.; Gibson, G. J. **Ventilatory Function in the Eisenmenger Syndrome.** *Thorax* 34(3): 348-353, June 1979.

Ventilatory function and carbon monoxide transfer factor were studied in nine adult patients (i.e., five men, of whom two were smokers, and four women) with posttricuspid intracardiac defects and the Eisenmenger syndrome. A consistent mild defect of ventilatory function was found, with raised residual volume and closing capacity and reduction of other lung volumes and maximal expiratory flows. Maximal expiratory flow was particularly reduced at low lung volumes. One of the four subjects in whom pressure-volume studies were performed showed loss of normal elastic recoil at low lung volumes. A significant poorly ventilated space was excluded since the volume of distribution of helium during a 10-sec breath-hold at full inspiration was close to plethysmographically measured total lung capacity. Carbon monoxide transfer factor, after correction for hemoglobin concentration, was 77 percent of predicted normal values. Sequential assessment of carbon monoxide transfer factor might be useful in the early diagnosis of pulmonary vascular disease in infants with large intracardiac defects, since it is likely to be raised early due to high pulmonary blood flow and will fall with the development of pulmonary vascular disease. (Auth. Abs.)

80-1689. Musk, A. W.; Bevan, C.; Campbell, M. J.; Cotes, J. E. **Factors Contributing to the Clinical Grade of Breathlessness in Coalworkers With Pneumoconiosis.** *Bulletin Europeen de Physiopathologie Respiratoire* 15(2): 343-355, 1979.

Factors underlying the capacity for exercise have been investigated in 125 coal miners with pneumoconiosis first investigated in 1969 (mean age 52 years) and followed up in 1978. Investigations included the British Medical Research Council questionnaire on respiratory symptoms, anthropometry, spirometry, measurements of transfer factor, lung mechanics, and the physiological response to exercise. Smoking was scored 1, 2, 3, and 4 for nonsmokers, ex-smokers, and smokers of less or more than 14 cigarettes/day, respectively. Subjects smoked a little less in 1978 than in 1969 (smoking scores of 2.29 vs. 2.41, respectively). Multiple regression and discriminant function techniques were used to describe the clinical grade of breathlessness and the symptom-limited maximal oxygen uptake in 1978, and the change in clinical grade of breathlessness between 1969 and 1978 in terms of the other variables. Approximately 40 percent of the variance in the symptom-limited maximum oxygen uptake and in the clinical grade of breathlessness could be described by the anthropometric, spirometric, and gas transfer variables. Measurements of pulmonary compliance, conductance, and recoil pressures did not add significantly to the predictive value of the pulmonary function tests. A deterioration in breathlessness in previously healthy subjects was presaged by a relatively low symptom-limited maximum oxygen uptake, high exercise ventilation, low forced expiratory volume in 1 sec, and relative to the forced expiratory volume a high airways conductance, the latter combination possibly reflecting narrowing of the small lung airways. An increase in breathlessness was associated with a gain in body mass and increase in the questionnaire score for bronchitis. Those whose breathlessness had increased were then heavier, had a lower transfer coefficient and forced vital capacity, and had a higher volume of isoflow but similar specific conductance, closing volume and bronchitis score compared with those whose breathlessness remained the same or was reduced. These findings may be of use in designing means of reducing the progression of exercise-related symptoms in coal miners with pneumoconiosis. (Auth. Abs. Mod.)

80-1690. National Institute for Occupational Safety and Health. **Health Hazard Evaluation Determination Report 77-42-4522. Cleaning House at the Wire Mill, Bethlehem Steel Corporation, Johnstown, Pennsylvania.** U.S. Department of Commerce, National Technical Information Service, PB-278 061, December 1977, 22 pp.

A Health Hazard Evaluation investigation was conducted by the National Institute for Occupational Safety and Health on February 9-10, and June 22-23, 1977, in the Wire Cleaning House of the Bethlehem Steel Corporation in Johnstown, Pennsylvania. The survey was prompted by a request from an authorized employee representative concerning exposure to toxic chemicals and alleging a high incidence of respiratory disorders among the employees. Exposure of employees to hydrochloric acid and sulfuric acid exceeded accepted exposure limits, while exposure to carbon monoxide potentially exceeded accepted limits during the use of gasoline-powered equipment. Medical interviews elicited symptomatology suggesting chronic respiratory disease in most of the crane operators and picklers, but its incidence correlated more closely with smoking history than with occupational exposure to acid mists and/or gases. There was a high correlation between positive smoking history and respiratory complaints. Of the 13 individuals sampled, 8 reported smoking cigarettes, the average duration being 28.7 pack-years. One individual smoked cigars, approximately four/day, for about 10 years. All of the smokers complained of significant cough except for one individual who complained of only occasional cough. Most of the coughs were productive of significant quantities of sputum. No nonsmokers in either group complained of a significant cough, but two nonsmokers did complain of slight shortness of breath while on the job. One-third of the cranemen and picklers described premature tooth erosion and loss, while 44 percent of the workers in this group reported skin problems probably related to skin contact with the materials in use. Recommendations are made for reducing worker exposures. (Auth. Abs. Mod.)

80–1691. Peat, J. K.; Woolcock, A. J.; Leeder, S. R.; Blackburn, C.R.B. **Asthma and Bronchitis in Sydney Schoolchildren. II. The Effect of Social Factors and Smoking on Prevalence.** *American Journal of Epidemiology* 111(6): 728-735, 1980.

During a study of the prevalence of asthma and bronchitis in Sydney schoolchildren from 1971 to 1976, some social and environmental factors were documented to ascertain if these affected the prevalence of either of these diseases. Socioeconomic status obtained from father's occupation, area of residence, family size, nationality, country of birth, and smoking habits were examined. Although small differences were found between groups, no consistent relationship was found between social class and lung disease with the exception of increased prevalence of asthma in boys in social class I and girls in social class II. There were no major associations with area of residence or with family size. Children who were born outside Australia tended to have less asthma and bronchitis than children born inside Australia, and migrant children tended to first develop asthma several years after arriving in Australia. Bronchitis was found to be more common in children with an earlier history of bronchitis in infancy and early childhood. The prevalence of recent bronchitis in the group of children who smoked 20 or more cigarettes per week was higher than that in the nonsmoking group of children (those who smoked less than one cigarette a week). (Auth. Abs. Mod.)

80–1692. Pratt, P. C.; Vollmer, R. T.; Miller, J. A. **Epidemiology of Pulmonary Lesions in Nontextile and Cotton Textile Workers: A Retrospective Autopsy Analysis.** *Archives of Environmental Health* 35(3): 133-138, May-June 1980.

A collection of 565 unselected inflation-fixed lungs was divided into three groups: (1) normal (209 lungs), (2) centrilobular emphysema (231 lungs), and (3) "other" (125 lungs), the last including examples of fibrosis, tuberculosis, cancer, and other forms of emphysema. Clinical hospital records were reviewed to ascertain smoking history (no smoking (105 lungs), >0.5 pack cigarettes/day (427 lungs), or pipe/cigar (33 lungs)) and occupation (nontextile (521 lungs) or textile (44 lungs)). Lungs were subjected to morphometric determination of the extent of centrilobular emphysema, mucus gland hyperplasia in large bronchi, and goblet cell metaplasia in bronchioles. Extent of tissue pigmentation in normal lungs was also measured. Associations between morphologic data and background factors were examined by covariance analysis. As in many previous studies, data show highly significant cigarette smoking effects on all factors measured ($p<0.05$ to 0.0001). Significant pipe smoker effects were also found ($p<0.05$). When the cigarette group was excluded, a significant association was found between cotton dust exposure and both mucus gland hyperplasia ($p=0.02$) and goblet cell metaplasia ($p=0.03$), but not emphysema. The results suggest that centrilobular emphysema is not associated occupationally in the textile industry, although bronchitis and bronchiolitis probably are. If byssinotic symptoms and physiologic impairment are as prevalent as some have reported, they must be primarily related to airway lesions. It might follow that they should be reversible. Textile workers with irreversible impairment and morphologic emphysema who are also smokers probably have little or no justification for attributing this to their occupation. (Auth. Abs.)

80–1693. Rawbone, R. G.; Adams, L.; Robinson, M.; Lonsdale, D.; Jenkins, A.; MacRae, K.; Guz, A. **A Four Year Longitudinal Study of Smoking Habits and Respiratory Function in a Cohort of Children Aged Twelve to Fifteen Years.** *Bulletin Europeen de Physiopathologie Respiratoire* 16(1): 9, January-February 1980.

Data on smoking, respiratory symptoms, and knowledge and attitude to health were obtained from 10,498 British secondary school children in March 1975. Of these, 405 entered a 4-year longitudinal study which will be completed in April 1979. Pulmonary function tests were performed and prevalence of smoking was assessed. As predicted from analysis of an age-matched population defined from the 1975 cross-sectional study, the prevalence of smoking during the 4 years increased from 3.5 to 26.4 percent. Analyses of respiratory parameters are at a preliminary stage. Subgroups have been defined on the basis of smoking and respiratory symptoms, and differences between these groups will be tested by different methods of analysis.

80–1694. Rivero, O. **Etude sur la Physiologie Comparee des Fumeurs et des Non Fumeurs. [Comparative Physiology of Smokers and Nonsmokers.]** *Bulletin of the International Union Against Tuberculosis* 51: 565-567, 1976, French.

The association of the principal respiratory diseases with the smoking habits of the Mexican population has been studied, based on official statistics and the author's research data. According to statistics, the average annual consumption of cigarettes in Mexico is 22 packs/person and is decreasing while cigarette production is increasing. Mortality from bronchogenic cancer at present is 3.6 per 100,000 compared to 1.7 per 100,000 in 1954. The mortality from chronic bronchitis was 16 per 100,000 in 1971. In 1974, in a single hospital, obstructive emphysema was found in 15.2 percent of 970 autopsies, while in 1957 9.1 percent of 155 autopsies revealed this disease. The research completed thus far consisted of spirometric determination of the respiratory function parameters in smoking and nonsmoking in 18- to 24-year-old medical students, selected in Mexico City and Oaxaca City, the former community having a high level of atmospheric pollution and the latter having no pollution. Preliminary data show respiratory obstruction of the bronchioles in young smokers. In the next phase of research, similar, but, older (38-44 years) groups of subjects from the two cities will be tested and the results will be compared with those from the younger group.

80–1695. Rossiter, C. E.; Heath, J. R.; Harries, P. G. **Royal Naval Dockyards Asbestosis Research Project: Nine-Year Follow-Up Study of Men Exposed to Asbestos in Devonport Dockyard.** *Journal of the Royal Society of Medicine* 73(5): 337-344, May 1980.

Progression of asbestos-related disease was assessed in a group of 253 dockyard workers examined in 1966 and 1975. Despite the almost complete protection from exposure to asbestos since 1966, radiographic parenchymal abnormalities increased and occurred more frequently during the next 9 years in those men who had been more heavily exposed to asbestos. Lung function values were lower in those most heavily exposed and were declining at a faster rate than in those with less dust exposure. The most sensitive lung function index was the transfer factor. Analysis of the lung function patterns in relation to occupation group, smoking habit, presence in 1966 of persistent crackles and chest wheezes, and to radiographic appearance in 1966, showed that the transfer factor of the lung differentiated most sharply between the occupation groups and also related strongly to smoking habit and to the presence of persistent crackles. Progression of disease was greater for smokers than for nonsmokers, with those who gave up smoking between 1966 and 1975 suffering the greatest changes. This latter group showed most increase in small opacities and included almost all new cases of diffuse pleural changes. They also showed the greatest declines in forced expiratory volume and forced vital capacity. (Auth. Abs. Mod.)

80–1696. Rubino, G. F.; Newhouse, M.; Murray, R.; Scansetti, G.; Piolatto, G.; Aresini, G. **Radiologic Changes After Cessation of Exposure Among Chrysotile Asbestos Miners in Italy.** *Annals of the New York Academy of Sciences* 330: 157-161, 1979.

A preliminary investigation has been conducted with available data from a morbidity study of chrysotile asbestos miners in northern Italy in order to determine whether asbestosis appeared or progressed after cessation of exposure. The men under investigation were retired workers for whom chest X-ray films at exposure termination were obtained. There were 56 men among 265 respondents who satisfied the following study criteria: >3 years since cessation of asbestos exposure, a film available ±1 year after exposure ceased, and no radiologic change due to other pulmonary conditions (e.g., tuberculosis, tumor, trauma). The mean interval between the two films for each subject was approximately 12 years. The rate of irregular opacities was somewhat higher at cessation of exposure in smokers ($n=41$) than in nonsmokers ($n=15$). There were 16 vs. 2 ILO/UC category $\geq 1/0$, respectively, first film opacities. There was no evidence that smoking either influenced the appearance or progression of irregular opacities after cessation of exposure.

80–1697. Shephard, R. J.; Collins, R.; Silverman, F. **"Passive" Exposure of Asthmatic Subjects to Cigarette Smoke.** *Environmental Research* 20(2): 392-402, 1979.

Fourteen asthmatic subjects volunteered for a controlled trial of 2-hour passive exposure to cigarette smoke. Seven cigarettes were burnt in a closed room of 14.6 m^3 capacity, producing a carbon monoxide concentration 24 ppm above ambient and a suspended particulate concentration of 2-4 mg/m^3. Most symptomatic responses were as in normal individuals, but there were more complaints of wheezing (36 percent) and tightness in the chest (43 percent). Changes of pulmonary function were slight. Relative to the corresponding control exposure, there was a small decrease of total lung capacity as assessed by helium mixing ($p<0.02$), possibly indicating an effect of the smoke on small airways. There was also some evidence of arousal and/or emotional excitement, including a slight tachycardia (at 80-min exposure, $p<0.05$) and a slight increase of forced vital capacity ($p<0.05$ at 90-min exposure). However, dynamic lung volumes ($FEV_{1.0}$, $V_{max\ 50\ percent\ VC}$, $V_{max\ 25\ percent\ VC}$) were unaltered. Examining separately the four subjects who claimed sensitivity to cigarette smoke, the only significant difference from the remaining asthmatic individuals was a greater $FEV_{1.0}$ relative to the corresponding time in the control exposure. The data thus do not suggest that asthmatic subjects have an unusual sensitivity to cigarette smoke. (Auth. Abs.)

80–1698. Sprince, N. L.; Kanarek, D. J.; Weber, A. L.; Chamberlin, R. I.; Kazemi, H. **Reversible Respiratory Disease in Beryllium Workers.** *American Review of Respiratory Disease* 117(6): 1011-1017, June 1978.

A medical and environmental survey was carried out in a beryllium extraction and processing plant in 1971, and a followup study was done in 1974. Peak air concentrations of beryllium of as much as 50 times the accepted peak limit value were found in the plant in 1971. Of 111 workers who had not changed their smoking status (55 smokers, 36 ex-smokers, and 20 nonsmokers) between 1971 and 1974, radiographic changes of interstitial disease were found in 31 workers, hypoxemia was found in 20, and a combination of hypoxemia and radiographic changes of interstitial disease was seen in 11 workers. The followup in 1974 showed that air concentrations had all decreased significantly, mainly because of improvements in engineering and ventilation in the plant. Improvement in hypoxemia and decreased alveolar-arterial O_2 tension difference at rest were noted in 13 of the 20 men who had hypoxemia in 1971 and were available for followup. In addition, radiographic abnormalities of interstitial disease were reversed in some workers. No significant change in mean forced expiratory volume in 1 sec or forced vital capacity for the group as a whole and for each smoking category, comparing 1971 to 1974, was seen. Mean peak expiratory flow rate decreased slightly for smokers, ex-smokers, and the total group, but did not change significantly for nonsmokers. Mean partial pressure of oxygen in arterial blood increased slightly, but significantly, for each smoking category (smokers, $p<0.01$; ex-smokers, $p<0.01$; nonsmokers, $p<0.05$) and for the group as a whole ($p<0.01$). Mean partial pressure of carbon dioxide in arterial blood decreased significantly ($p<0.01$) for smokers, ex-smokers, and the group as a whole. The survey results suggest that improvement in gas exchange and radiographic findings of interstitial disease occurs when peak air concentrations of beryllium are decreased. (Auth. Abs. Mod.)

80–1699. Tamura, M.; Washizaki, M.; Kanemitsu, T. **Kitsuen to Shin hai Kino. 2. Kido Teiko. [Effects of Cigarette Smoke on Airway Resistance.]** *Japanese Journal of Thoracic Diseases* 15(9): 558-564, September 1977, Japanese.

The effects of cigarette smoking on airway response were studied in normal adults. The ratio of the forced expiratory volume in 1 sec to forced vital capacity and the maximum mid-expiratory flow rate were measured in 175 normal adults. In smokers of over 30 pack-years a significant drop in FEV_1/FVC was detected. Airway resistance (Raw) and specific airway conductance (SGaw) were measured by using a constant pressure whole body plethysmograph in 55 volunteer subjects. There was no substantial difference between nonsmokers and smokers in Raw or SGaw. To investigate the immediate effects of cigarette smoke on the large airways, SGaw was measured 3 times in 19 healthy adults, namely before smoking, and 5- and 30-min after smoking one cigarette. SGaw fell significantly 5 min after smoking one unfiltered cigarette in 7 out of 12 smokers, whereas filtered cigarettes caused no significant fall in SGaw. This experiment strongly suggests that the response to smoking can be reduced, though not abolished, by filtering out the particulate phase of the smoke. To evaluate the role of β-adrenergic input to the bronchial smooth muscles in the occurrence of large airway obstruction due to cigarette smoke, two healthy smokers received 30 mg of propranolol in advance and 90 min later SGaw was measured before and after smoking one cigarette. The same procedures were repeated using a placebo. Smoking after propranolol administration caused a more pronounced drop in SGaw than smoking after placebo administration. These findings suggest that propranolol may render the muscles more sensitive to the contractile effect of cigarette smoke. Raw and SGaw were measured in 6 patients with bronchial asthma before and 5 min after smoking a herbal cigarette. It is confirmed that the herbal cigarette smoking is significantly beneficial to Raw and SGaw. (Auth. Abs.)

80–1700. Uragoda, C. G. **Respiratory Disease in Tea Workers in Sri Lanka.** *Thorax* 35(2): 114-117, February 1980.

Blending of different grades of tea is a very dusty process which exposes workers to a fine fluff. One hundred and twenty-five (108 men, 17 women) tea blenders with an average service of 22.9 years in the industry were interviewed according to a standard questionnaire and submitted to a clinical and radiographical examination. The examination was repeated 8 and 31 months later. A total of 46 workers (36 percent) had respiratory illness, 31 had chronic bronchitis (24.8 percent), 8 had asthma (6.4 percent), and 7 had active or inactive pulmonary tuberculosis (5.6 percent). The prevalence of chronic bronchitis and asthma was more than that expected in the general population. The workers with chronic bronchitis smoked an average of 5.7 cigarettes/day (S.D. 6.1), while the average for the study population was 4.7 cigarettes/day (S.D. 4.9). There were 9 nonsmoking workers with chronic bronchitis, while there were 29 nonsmoking workers in the study population. None of the women were smokers. It is suggested that chronic bronchitis and asthma are etiologically related to long-term exposure to tea fluff. (Auth. Abs. Mod.)

80–1701. Woolcock, A. J.; Leeder, S. R.; Peat, J. K.; Blackburn, C.R.B. **The Influence of Lower Respiratory Illness in Infancy and Childhood and Subsequent Cigarette Smoking on Lung Function in Sydney Schoolchildren.** *American Review of Respiratory Disease* 120(1): 5-14, July 1979.

A prospective study of respiratory illness history and lung function of 10,898 schoolchildren in Sydney was begun in 1971. At the first visit, a history of previous asthma and of bronchitis before and after the first 2 years of life was obtained from a parental questionnaire, and maximal expiratory flow-volume curves were recorded. These lung function studies were repeated yearly between 1972 and 1974, at which time a history of respiratory illness (e.g., bronchitis, asthma) during the previous 12 months and a personal smoking history were recorded. Two groups of children from a random selection of primary and secondary schools in Sydney were studied. Their respective mean ages were 8.9 years (primary group) and 12.6 years (high school group) in 1971. Mean values for the maximal flow at 50 percent of the forced vital capacity ($Vmax_{50}$) were lower in children with a history of bronchitis and/or asthma than those in the control group. This was true in both age groups in both sexes. No differences were found in the 0.5-sec forced expiratory volume or Vmax. The differences in $Vmax_{50}$ were present again in 1974. In 1974, the data from smokers were compared with those from nonsmokers; small differences were found. Analysis of the data showed that children smoking ≤ 1 cigarette/week were slightly older than nonsmokers. There was no overall relationship between smoking and lung function, although high school boys who had had bronchitis and who smoked had significantly lower values for Vmax_{50} and Vmax than those who did not, differences that were not accounted for by differences in age and height. There were no differences between the corresponding groups of girls. In both cohorts, girls with previous respiratory illness manifested no consequences of smoking on measurements of lung function, but girls without a history of respiratory illness who smoked had slightly lower values for Vmax_{50} and Vmax than their nonsmoking contemporaries. The results suggest that bronchitis in infancy and childhood as well as asthma may affect lung function as children grow, that the abnormality may not be detected by the forced expiratory volume, that the abnormality persists, and that it is possible that the abnormality is further affected by smoking. (Auth. Abs. Mod.)

80–1702. Zaborski, W.; Pryjma, K.; Heczko, P.; Przybylkiewicz, Z. **Wplyw Palenia Tytoniu Oraz Zanieczyszczen Atmosferycznych na Czestosc Wystepowania Haemophilus Influenzae w Jamie Gardlowej. [Effect of Tobacco Smoking and Air Pollution on the Incidence of Haemophilus Influenzae in the Pharyngeal Cavity.]** *Przeglad Lekarski* 36(2): 271-274, 1979, Polish.

A bacteriologic study of pharyngeal smears was performed in 155 male workers in a basic phosphate slag (Thomas slag) plant. The incidence of *H. influenzae* was determined in workers exposed to different degrees of air pollution (dust, fluoride dust, and gaseous fluoride). The bacterium was found in the respiratory tract of 70.9 percent of the workers and was significantly more frequent in those with symptoms of respiratory tract inflammation. *H. influenzae* was isolated from the respiratory tract of 78.0 percent of the 105 smokers compared to 56.0 percent of the 50 nonsmokers ($p<0.05$). This difference was particularly evident within the group of workers without inflammation (70.3 percent in smokers vs. 29.4 in nonsmokers). In contrast, the incidence of *H. influenzae* in workers exposed to different degrees of industrial pollution was nearly identical. The study suggests that tobacco smoking is an additional risk factor contributing to respiratory infection with *H. influenzae*. The associated effects of smoking and air pollution may be important in the development of chronic airway disease.

See also, **80–1631, 80–1669**

CARDIOVASCULAR DISEASES

80–1703. Benfari, R. C.; McIntyre, K.; Eaker, E.; Blumberg, S.; Paul, O. **The Psychological Effects of Differential Treatment of a High Risk Sample in a Randomized Clinical Trial.** *American Journal of Public Health* 69(10): 996-1000, October 1979.

A study was carried out using 616 participants in a randomized clinical trial at the Harvard MRFIT (Multiple Risk Factor Intervention Trial) Clinical Center, to test if there were differences in the psychological dimensions of anxiety, depression, and functional heart symptoms in groups given different levels of treatment in a coronary heart disease intervention program. The program was designed to achieve and maintain a lower serum cholesterol, a reduction in blood pressure, and cessation of cigarette smoking (64 percent of the subjects were smokers). A theoretical framework was given to justify a number of hypotheses as to the induction of adverse psychological effects. At the end of 2 years in the MRFIT Program there were no significant differences between the special intervention group and the usual care group in the selected psychological variables. (Auth. Abs. Mod.)

80–1704. Bengtsson, C.; Lindquist, O. **Coronary Heart Disease During the Menopause.** In: Oliver, M. F. (Editor). *Coronary Heart Disease in Young Women.* Edinburgh, Churchill Livingstone, 1978, pp. 234-242.

Results from previous studies and investigations indicate that there is an increased incidence of myocardial infarction in women with early menopause. An attempt has been made to find out whether such an increased incidence can be ascribed to the menopause per se or to differences between women with early and women with late menopause in risk factors for myocardial infarction. It was found that blood lipids were higher in women with early menopause than in women of the same age with late menopause. A tendency to the opposite direction was found for blood pressure. Smoking was more common in women with early menopause. The number of smokers was 44/175 in premenopausal and 84/173 in postmenopausal 50-year-old women. There was a tendency toward heavier smoking among the postmenopausal smoking women: 21/84 postmenopausal smokers (25 percent) consumed ≥ 15 cigarettes/day compared to 8/44 premenopausal smokers (18 percent). This difference was, however, not statistically significant. The number of women with myocardial infarction who had their menopause at or earlier than 45 years included 10/46 (22 percent) for the total group and 8/37 for smokers only (22 percent). As smoking is an important risk factor for myocardial infarction in women, it appears that the increased number of smokers among women with early menopause can explain part of the increased incidence of myocardial infarction in women with early menopause.

80-1705. Blumenthal, J. A.; Williams, R. B., Jr.; Kong, Y.; Schanberg, S. M.; Thompson, L. W. **Type A Behavior Pattern and Coronary Atherosclerosis.** *Circulation* 58(4): 634-639, October 1978.

Previous research has demonstrated an increased rate of clinical coronary heart disease (CHD) events among people who exhibit a "coronary prone" (Type A) behavior pattern. This study was undertaken to determine whether the association between behavior pattern Type A and CHD might be extended beyond clinical CHD events to include also the coronary atherosclerotic process. In addition to usual clinical evaluation, 156 consecutive patients referred for diagnostic coronary angiography were independently assessed on the basis of a structured interview and assigned a rating of Type A, Type B, or Type X (indeterminate). Traditional physiologic factors (i.e., age, sex, cholesterol, and cigarette smoking) were found to correlate with atherosclerotic disease. When past or present cigarette smokers were considered together, as compared to those who had never smoked cigarettes, smokers were found in significantly greater proportions in the moderate and severe coronary occlusion groups than in the mild coronary occlusion group (74 vs. 50 vs. 42 percent, respectively, $p<0.02$). Type A patients were found in increasing proportions among groups of patients with coronary occlusions of moderate to severe degree compared with patients with only mild occlusions (69 vs. 93 vs. 44 percent, respectively, $p<0.001$). This increasing proportion of Type A patients with increasing disease severity remained significant, even when age, sex, blood pressure, serum cholesterol level, and cigarette smoking history were all simultaneously covaried. These findings suggest that, independently of traditional risk factors, behavior pattern Type A may contribute to the risk of clinical CHD events via effects on the atherosclerotic process. (Auth. Abs. Mod.)

80-1706. Brooks, S. H.; Blankenhorn, D. H.; Chin, H. P.; Sanmarco, M. E.; Hanashiro, P. K.; Selzer, R. H.; Selvester, R. H. **Design of Human Atherosclerosis Studies by Serial Angiography.** *Journal of Chronic Diseases* 33(6): 347-357, 1980.

Atherosclerosis has been studied by femoral angiography in men with myocardial infarction. Smoking history recorded at entry was the strongest variable associated with degree of atherosclerosis at the first angiogram ($p<0.01$). Fifty-four men had two angiograms; 28 of these men had one additional angiogram. Two variables recorded in the interval between angiograms were significantly correlated with atherosclerosis change in one femoral segment; α-lipoprotein level determined by electrophoresis (a negative correlation, $p<0.01$) and maximum systolic blood pressure occurring during an exercise tolerance test (a positive correlation, $p<0.01$). An estimate of angiographic atherosclerosis assessment error has been obtained and used to calculate sample size for future clinical trials which employ this endpoint. Testing the effect of therapy on lesion change with angiography requires fewer study subjects observed for a shorter period than is necessary to test a commensurate effect on atherosclerosis-related morbidity or mortality rates. For example, an angiographic study with design features comparable to those of the Coronary Drug Project could require a 1-year observation period and 132 subjects per test group. (Auth. Abs.)

80-1707. Bucknall, T. E.; Bowker, T.; Leaper, D. J. **Does Increased Movement Protect Smokers From Postoperative Deep Vein Thrombosis?** *British Medical Journal* 280(6212): 447, February 16, 1980.

In an investigation of a reason why smokers have a decreased incidence of postoperative deep venous thrombosis, it was postulated that increased movement (e.g., for a surreptitious cigarette) was responsible. This theory was tested 24 hours preoperatively and 4 days postoperatively in 40 patients undergoing inguinal herniorrhaphy. The patients were given a pedometer, and divided into smokers and nonsmokers. The median age of smokers was 47 years, whereas for nonsmokers, it was 54.5 years ($p<0.025$). Smokers smoked an average 22 cigarettes/day. For the 24 hours preoperatively, the median distance moved by the smokers vs. nonsmokers was 2.01 vs. 1.93 km, respectively. Corresponding 4th day figures were 3.06 vs. 2.41 km. These differences were not significant.

80-1708. Butts, N. K.; Golding, L. A. **Effect of 24 Hours of Smoking Withdrawal on Cardiorespiratory Functions at Rest and Exercise.** *Journal of Sports Medicine* 19(4): 389-396, 1979.

The purpose of this study was to assess the physiological responses of chronic smokers to a 24-hour period of no smoking. Twenty-eight smokers (9 women and 19 men) who averaged over a pack of cigarettes per day for at least 8 years were evaluated on various physiological functions during rest and exercise before and after cessation of smoking. Results indicated that no significant changes occurred in red blood cell counts, spirometry measures, resting blood pressure, or oxygen consumption. Significant decreases ($p<0.05$) occurred in hemoglobin concentrations, hematocrits, diastolic blood pressure during exercise, and heart rates during rest, exercise, and recovery. Furthermore exercise oxygen pulse was significantly increased ($p<0.05$). It was concluded that chronic smokers who cease smoking for a 24-hour period demonstrate significant improvements in cardiac efficiency at rest and during submaximal exercise. (Auth. Abs.)

80-1709. Criqui, M. H.; Barrett-Connor, E.; Holdbrook, M. J.; Austin, M.; Turner, J. D. **Clustering of Cardiovascular Disease Risk Factors.** *Preventive Medicine* 9(4): 525-533, July 1980.

In a population-based cardiovascular disease study the associations among five risk factors—cholesterol, triglycerides, blood pressure, obesity, and cigarette smoking—were analyzed. Two methods were used: standard correlation analysis, and a percentile analysis method limited to associations at higher levels of these risk factors. The study population, 4,839 men and women aged 30-89, showed significantly positive standard and age-adjusted correlation coefficients for all comparisons between any two risk factors except for those comparisons involving smoking. In the percentile analysis, subjects with moderate or greater ($\geq$70th percentile) or high ($\geq$90th percentile) levels on one of these risk factors showed clustering of elevations in other risk factors in that observed/expected ratios were generally greater than unity, again excepting smoking comparisons. Clustering was strongest in subjects at the highest levels of these risk factors. Because of the particularly high risk of subjects with several risk factors, this finding should caution clinicians who identify a patient with an elevation of one of these risk factors to evaluate the patient carefully for elevations of other risk factors. (Auth. Abs. Mod.)

80–1710. Deubner, D. C.; Wilkinson, W. E.; Helms, M. J.; Tyroler, H. A.; Hames, C. G. **Logistic Model Estimation of Death Attributable to Risk Factors for Cardiovascular Disease in Evans County, Georgia.** *American Journal of Epidemiology* 112(1): 135-143, July 1980.

Linear logistic analysis of the relationship of cardiovascular disease risk factors to an overall measure of health effect, 10-year mortality, revealed significant associations of death with systolic blood pressure, age, sex, diabetes mellitus, smoking, cholesterol, obesity (Quetelet index), race, and social index. Attributable risk and population attributable risk estimates were derived from the model by changing actual variable values to target values. The results confirmed systolic hypertension and smoking as major public health problems and diabetes mellitus as a powerful risk factor for death. In addition, both the attributable risk due to smoking and smoking prevalence (proportion of population in exposed group) are higher in males (white males, 4.2, 0.654; black males, 4.7, 0.689) than in females (white females, 2.8, 0.168; black females, 3.1, 0.162). This results in markedly elevated population attributable risks and population attributable fractions in males (white males, 2.7, 0.126; black males, 3.2, 0.123) compared to females (white females, 0.5, 0.039; black females, 0.5, 0.032). The potential overall effect of eliminating smoking from this population is a 9.1 percent reduction in the overall death rate. The small detrimental effect of cholesterol on probability of death limits the potential for an overall beneficial effect of preventive intervention. In fact, drug intervention in two reported trials of cholesterol reduction had negative overall effects. Demonstration of the association of a characteristic with a specific disease state does not alone justify attempts to eliminate the high risk state from a population. An overall detrimental health effect must be documented by suitable studies before trials of preventive intervention are undertaken or recommendations made to the public. (Auth. Abs. Mod.)

80–1711. Friedman, G. D. **Cigarette Smoking and Coronary Heart Disease: New Evidence and Old Reactions.** *American Heart Journal* 99(3): 398-399, March 1980.

The relationship between smoking and coronary heart disease (CHD) is sometimes attributed to predisposing constitutional and genetic factors already present in smokers. Some research supports the hypothesis that smoking does not induce clinical CHD. In a study of the relationship of cigarette smoking to total and CHD mortality, 4,004 middle-aged smokers, ex-smokers, and nonsmokers were followed for a period of 11 years. An association between smoking and CHD was not established. However, the smokers' total death rate was 2.1 times higher than the nonsmokers', and the adjusted smoker/nonsmoker ratio for CHD was 3.6. Results of this study (which was funded by the tobacco industry), and of another on the relation of alcohol to cardiovascular disease (which was supported by the U.S. Brewers' Association), suggest that reliance should be placed on the integrity and objectivity of researchers and the results not judged by the source of the research support. Research funding, regardless of source, is necessary for determining the role, if any, of smoking in the pathogenesis of CHD.

80–1712. Hennekens, C. H.; Lown, B.; Rosner, B.; Grufferman, S.; Dalen, J. **Ventricular Premature Beats and Coronary Risk Factors.** *American Journal of Epidemiology* 112(1): 93-99, July 1980.

Of 10,119 men (3,046 smokers, 6,801 nonsmokers) aged 35-57 years studied in the Multiple Risk Factor Intervention Trial (MRFIT), ventricular premature beats (VPBs) were more frequent among the 112 men who reported having previously had a myocardial infarction (22.3 percent), as well as among the 143 men with a history of diabetes mellitus (13.3 percent) than among the remaining 9,864 (7.6 percent). There was no demonstrable association between VPBs and cholesterol or blood pressure levels, either overall or in various age groups. There was, however, a strongly positive relationship between VPBs and age ($p<0.001$). A positive association between VPBs and smoking was present which was statistically significant overall (risk ratio = 1.21, 95 percent confidence limits from 1.03 to 1.43), as well as in the age groups 35-39 years (1.74, 1.13 to 2.68) and 55-57 years (1.50, 1.01 to 2.22). This association, if confirmed, raises the question of whether VPBs offer a mechanism for the increased morbidity and mortality from coronary heart disease among cigarette smokers compared with nonsmokers. (Auth. Abs.)

80–1713. Hollnagel, H.; McCartney, P. **Relationship of Fasting and Post-Load Serum Glucose to Prevalence of ECG Abnormalities in Forty-Year-Old Men: From the Glostrup**

Population Studies. *Journal of Chronic Diseases* 32(11/12): 709-719, 1979.

In a prevalence study in a general population in Glostrup, Copenhagen, 504 men, age 40, were examined (response rate of 88 percent). Resting and post-exercise electrocardiogram (ECG) abnormalities were examined in relation to serum glucose measured fasting and 1, 2, and 2 1/2 hours after an oral glucose load of 1 g glucose/kilogram bodyweight. A significant positive correlation was found between all four serum glucose variables and body mass index, systolic and diastolic blood pressures, and serum uric acid. No consistent relationship between glucose variables and resting ECG abnormalities was found. For post-exercise ECG abnormalities, all glucose variables showed a positive association both in the univariate analyses and in the multivariate analyses and the 1-hour post-load glucose values were significantly correlated with the prevalence of post-exercise ECG abnormalities independent of other risk factors for coronary heart disease, such as systolic blood pressure, cholesterol, body mass index, and cigarette smoking. (Auth. Abs.)

80–1714. Holme, I.; Helgeland, A.; Hjermann, I.; Leren, P.; Lund-Larsen, P. G. **Four and Two-Thirds Years Incidence of Coronary Heart Disease in Middle-Aged Men: The Oslo Study.** *American Journal of Epidemiology* 112(1): 149-160, July 1980.

The association between 4 2/3 years incidence of coronary heart disease (CHD) and serum cholesterol, blood pressure (BP), height, body weight, cigarette smoking, and physical activity at work and at leisure was studied in 14,816 Oslo men who were 40-49 years of age at entry to the study and who were free of cardiovascular disease and diabetes. A weak dose—response relationship is revealed among cigarette smokers; a 50 percent higher risk exists between those smoking 25 cigarettes/day compared with those smoking 1-4 cigarettes/day. Never- and ex-cigarette smokers have about 40 percent of the risk of cigarette smokers. Serum cholesterol is a stronger predictor for CHD than the other continuous factors studied in this cohort. The predictive power was only moderately increased by combining cholesterol, systolic BP, and cigarette smoking. The risk ratio between men physically active and sedentary at leisure was 1:3. Only 35 percent of this difference could be explained by the other risk factors. The epidemiology of CHD in Oslo in relation to serum cholesterol and BP was found to be unchanged from that found in a previous study in Oslo. (Auth. Abs. Mod.)

80–1715. Horowitz, M. J.; Hulley, S.; Alvarez, W.; Reynolds, A.-M.; Benfari, R.; Blair, S.; Borhani, N.; Simon, N. **Life Events, Risk Factors, and Coronary Disease.** *Psychosomatics* 20(9): 586-592, September 1979.

To investigate whether high levels of stress from life events are correlated with increased levels of smoking, blood pressure, and cholesterol, a life events questionnaire was used to determine the level of presumptive stress in a sample of 575 middle-aged men who were initially selected for inclusion in the larger, collaborative Multiple Risk Factor Intervention Trial. There was a significant negative correlation between number of cigarettes smoked (a factor in the selection formula) and diastolic blood pressure ($r=-.26$, $p<0.001$). Cholesterol and diastolic blood pressure were negatively correlated in both smokers and nonsmokers. Significant ($p<0.01$) differences in stress levels were found only between smokers ($n=288$) and nonsmokers ($n=287$). An independent t-test on the presumptive stress score as a function of smoking behavior showed smokers reporting more frequent and/or more serious life events than nonsmokers ($t=3.9$, $p<0.0001$). It is suggested that smoking behavior be included as a variable in studies of the relationship between psychological stress and illness. (Auth. Abs. Mod.)

80–1716. Jonsson, B. G.; Astrand, I. **Physical Work Capacity in Men and Women Aged 18 to 65.** *Scandinavian Journal of Social Medicine* 7(3): 131-142, 1979.

A probability sample ($n=1,050$) of the population of a region in central Sweden performed a submaximal exercise test on bicycle ergometers. The participants were men and women aged 18-65 taking part in the REBUS study 1969-71. In multivariate analyses, heart rate at the rate of work 100 W and 50 W was found to correlate significantly with sex, body weight and stature, blood pressure at rest, physical training in leisure time, and physical strain at work. No significant partial correlation was found between heart rate at work and alcohol consumption. Smoking was found to correlate significantly ($r=0.07$) with heart rate at the rate of work 50 W. A linear function with heart rate at work on the Y-axis and rate of work on the X-axis was estimated for each subject. It was found that the mean intercept of these functions grew smaller with age and that women had a steeper slope than men. Subjects of the same sex had approximately the same slope, irrespective of age. It must be an implication of this finding concerning the relation between age and heart rate at work that in research work the use of W_{170} as an indicator of physical work capacity will introduce a systematic deviation when comparing older subjects with younger ones. An overestimation of the capacity of the older subjects will result. (Auth. Abs. Mod.)

80–1717. Keys, A. **Seven Countries. A Multivariate Analysis of Death and Coronary Heart Disease.** Cambridge, Massachusetts, Harvard University Press, 1980, 391 pp.

The findings of cross-cultural test of the predictive value of age, blood pressure, weight, smoking habits, exercise, and serum cholesterol for mortality and coronary disease in 16 cohorts (including Italy, Japan, Finland, Greece, Yugoslavia, South Africa, and the United States) are reported. The incidence of death (all causes or coronary) was roughly three times greater for men who smoked ≥ 20 cigarettes/day than for nonsmokers. In Finland and in the Netherlands, the

best copy available

relationship between smoking habits and 10-year incidence was similar except that the former smokers had about twice the death rate of the men who had never smoked. In Yugoslavia, the death rate of heavy smokers was twice that of men who never smoked and clearly greater than the rates of the rest of the men, but ex-smokers did not differ significantly from men smoking ≤ 19 cigarettes/day. In Italy and Greece, cigarette smoking could not be shown to be a very important risk factor for 10-year death, although the mortality of the heavy smokers was 34 percent higher than that of the men who had never smoked. In Japan the 10-year death rate was unrelated to smoking habits. These analyses of the relationships of smoking habits to 10-year incidence of death or disease take no account of the possibility that differences in the type of tobacco, curing method, or the manner of smoking are important. Until recently in southern Europe, only a small fraction of the cigarettes smoked were of the flue-cured Virginia type, different varieties of *Nicotiana tabacum* being dominant and sun curing the rule. Also, cigarettes are commonly smoked to a shorter length and are often allowed to stop burning to be relit from time to time. The latter custom is made possible by the absence of additives that keep the tobacco burning once it is lit. There are, it appears, important questions about the kind of tobacco, curing method, additives used, and the way the cigarettes are smoked that need careful study in connection with the long-term effects of smoking.

80-1718. Krueger, D. E.; Ellenberg, S. S.; Bloom, S.; Calkins, B. M.; Maliza, C.; Nolan, D. C.; Phillips, R.; Rios, J. C.; Rosin, I.; Shekelle, R. B.; Spector, K. M.; Stadel, B. V.; Stolley, P. D.; Terris, M. **Fatal Myocardial Infarction and the Role of Oral Contraceptives.** *American Journal of Epidemiology* 111(6): 655-674, June 1980.

The association between use of oral contraceptives (OC) and death from myocardial infarction (MI) in young women was investigated in a collaborative case—control study conducted in the five largest metropolitan areas in the United States. Potential cases were identified from computer tapes of the National Center for Health Statistics. Controls were selected from among women who died from, or were hospitalized for, acute conditions other than heart disease or from accidents, and were matched to cases on age, geographic area, and year of death. Information on cause of death, personal characteristics, OC use, and the presence of conditions predisposing to MI (e.g., smoking) was obtained from interviews with relatives and abstracts of hospital, clinic, and physician records. Odds ratios as approximations to relative risks for fatal MI in relation to OC use, and the associated 95 percent confidence limits, were calculated overall and for subgroups determined by demographic and health history characteristics. Odds ratios in the total study population were not significantly different from one. However, odds ratios significantly different from one were found when attention was restricted to white subjects without contraindications to OC use, and including only those cases whose deaths were attributed to MI with the greatest degree of certainty. Smoking was found to be a significant risk factor for fatal MI. The odds ratio for all smokers (cases, $n=37$; controls, $n=103$) as compared with all nonsmokers (cases, $n=7$; controls, $n=93$) was 1.97. (Auth. Abs. Mod.)

80-1719. McEwan, J. **Smoking, Age and the Pill.** *International Journal of Gynaecology and Obstetrics* 16(6): 529-534, 1979.

A brief outline is given of alterations in normal physiology caused by cigarette smoking, aging, and taking combined oral contraceptive pills. There are similarities in some of these effects. Evidence is considered regarding the health risks (e.g., thrombosis, embolism, myocardial infarct) of using the pill and the relationship of these risks to the age of the woman and her smoking habit. This evidence is derived from recent epidemiologic studies carried out in Britain on some aspects of morbidity and the death rates ascribable to the pill. In one oral contraception study, death rates per 100,000 woman-years from circulatory disease were significantly higher in smokers than in controls ($p<0.01$). The implications for prescribing clinicians are discussed and indications are given for reducing the overall risks to life and health for pill users. (Auth. Abs. Mod.)

80-1720. Mikhail, M.; El-Gohary, M.; Ayad, W.; Mahfouz, M.; El-Sawy, M. **Effect of Tobacco Smoking on Blood Glucose, Free Fatty Acids, Fibrinolytic Activity and Blood Coagulation Before and After Beta Adrenergic Receptor Blockade.** *Journal of Drug Research* 10(1/2): 1-11, 1978.

Twenty-eight healthy nonsmoking male volunteers (average age 38 years) were selected in order to study the acute changes in blood coagulation, fibrinolytic activity, plasma free fatty acids, platelet adhesiveness, and blood glucose induced by tobacco smoking. Blood sugar and blood fibrinolytic activity showed no statistically significant change after smoking. Yet pindolol (beta adrenergic receptor blocker) alone caused a significant decrease of blood fibrinolytic activity as evidenced by an increase of the euglobulin clot lysis time. Plasma free fatty acids as well as platelet adhesiveness showed a significant increase ($p<0.05$ and $p<0.001$, respectively), while the cephalin kaolin (K.C.) clotting time decreased significantly following smoking. Pindolol was found to prevent the significant change on plasma free fatty acids and K.C. clotting time. The changes already induced by smoking on platelet adhesiveness were found to be reversed by pindolol. (Auth. Abs. Mod.)

80-1721. National Heart, Lung, and Blood Institute. **Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, NIH Publication No. 79-1610, May 1979, 471 pp.

A conference to discuss the decline in the U.S. coronary heart disease (CHD) mortality rate was convened October 24-

25, 1978, by the National Heart, Lung, and Blood Institute in Bethesda, Maryland. The purposes of the conference were (1) to consider whether the greater than 20 percent decline in coronary heart disease mortality since 1968 is real, (2) to discuss possible causes for this decline, and (3) to recommend further studies to elucidate these causes. One of four conference sessions was concerned with trends in CHD risk factors such as smoking, hypertension, and diet. Data on smoking habits and mortality rates from the American Cancer Society study, the study of British Physicians, the Pooling Project, the Framingham Study, and the Veterans Administration study are among those reviewed and evaluated. Although the percentage of smokers and the amount of tar and nicotine in cigarettes have dropped in the country since the release of the first Surgeon General's report in 1964, the frequency of individuals smoking two or more packs of cigarettes has not changed, according to at least one survey. In addition, women, who have had the greatest decline in mortality, have increased their smoking levels. Nonetheless, changes in coronary heart disease care, blood pressure, blood cholesterol, and smoking habit may help explain much of the decline in CHD mortality in middle-aged men. Future strategies for quantifying and studying CHD trends are proposed.

80–1722. Noppa, H. **Body Weight Change in Relation to Incidence of Ischemic Heart Disease and Change in Risk Factors for Ischemic Heart Disease.** *American Journal of Epidemiology* 111(6): 693-704, June 1980.

A representative population sample of middle-aged women was studied in 1968-1969 and restudied in 1974-1975. A total of 1,302 women participated in both studies (80.3 percent of the initial sample). A statistically significant correlation was found between weight gain and the incidences of angina pectoris and arterial hypertension ($p<0.05$). A statistically significant correlation was also found between cessation of smoking and weight gain ($p<0.001$). Weight loss was found to be correlated to commencement of smoking ($p<0.001$). Regression analysis revealed statistically significant changes of systolic and diastolic blood pressures, fasting blood glucose, serum cholesterol, serum triglycerides, and serum uric acid with changes in body weight. (Auth. Abs. Mod.)

80–1723. Oleynikov, S. P.; Glazunov, I. S.; Chazova, L. V.; Lisitsin, V. Yu.; Shishova, A. M.; Balavadze, M. B.; Dobordzhginidze, L. M.; Podolskaya, O. V. **Tobacco Smoking and Prevention of Ischaemic Heart Disease.** *Cor et Vasa* 21(2): 81-89, 1979.

The tobacco smoking habits of 210 men aged 40-59 years, inhabitants of the territory of one therapeutic section of the District Polyclinic No. 78 in Moscow, are reported. The study was carried out in a research program of multifactorial prevention of ischemic heart disease (IHD), checking the presence of arterial hypertension, tobacco smoking, hypercholesterolemia, obesity, and low physical activity as risk factors. The population was examined for the presence of IHD and intermittent claudication. There were 42.6 percent among the population who were smokers. In the age category 40-49 years, the percentage of smokers (51.2 percent) was higher than in the age category 50-59 years (38.8 percent). The distribution of IHD was practically equal among the smokers (18.6 percent), nonsmokers (18.5 percent), and ex-smokers (22.0 percent). None of the nonsmokers had ever experienced myocardial infarction or intermittent claudication ($p<0.05$). Among the nonsmokers, elevated cholesterol and triglyceride levels were significantly ($p<0.05$) more frequent; the Quetelet index and percentage of overweight were also significantly higher in this group. Among the smokers, the proportion of chronically diseased patients was significantly higher. The antismoking program as a component of multifactorial prevention of IHD is briefly described. (Auth. Abs.)

80–1724. Oliver, M. F. **Clinical Characteristics and Prognosis of Angina and Myocardial Infarction in Young Women.** In: Oliver, M. F. (Editor). *Coronary Heart Disease in Young Women.* Edinburgh, Churchill Livingstone, 1978, pp. 221-233.

The basis of this report is 150 women who developed symptoms of coronary heart disease (CHD) under the age of 45. There were 81 who presented with myocardial infarction and 64 with angina; there was a definite nonatherosclerotic cause for the premature onset of CHD in the remaining 5 patients. Hypercholesterolemia, hypertension, or excessive cigarette smoking each occurred in a large minority, and more than one of these major risk factors was present in most patients. Excessive cigarette smoking was more common in women with myocardial infarction than in those with angina (47 vs. 26 percent, respectively, smoked ≥ 15 cigarettes/day). The latter did not differ in their cigarette smoking habits from the normal population. A premature menopause had occurred in 20 percent of these women, but there was no relation between the early onset of CHD with age of menarche, parity, or the incidence of abortion. Oral contraceptives increased the risk of myocardial infarction most when one of the major risk factors was also present. Altogether, 75 percent of patients with angina or myocardial infarction survived 12 years. Coexisting hypertension worsened the prognosis. The prognosis after myocardial infarction was similar in these women to that previously described for men under the age of 40. (Auth. Abs. Mod.)

80–1725. Richardson, C. **Regional Project for the Prevention of Coronary Heart Disease.** *Journal of Human Nutrition* 32(5): 373-374, 1978.

In 1976, for the first time in the Bath Health District, Wiltshire, a pilot coronary heart disease prevention program was established. The program was composed of four 10-session health education courses whose emphasis was on diet, smoking, and exercise. Local male residents aged 30-60 whose level of exercise had declined were invited to attend. Course members were urged to stop cigarette smoking as its ill effects on the heart had been long established. Exercise was reintroduced. A survey of the 63 men who completed the courses held from fall 1976 to spring 1977 showed that of 12 individuals who smoked at the beginning of the course, 4 had quit at the end of the course, and 7 had reduced the amount they smoked.

80–1726. Rose, G.; Heller, R. F.; Pedoe, H. T.; Christie, D.G.S. **Heart Disease Prevention Project: A Randomised Controlled Trial in Industry.** *British Medical Journal* 280(6216): 747-751, March 15, 1980.

Twenty-four factories or other occupational groups, employing 18,210 men aged 40 to 59, were formed into matched pairs. One of each pair was allocated randomly to receive a 5- to 6-year program of medical examinations and intervention to reduce the levels of the main coronary risk factors. Men at factories in the intervention group were given advice on dietary reduction of plasma cholesterol concentrations, stopping or reducing cigarette smoking, regular exercise for the sedentary, and reduced energy intake for the overweight; and hypertension was treated. The program was delivered mainly through existing occupational medical services, helped by a small central staff. Personal consultations were largely confined to men with a high risk of developing coronary heart disease. Changes in risk factors were assessed by regular standardized examinations of random samples of men. The spread of information by general propaganda proved easy, but a change in habits seemed to require personal contact. Small but significant reductions occurred, mainly in the high-risk group, but these were not sustained when pressure was relaxed. With reference to cigarette smoking, high-risk men reported a decline in mean daily cigarette consumption of 29 percent at the end of the first year, and this effect was sustained. At the time of the final examination 12 percent of those who were smokers at entry had stopped completely. As to the remainder of the study population, reported cigarette consumption was substantially reduced by intervention throughout the trial: at the final examination consumption had decreased by 16 percent. About 7 percent of smokers had by then been persuaded to stop. (Auth. Abs. Mod.)

80–1727. Rosenberg, L.; Slone, D.; Shapiro, S.; Kaufman, D. W.; Stolley, P. D.; Miettinen, O. S. **Coffee Drinking and Myocardial Infarction in Young Women.** *American Journal of Epidemiology* 111(6): 675-681, June 1980.

The effect of consumption of caffeine-containing coffee on the risk of myocardial infarction (MI) in women 30-49 years of age was investigated. In this study 487 patients with first infarctions were compared with 980 controls whose admissions were for acute emergencies. Overall and in various subgroups, coffee drinking and myocardial infarction were weakly and not significantly associated: the overall estimated relative risk for women drinking at least five cups daily, compared with women drinking none, was 1.4 after control for all identified potential confounding factors, such as age, smoking history, overweight, abnormal blood lipids, oral contraceptives history, and diabetes mellitus (95 percent confidence interval (CI), 1.0-1.9). The relation of MI and coffee drinking was similar within categories of cigarette smoking: there was no consistent trend of increasing relative risk with increasing consumption of coffee. Contrasting those who drank at least five cups daily with those drinking none, the overall estimated relative risk for smokers and nonsmokers was 1.2 (95 percent CI, 0.8-1.7) and 1.1 (95 percent CI, 0.5-2.3), respectively. The frequency of coffee drinking was greater among controls, whose admissions were for acute emergencies, than among other patients without myocardial infarction, most of whom had been admitted for chronic conditions. If patients of the latter type tend to avoid coffee, then their inclusion in the control series of previous hospital-based studies may have led to overestimation of the magnitude of the association between coffee and myocardial infarction. (Auth. Abs. Mod.)

80–1728. Salonen, J. T. **Stopping Smoking and Long-Term Mortality After Acute Myocardial Infarction.** *British Heart Journal* 43(4): 463-469, April 1980.

A prospective followup study was carried out to investigate the relation between smoking and risk of death after an acute myocardial infarction. The study consisted of male patients under the age of 65 years, who had had an acute myocardial infarction between 1972 and 1975 in North Karelia, Finland. Of these patients, 888 survived the first 6 months after the acute infarction and were followed up for 3 years after the infarction. It was found that the cumulative all-causes mortality rate of the patients who were still smoking 6 months after the acute myocardial infarction was 1.7 times that of the patients who had stopped smoking within the first 6 months. There was a dose—response relation between the number of cigarettes smoked daily and the mortality. The impact of smoking was greatest in the subgroups of patients with an otherwise good prognosis. It was estimated that 28 percent of the deaths in the whole group of initial smokers was attributable to continuing smoking after the infarction. On the basis of these findings it is suggested that antismoking advice should be an important part of the modern comprehensive care of patients with an acute myocardial infarction. (Auth. Abs.)

80–1729. Schroll, M.; Hagerup, L. **Relationship of Fasting Blood Glucose to Prevalence of ECG Abnormalities and 10 Yr Risk of Mortality From Cardiovascular Diseases in Men Born in 1914: From the Glostrup Population Studies.** *Journal of Chronic Diseases* 32(11/12): 699-707, 1979.

A total population of 50-year-olds in Glostrup, Denmark, was invited to a health examination in 1964. Approximately 85 percent of those eligible were examined. Fasting blood glucose levels obtained on 388 men were analyzed for possible relationship of asymptomatic hyperglycemia to prevalence of electrocardiogram (ECG) abnormalities at baseline. No positive association between baseline glucose level and ECG abnormalities was found, either in univariate analysis or in multiple logistic analysis. In the multivariate analyses, only systolic pressure showed a significant independent positive association with prevalence of ECG abnormalities, as defined by one set of criteria (London Whitehall Study). While heavy tobacco use was also positively associated with the ECG endpoints, the difference in tobacco consumption between those with and without ECG findings was not statistically significant. No differences were found in cholesterol in the cases and noncases in this cross-sectional analysis. A significant positive association was found between blood glucose and blood pressure, resting heart rate, serum triglycerides, and body mass index. Serum insulin was associated with diastolic pressure, body mass index, and triglycerides. While these associations were statistically significant, they were not of a high order. In a 10-year followup, baseline glucose level was not found to be predictive of mortality or of combined fatal and nonfatal cardiovascular events. The 46 men who died did not differ significantly from the survivors in initial glucose level, nor did those with cardiovascular events (44) or myocardial infarctions (29) differ on this variable from men without such events. The only statistically significant differences between those with and without events were in systolic pressure and heavy use of tobacco. Even though clinical observation has found that symptomatic diabetes mellitus carries a higher risk of cardiovascular disease (CVD), asymptomatic hyperglycemia—as recorded in fasting blood glucose—was not associated in this study with a higher incidence of cardiovascular disease. (Auth. Abs.)

80–1730. Stamler, R.; Stamler, J.; Lindberg, H. A.; Marquardt, J.; Berkson, D. M.; Paul, O.; Lepper, M.; Dyer, A.; Stevens, E. **Asymptomatic Hyperglycemia and Coronary Heart Disease in Middle-Aged Men in Two Employed Populations in Chicago.** *Journal of Chronic Diseases* 32(11/12): 805-815, 1979.

Studies of possible association between asymptomatic hyperglycemia and coronary disease were conducted among middle-aged employed men in Chicago. In one cohort, frequency of electrocardiogram (ECG) abnormalities at baseline was examined among men at different glucose levels. In univariate analysis, those at the highest glucose levels had higher rates of ECG evidence of coronary heart disease (CHD), but this association did not remain when such factors as age, blood pressure, weight, cholesterol, and cigarettes were taken into account in multivariate analysis. Among the studies, long-term followup to mortality (10, 13, and 15 years) showed inconsistent findings on association of hyperglycemia and subsequent death. In one cohort (Gas Company men followed for 10 years after a 1-hour post-load glucose determination) a positive glucose-mortality association was found in both univariate and multivariate analysis. But in the two remaining cohorts (Gas Company men 13 years after casual glucose determination and Western Electric men 15 years after 2-hour post-load determination), no such association was found. Cigarette use was not found to be associated with glucose level. While differences in method and sample size as well as other limitations may have contributed to the differences observed, the inconsistency of the findings does not permit a conclusion that asymptomatic hyperglycemia is a positive coronary risk factor. (Auth. Abs.)

80–1731. Stamler, R.; Stamler, J.; Schoenberger, J. A.; Shekelle, R. B.; Collette, P.; Shekelle, S.; Dyer, A.; Garside, D.; Wannamaker, J. **Relationship of Glucose Tolerance to Prevalence of ECG Abnormalities and to 5-Year Mortality from Cardiovascular Disease: Findings of the Chicago Heart Association Detection Project in Industry.** *Journal of Chronic Diseases* 32(11/12): 817-828, 1979.

A cohort of 7,841 middle-aged men employed in Chicago industry was studied to determine the significance of symptomatic hyperglycemia for coronary disease (CHD). Most univariate analyses indicated that men at the upper end of the glucose distribution had a higher rate of electrocardiographic (ECG) abnormalities. Those in the upper 2 1/2 percent of the glucose distribution were seen to have ECG abnormality rates considerably above those in the lowest 2 1/2 percent, as well as above those of the group as a whole. In bivariate analysis, among those who never smoked, hyperglycemia was associated with a higher risk of ECG abnormalities, but in smokers and ex-smokers (both of whom had the highest rates of such abnormalities), glucose level was not associated with an additional excess. Subsequent risk of mortality associated with initial glucose level was also assessed in a 5-year followup study. In bivariate analysis, when such additional variables as diastolic or systolic pressure, or cigarette smoking were taken into account, no association was seen between glucose level and cardiovascular (CV) or CHD mortality. A slight excess in mortality from all causes was observed in the upper glucose quintile when diastolic pressure above the median was also present; the same excess for CV and CHD mortality was observed in the nonsmoking subgroup, in the upper end of the glucose distribution. When such variables as age, systolic pressure, serum cholesterol, body mass index, and smoking status were taken into consideration in multiple logistic analyses, glucose level was not associated with CHD or CV death, even though it was associated with mortality from all causes. Such major coronary risk factors as blood pressure, serum cholesterol, and cigarette smoking were strongly associated with all mortality endpoints in the multiple logistic analyses. Previous assumptions that elevated glucose level in itself is a major coronary or cardiovascular risk factor do not receive support from the data in this study.

See also, **80–1649, 80–1732, 80–1749, 80–1784**

PREGNANCY AND INFANT HEALTH

80–1732. Asmussen, I. **Fetal Cardiovascular System as Influenced by Maternal Smoking.** *Clinical Cardiology* 2(4): 246-256, August 1979.

The umbilical artery, umbilical vein, and vessels of the placental villi were studied in 28 clinically normal women (15 nonsmokers and 13 heavy smokers). In the smokers group, the basement membrane of the umbilical artery was considerably widened and of a continuous type. In the umbilical vein, a remarkable decrease was found in the collagen content of the media and adventitia, and the myocytes seemed to have accumulated large amounts of glycogen. These alterations within the fetal cardiovascular system were never found in the children of nonsmoking mothers.

80–1733. Harrison, K. L. **The Effect of Maternal Smoking on Neonatal Leucocytes.** *Australian and New Zealand Journal of Obstetrics and Gynaecology* 19(3): 166-168, 1979.

This study was performed to investigate whether maternal smoking during pregnancy had any effect on cord blood leukocytes, and if a change was noted, to follow it through the neonatal period. Cord blood specimens were collected from 257 infants whose birthweight was over 2,500 g, and whose neonatal period was uneventful. The mothers were required to have had an uncomplicated pregnancy and stable smoking habits during pregnancy. There were 130 nonsmokers (50 percent). Smokers were categorized by approximate consumption into the following three groups: 10 cigarettes/day (20 percent), 20 cigarettes/day (21 percent), and ≥ 30 cigarettes/day (9 percent). There was no significant difference between cord blood mean corrected total leukocyte counts for nonsmokers ($13.12 \times 10^9/l$) and for smokers ($12.56 \times 10^9/l$). However, a significant depression in total cord blood leukocyte counts was seen in infants of mothers smoking ≥ 20 cigarettes/day (i.e., $11.51 \pm 3.33 \times 10^9/l$) as compared with counts found in infants of nonsmoking mothers (i.e., $13.12 \pm 3.50 \times 10^9/l$), $p < 0.005$. No significant reductions were shown in the lymphocyte, monocyte, or eosinophil numbers.

80–1734. Hopkins, J. **Foetal Health Warning.** *Food and Cosmetics Toxicology* 17(2): 172-174, 1979.

This is a review of studies on the effects of smoking during pregnancy on the fetus. One study lends strong support to the theory that smoking during pregnancy may increase the chances of spontaneous abortion. A second study showed that perinatal mortality increased by 20 percent among smokers of $<$one pack/day and 35 percent among smokers of $>$one pack/day. The same study found that maternal smoking has a significant, independent influence on the risks of premature delivery. An association between maternal smoking and early fetal death caused by abruptio placenta has been demonstrated. Another study suggests that cigarette smoke is teratogenic. Although one study suggested an association between cigarette smoking during pregnancy and congenital heart disease in the offspring, other studies have failed to show any link between them. There is still debate whether the cause of reduced birthweight is due to parental lifestyle or smoking itself. In a clinical study, placentas of smokers showed a significantly higher frequency of trophoblast abnormality.Other experiments showed that placental microsomes of smokers had a greater ability than those of nonsmokers to metabolize benzo(*a*)pyrene suggesting that maternal smoking during pregnancy might increase the susceptibility of the offspring to cancer after birth. Current research suggests the need for education on the broader effects of smoking especially on fetal health.

80–1735. Matsunaga, E.; Shiota, K. **Ectopic Pregnancy and Myoma Uteri: Teratogenic Effects and Maternal Characteristics.** *Teratology* 21(1): 61-69, February 1980.

Morphological data from 3,614 well-preserved human embryos derived from artificial termination of pregnancy were used to determine whether ectopic implantation or enlarged myomas could enhance the prevalence of localized malformations of the embryo. The frequency of malformed embryos was 11.6 percent among 43 recovered from ectopic pregnancies, 6.2 percent among 97 from myomatous pregnancies, and 3.3 percent among 3,474 from normally implanted pregnancies not complicated by myomas. Unilateral amelia in the ectopic cases and caudal dysplasia in the myoma cases were significantly increased. Both malformations were quite unusual in the control group. It is argued that spatial restrictions could be a teratogenic agent in human embryos. Mothers of both ectopic and myoma cases were on average much older than mothers of the control specimens, and those with myomas had a higher frequency of previous pregnancy wastages. Ectopic pregnancy was found to be associated with lowered parity, previous ectopic pregnancy, and maternal smoking and drinking. The percentages of smokers among the mothers with myomatous pregnancy, ectopic pregnancy, or induced abortion were 9.4, 29.2, and 15.1, respectively. The difference between the latter two categories approached significance ($p = 0.06$). (Auth. Abs. Mod.)

80–1736. Obel, E. B. **Pregnancy Complications Following Legally Induced Abortion: An Analysis of the Population With Special Reference to Prematurity.** *Danish Medical Bulletin* 26(4): 192-199, July 1979.

Pregnant women ($n = 7,270$) were registered prospectively with the purpose of evaluating if there were an increased risk of pregnancy and delivery complications in women who had previously had a pregnancy terminated by legally induced abortion. Two interviews with the women were conducted: one at first hospital contact, the second at about 28 weeks. The patient material was classified according to the termination of the previous pregnancy (i.e., legally induced abortion, spontaneous abortion or stillbirth, live birth) or to whether there had been no previous pregnancy. The group of women whose previous pregnancy had been terminated by a legally induced abortion differed from one or more of the other groups with regard to age, number of previous pregnancies and their outcome, previous illnesses, previous gynecological operations, medication during pregnancy, smoking habits (a greater percentage smoked in this group (63) than in the other three groups (51.1, 49.0, and 55.7, respectively)), and socioeconomic status. These factors were all correlated with low birthweight and/or short gestational age. Frequency of birthweight below 2,501 g was significantly ($p<0.05$) higher in smokers at both interviews; but only at the second interview was smoking correlated with gestational age under 37 weeks ($p<0.05$). These variables must be taken into consideration in an investigation of risk of pregnancy and delivery complications following legally induced abortion. (Auth. Abs. Mod.)

80–1737. O'Herlihy, C.; Martin, R. H. **Screening for Fetal Risk With Urinary Oestrogen:Creatinine Ratio at 34 Weeks.** *British Journal of Obstetrics and Gynaecology* 87(5): 388-392, May 1980.

The urinary estrogen:creatinine (O/C) ratio was measured in first morning specimens of 261 women at 34 weeks gestation to assess prospectively its potential in predicting fetal complications. Forty-two patients (16.1 percent) had low O/C ratios. All three stillbirths in the series occurred in this group, and the incidences of fetal growth retardation and fetal distress in labor were also significantly increased. These complications were unusual when the O/C ratio was high. The O/C ratio at 34 weeks reflected fetal well-being more accurately than clinical examination or blood pressure measurement. The mean O/C ratios of cigarette smokers and nonsmokers were similar, although smokers delivered lower birthweight babies. Measurement of the urinary O/C ratio is simple and inexpensive. Used as a screening test at 34 weeks, it appears to provide an early and fairly reliable indication of reduced placental function. (Auth. Abs.)

80–1738. Rantakallio, P. **Social Background of Mothers Who Smoke During Pregnancy and Influence of These Factors on the Offspring.** *Social Science and Medicine* 13A: 423-429, 1979.

The survey consisted of 1,819 mothers who smoked during pregnancy, out of a parturient population of 12,068 cases in North Finland in 1966. Perinatal mortality (births with a minimum birthweight of 600 g, stillbirths, and deaths up to the age of 28 days) did not increase among the smokers, but birthweight was lower, and both morbidity and mortality up to the age of 5 years were higher than among the controls. These differences could not be explained by socioeconomic factors, but the ill effects were more pronounced in the lower socioeconomic groups. (Auth. Abs. Mod.)

80–1739. Shiono, P. H.; Harlap, S.; Ramcharan, S.; Berendes, H.; Gupta, S.; Pellegrin, F. **Use of Contraceptives Prior to and After Conception and Exposure to Other Fetal Hazards.** *Contraception* 20(2): 105-120, August 1979.

In a large prospective study, set up to determine whether reproductive outcomes are affected by prior contraceptive use, 34,344 women were recruited at their first antenatal visit and followed until pregnancy termination. This paper summarizes the study methods and describes the demographic differences between subjects who used various contraceptives shortly before conception, and those who experienced contraceptive failures. Thirty percent of the women had used oral contraceptives (OCs) during the 5 months prior to conception and 2.4 percent had continued using them after their last menstrual period. Intrauterine devices (IUDs) had been used by 5.8 percent of women before conception and 1.1 percent after. If these rates are also true for the whole population of the United States, they indicate that approximately 70,000 babies are born each year following pill-failures and 30,000 following IUD failures. Exposure to other fetal hazards was common, with 48 percent of pregnant women drinking alcohol, 28 percent smoking (with 47 percent smoking ≥ 1 pack/day), and 15 percent receiving diagnostic X-rays. Oral contraceptive use was more common among smokers ($p<0.001$) and drinkers, and OC failures were significantly associated with the use of antiepileptics, aspirin, and with radiation exposure. (Auth. Abs. Mod.)

80–1740. Sokol, R. J.; Miller, S. I.; Reed, G. **Alcohol Abuse During Pregnancy: An Epidemiologic Study.** *Alcoholism: Clinical and Experimental Research* 4(2): 135-145, April 1980.

In a medical-record-based prospective cohort study of 12,127 pregnancies consecutively delivered during a 52-month period at a single institution, 204 (1.7 percent) were complicated by maternal alcohol abuse. A profile of these patients' concomitant risk factors and their pregnancy outcomes indicated that they tended to be older multigravidas who were not currently married. Their obstetric histories were marked by excesses of previous spontaneous abortions, low birthweight infants, and fetal anomalies. They were more likely to smoke cigarettes and abuse other drugs. The risk of intrauterine growth retardation was estimated to be increased 2.4-fold in association with alcohol abuse alone, 1.8-fold with smoking alone, and 3.9-fold with these risks together. A significant increase in congenital anomalies (38 percent) was also identified, although there were no differences between the mortality rates or placental pathology of the alcohol-abusing and comparison groups. These adverse infant outcomes did not appear to be related to demographic factors, the medical care, or nutritional status of the alcohol-abusing patients.

Moreover, cigarette smoking did not appear to account for the congenital anomalies observed in the alcohol-abusing group. The findings of this study suggest that alcohol abuse during pregnancy constitutes a significant risk for a range of adverse perinatal outcomes in as many as 50 percent of the offspring, and hence, should be considered a major public health concern. (Auth. Abs. Mod.)

See also, **80-1609, 80-1619, 80-1644**

OTHER DISEASES AND CONDITIONS

80-1741. Dupree, E. A.; Meyer, M. B. **Role of Risk Factors in Complications of Diabetes Mellitus.** *American Journal of Epidemiology* 112(1): 100-112, July 1980.

To compare antecedent and concurrent factors in diabetics and matched controls, and to measure the role played by these factors in mortality, 371 diabetics aged 20-75 years were identified from a source population of 26,000 participants in a community-wide health survey carried out in Washington County, Maryland, in 1974. (Diabetics were those who reported on the survey questionnaire that they had taken antidiabetic medications in the previous 48 hours.) Two sex- and age-matched controls for each diabetic were selected from the survey population. Comparison characteristics included blood pressure measured at the time of the survey; marital status; grades of school completed; cigar, cigarette, and pipe smoking; use of aspirin, oral contraceptives, and female hormones; and treatment for hypertension. Deaths were ascertained prospectively for 39 months following the 1974 survey. Diabetics had higher systolic blood pressures (SBPs) than controls, the distribution for diabetics resembling that of controls who were 10 years older. Smoking habits did not differ between diabetics and controls except for more cigar smoking ($p<0.01$) among male diabetics. Comparisons by cigarette smoking showed only a slight increase in mortality associated with smoking in both diabetics and controls and no evidence of interaction between diabetes and cigarette smoking in their effect on mortality. Diabetics had significantly higher mortality rates than controls in the 39 months after the survey. For all causes of death, diabetic rates were 2.5 times control rates. The increase was 1.5 times for orally treated and eight times for insulin-treated diabetics compared with their own controls. For vascular causes, the mortality risk for diabetics was increased 4.8 times overall, 2.5 times for the orally treated, and 14.2 times for the insulin-treated compared with their own controls. (Auth. Abs. Mod.)

80-1742. Farman, A. G. **Cellular Changes and *Candida* in Diabetic Outpatients Having Glossal Central Papillary Atrophy.** *Journal of the Dental Association of South Africa* 33(8): 425-429, August 1978.

Cytological studies showed that the karyopyknotic index score in diabetic outpatients was not significantly altered by glossal central papillary atrophy, but it was higher in smokers than in nonsmokers. Vacuolated cytoplasm was significantly linked with the tongue condition, while nuclear changes and perinuclear "haloing" were significantly associated with tobacco smoking. No link was found between the presence of *Candida* and vacuolated cytoplasm, nuclear changes, or perinuclear "haloing." (Auth. Abs.)

80-1743. Gersel-Pedersen, N. **Tranexamic Acid in Alveolar Sockets in the Prevention of Alveolitis Sicca Dolorosa.** *International Journal of Oral Surgery* 8: 421-429, 1979.

The effect of *trans*-4-amino-methyl-cyclohexane acid (AMCA) and a placebo preparation on the development of alveolitis sicca dolorosa (ASD) was investigated in a double-blind designed experiment. The preoperative registrations were age, sex, use of oral contraceptives, menstrual cycle, smoking, degree of impaction, and operation time. The study included 120 healthy persons. Each person had bilateral impacted mandibular molars removed surgically at one session. After the operation AMCA (160 mg/extraction site) or placebo was applied in each socket. The postoperative course was evaluated on average 5 days later by the use of 13 different variables describing local and general discomfort. The incidence of ASD was 7.5 percent in the AMCA side and 5.0 percent in the placebo side. The result shows that a local inhibition of plasminogen activation by AMCA is insufficient to prevent the development of ASD. The occurrence of ASD and postoperative discomfort was not increased in women operated on during the menstrual period. The usage of oral contraceptives is known to be associated with a high frequency of ASD. Women taking oral contraceptives may therefore postpone the operation to the withdrawal period of the pill to reduce the risk of developing ASD. Postoperative pains and the consumption of analgesics were significantly ($p<0.001$) increased in patients who were habitual smokers. (Auth. Abs.)

80-1744. Marks, I. N.; Wright, J. P.; Denyer, M.; Garisch, J.A.M.; Lucke, W. **Comparison of Sucralfate With Cimetidine in the Short-Term Treatment of Chronic Peptic Ulcers.** *South African Medical Journal* 57(15): 567-572, April 12, 1980.

One hundred and twelve patients with endoscopically proven peptic ulcers participated in a controlled trial to assess the relative efficacy of sucralfate and cimetidine. The 57 patients with duodenal ulcers (DU) and the 55 patients with gastric ulcers (GU) were alternately assigned to sucralfate and cimetidine regimens, and repeat endoscopy was carried out after 6 weeks of therapy. Patients with unhealed ulcers underwent endoscopy for a third time after a further 6 weeks of therapy. In the DU group, 24 of 29 patients on sucralfate had healed at 6 weeks (83 percent); and the remaining 5 patients had all healed after a further 6 weeks of therapy (100 percent), compared with healing rates of 71 percent and 86 percent respectively in the cimetidine-treated patients. In the GU group, 17 of 27 patients on sucralfate healed at 6 weeks (63 percent) and, allowing for 3 late defaulters, the 12-week healing rate was 83 percent. Healing rates of 75 percent and 89 percent respectively were achieved with cimetidine. Symptomatic responses were similar in the two treatment groups for both DU and GU. Ninety of the 112 patients were smokers and although a few of them smoked less during the trial, only 3 stopped smoking completely. About half the smokers in each subgroup smoked more than 15 cigarettes a day. The incidence of smoking in the healed and nonhealed DU subgroups was identical at 67 percent. All 17 patients with nonhealed GUs at 6 weeks were smokers, but this was not significantly different from the 84 percent incidence found in the healed GU subgroups. Constipation was more common in the sucralfate-treated patients (10 percent); and mild headaches and, to a lesser extent, nausea, dizziness, or depression were more frequent in the cimetidine groups (15 percent). The study suggests that sucralfate is a safe preparation which compares favorably with cimetidine in the treatment of peptic ulceration. (Auth. Abs. Mod.)

80-1745. McNair, P.; Christensen, M. S.; Madsbad, S.; Christiansen, C.; Binder, C.; Transbol, I. **Bone Loss in Patients With Diabetes Mellitus: Effects of Smoking.** *Mineral and Electrolyte Metabolism* 3(2): 94-97, 1980.

The present study was performed in order to examine the role of tobacco smoking on the loss of bone mineral in diabetes mellitus. Among 163 adult insulin-treated diabetic patients, 114 were smokers. The bone mineral content (BMC) was measured by photon absorptiometry in both forearms and was on average reduced by 9.0 percent of normal in the entire group of diabetics ($p<0.001$). In smokers the mean BMC loss was 10.6 percent compared to 5.4 percent in nonsmokers, a significant difference ($p<0.01$). No difference was found between smokers and nonsmokers as regards age at onset, duration of diabetes, or fasting hyperglycemia and glycosuria. Evidently, tobacco smoking is an important risk factor in diabetic bone loss. (Auth. Abs.)

80-1746. Taylor, W. H.; Walker, V. **Smoking and Peptic Ulceration.** *Journal of the Royal Society of Medicine* 73(3): 159-161, March 1980.

Studies from the past 50 years on smoking and peptic ulceration are reviewed. It is concluded that there is an increased frequency of smoking in both duodenal and gastric ulcer patients when compared with controls, there is an increased prevalence of peptic ulcer (approximately twofold) among smokers of both sexes when compared with nonsmokers, the incidence of peptic ulcer increases with increasing numbers of cigarettes smoked, cigarette smoking retards the healing of both gastric and duodenal ulcers, and male cigarette smokers have a twofold greater chance of dying from peptic ulcer than male nonsmokers.

80-1747. Tellier, L.; Thuillier, A. **Etude Comparative de la Flore Bacterienne de la Plaque Dentaire Chez les Fumeurs et les Non Fumeurs. [Comparative Study of Bacteria in the Dental Plaque of Smokers and Nonsmokers.]** *L'Information Dentaire* 60(38): 37-43, September 21, 1978, French.

A bacteriologic study was made of dental plaque specimens from two groups of ten healthy men, 20-40 years of age. One group was composed of nonsmoking students, the other group of smokers of at least 20 cigarettes/day with a several-months-old dental calculus. The dental plaque was sampled twice from each subject, before and 4 days after a thorough cleaning of the teeth. Each time three plaque samples were taken to grow different bacteria in three culture media. Microscopic examination of the cultures was carried out after a minimum of 8 days from the date of inoculation, before and after staining by different techniques. Qualitative examination revealed the existence of different types of bacteria (including streptococci, staphylococci, neisseria, saccharomycetes) in the dental plaque of both smokers and nonsmoking controls. The incidence of each bacterial type was not significantly different within the two groups. It is concluded that smoking does not appreciably affect the quality of the bacterial flora of dental plaque. The toxicity of tobacco smoke to the buccal tissue remains unproved.

80-1748. Weber, A.; Fischer, T.; Grandjean, E. **Passive Smoking: Irritating Effects of the Total Smoke and the Gas Phase.** *International Archives of Occupational and Environmental Health* 43(3): 183-193, 1979.

In a ventilated climatic chamber 32 subjects (in groups of two or three) were exposed in one experiment to the total sidestream smoke of cigarettes and in another experiment to the gas phase of cigarette smoke, which was obtained by filtering out the particulate matter. The degree of air pollution corresponded to 10 ppm carbon monoxide (CO) in both experiments. The concentrations of the particulate matter, CO, nitric oxide (NO), nitrogen dioxide (NO_2), and formaldehyde (HCHO) were recorded. The degree of annoyance and of subjective irritation of the eyes, nose, and throat was determined with a questionnaire; eye blink rate was measured, as an objective indicator of eye irritation. Both exposures of air pollution produced nearly the same degree of annoyance, which indicates that the gas phase is to a large extent responsible for the annoyance due to cigarette smoke. On the other hand, the objective and subjective eye irritation

was very much lower with the gas phase than with the total smoke. This indicates that the particulate phase is to a very large extent responsible for the irritating effects. See also 80-0809. (Auth. Abs. Mod.)

See also, **80–1703**

BEHAVIORAL AND EDUCATIONAL RESEARCH

80–1749. Abramson, J. H.; Hopp, C.; Gofin, J.; Gofin, R.; Makler, A.; Habib, J.; Ronen, I. **A Community Program for the Control of Cardiovascular Risk Factors: A Preliminary Evaluation of the Effectiveness of the CHAD Program in Jerusalem.** *Journal of Community Health* 5(!): 3-21, 1979.

A community program for the control of cardiovascular risk factors, the CHAD program, was instituted in a family practice in western Jerusalem in 1971. The effectiveness of the program was evaluated by comparing the findings of community surveys conducted in 1970 and 1975 and by comparing the changes with those observed in an adjacent control neighborhood. This paper reports the findings among men aged ≥35 years. In the community exposed to the program there was a decrease in mean systolic and diastolic pressures, in the prevalence of hypercholesterolemia, and in the prevalence of cigarette smoking. The age-standardized percentage of those who smoked dropped by 12.9 (from 54.4 to 41.5 percent) in the CHAD population, and by 6.2 (from 44.6 to 38.4 percent) in the control population. The increase in nonsmokers was primarily due to a decrease in the light smoker population; there was little change in the ≥20 cigarettes/day smoker population. The changes in blood pressures and smoking habits were significantly greater than those observed in the control population. In the control population (only) there was a small increase in mean body weight and a decrease in reported physical activity. The findings suggest that it is possible for a program that operates in and through primary health care to have an appreciable influence upon cardiovascular risk factors in the community. (Auth. Abs. Mod.)

80–1750. Adams, P. I. **The Influence of Cigarette Smoke Yields on Smoking Habits.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 349-360.

Three experiments were conducted in order to determine whether changes in cigarette smoke yield influence personal smoking habits. In the first experiment, three types of filter cigarettes were tested. The particulate matter (water- and nicotine-free) yields were 17-23 mg and the nicotine levels were 1.6-1.7 mg. One cigarette type had a carbon filter and therefore about a 50 percent reduction in vapor phase delivery. The smokers took significantly larger puffs from the vapor phase-reduced cigarettes and therefore showed some change in habit despite otherwise fairly similar particulate matter and nicotine levels. The second experiment was designed to measure subjective opinions and to determine whether the first experiment results were representative of the general public. When nicotine and particulate matter were reduced simultaneously the smokers took an increased volume of smoke. For a low yielding, ventilated cigarette in which yield reduction was about 40 percent, the amount of compensation by puffing was 10 percent and the possible increase in cigarette usage was a further 5 percent. The smokers responded to higher pressure drop cigarettes by lowering their puffing flow rate so as to lower the perceived draw resistance. The third experiment investigated whether smokers increase consumption when a middle-tar brand is exchanged for a low-tar cigarette brand, whether being informed of this change affects consumption, and whether consequent changes in puffing habits are reproducible. For this experiment, there were 10 smokers informed of the brand change, 10 smokers uninformed of this change, and 12 controls (no change in brands). Data for consumption effects were inconclusive. Knowledge of the brand change had statistically nonsignificant effects on smoking habit. As for puffing variables (i.e., total volume, duration, time lit; number of puffs; amount of filter nicotine), the cigarette type had a significant effect at the 0.01 level or higher, on all variables except time lit. The response to the change from middle tar to low tar was to increase smoke volume by taking longer and more frequent puffs.

80–1751. Ashton, H.; Stepney, R.; Thompson, J. W. **Smoking Behaviour and Nicotine Intake in Smokers Presented With a "Two-Thirds" Cigarette.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 315-329.

Fourteen smokers smoked their own brand of middle-tar cigarettes in their usual way or smoked only two-thirds of the length of tobacco rod normally consumed over a 24-hour period. The majority of subjects found smoking full cigarettes more satisfying and enjoyable. There was a slight (nonsignificant) tendency for the heart rate increase to be greater in the two-thirds cigarette condition, possibly due to a faster rate of nicotine intake even though the total dose of nicotine was lower. Fingertip temperature fell to a greater extent in the full cigarette condition ($p<0.05$). Increase in puff duration or mean interpuff interval was noted in 13 of 14 subjects in the two-thirds cigarette condition. Over the 24-hour period, subjects obtained 99.8 percent of the expected delivery of the full cigarettes but 112.3 percent of the expected delivery of the two-thirds cigarettes as determined by butt analysis. The difference between these values is significant ($p<0.001$). Also, subjects obtained significantly more nicotine from their cigarettes smoked in the laboratory than from the 24-hour period away from the laboratory, both under the full and two-thirds cigarette conditions. Subjects under the two-thirds cigarette condition had plasma nicotine levels 71.7 percent of those of subjects smoking full cigarettes indicating they were obtaining more than the expected two-thirds dose. There was no correlation between the puff parameters studied and nicotine intake in the two-thirds cigarette condition, unlike the full cigarette condition. Aspects of smoking behavior other than those measured probably play a critical role in nicotine intake in the two-thirds cigarette condition. Samples of normal cigarette butts collected before and after experience with the two-thirds cigarette condition showed that subjects smoked less of the tobacco rod, but nicotine deliveries before and after experience with the two-thirds cigarettes were not significantly different.

80–1752. Baric, L.; Fisher, C. **Acquisition of the Smoking Habit.** *Health Education Journal* 38(3): 71-76, 1979.

The aim of this study was to explore the relationship between parents' and children's smoking by use of a theoretical model of primary socialization. The study explored the parents' smoking behavior, the children's awareness of smoking, exposure to smoking, experimentation with cigarettes, and the parents' health education practices concerning children's smoking. The study sample included 224 mothers with small children (0-5 years of age). The questionnaire consisted of a number of closed and open-ended questions covering the following areas of information: social characteristics, smoking behavior of parents, parents' beliefs and feelings about smoking, parents' attitudes and perception of norms related to smoking, children's smoking and health education, and mother's views on the child's awareness and reactions to smoking. The study showed that the first year of a child's life is probably the most sensitive period as far as awareness of the habit, acquisition of manipulative skills, imitation, and modeling on significant others is concerned. There was a marked difference in the educational problems of parents who smoke as compared with those who do not smoke. Both were willing to tell children about dangers from smoking early, but few smokers were willing and able to support this by their example. Mothers did not seem to understand the mechanisms of primary socialization such as modeling, imitation, observation, and identification, and tended to disregard their crucial role in this process.

80–1753. Barnea, E.; Barnea, M.; Ardelean, I.; Modval, R.; Taus, V.; Zahiu, L. **Contributions to the Evaluation of the Present Stage of Smoking Frequence to the University Youth.** *Sante Publique* 22(3): 273-287, 1979.

This study of smoking among Romanian youth has been carried on simultaneously in two provincial towns by questioning a total number of 3,030 young people of both sexes aged 18-23 years. The subjects were technical university students. Information about smoking frequency among the subjects, their families, and their environment was obtained through a questionnaire. The smokers were questioned as to when and why they began to smoke, and about their daily consumption of cigarettes. It was found that the number of smokers had gradually increased, especially among female young people, approaching the level reached in developed countries. Smoking among girls increased with the educational level. In most cases, the offspring of smoking families were also smokers. Imitation, curiosity, and the wish to appear more mature were mentioned as motivation for smoking among both sexes. The age of smoking onset decreased slowly, with boys still starting at an earlier age. The earlier onset and greater prevalence of smoking among urban populations as compared with rural populations were no longer evident. The increase in smoking among young people consisted of an increased growth in the proportion of male and female smokers who were smoking ≥ 20 cigarettes/day. (Auth. Abs. Mod.)

80–1754. Bland, J. M.; Bewley, B. R.; Banks, M. H. **Cigarette Smoking and Children's Respiratory Symptoms: Validity of the Questionnaire Method.** *Revue d'Epidemiologie et de Sante Publique* 27: 69-76, 1979.

Children who report smoking cigarettes also report increased respiratory symptoms. In order to test the validity of the questionnaire method, reports of children's respiratory symptoms obtained from children (3,098 boys and 3,232 girls aged 11-12 years) and from their parents were compared in England. Questions were asked about cough in the morning and at other times as well as children's and parents' smoking habits. Results indicated a significant association between parents' and children's reports, although the relationship was not close. Morning cough, for example, was reported by 3.7 percent of the children and 2.4 percent of the parents. The relationship between respiratory symptoms and cigarette smoking was consistent, whichever report was used. It was concluded that the questionnaire technique is valid, and that children who smoke cigarettes are more likely to have respiratory symptoms than nonsmokers.

80–1755. Briddell, D. W.; Rimm, D. C.; Caddy, G. R.; Dunn, N. J. **Analogue Assessment, Affective Arousal, and the Smoking Taste Test.** *Addictive Behaviors* 4(3): 287-295, 1979.

The relation between conditions of affective arousal (i.e., stress, relaxation) and smoking was evaluated by the utilization of the taste test as the dependent measure for smoking behavior in 36 subjects. Experimentally manipulated levels of arousal did not significantly influence smoking intensity or topography (e.g., number of puffs, length of puff, interpuff interval), however, the results are qualified by data which cast doubt on the validity of the taste test. There were no significant correlations between performance on the taste test and smoking in the natural environment. The implications regarding the validity of assessment analogue procedures are presented. (Auth. Abs. Mod.)

80-1756. Charlton, A. **Cancer and Cancer Education—Opinions of Some Secondary School Teachers in Northern England.** *Health Education Journal* 38(3): 77-83, 1979.

To provide information about teachers' attitudes to cancer and cancer education, a questionnaire survey of 131 men and 60 women teachers was conducted in 1974 in five comprehensive schools in northern England. These teachers were well-informed about cancer, especially that it is sometimes curable and susceptible to early treatment, although the majority still found it the most alarming disease. Most of the teachers (64 percent) had had no lectures or talks about cancer in their own educational background. Twelve percent had heard talks on cancer at secondary school, and some mentioned that these had been about smoking. While 95 percent of the teachers thought it right to teach about cancer in schools, all of them agreed that health education in general should be taught in schools. The reasons given against cancer education were that there is already too much in the curriculum, that it would worry pupils, that cigarette manufacturers get too much publicity and profit already, and that it should only be done by the medical profession, because faulty information would do more harm than good. However, 45 percent of the teachers were willing to teach about cancer if they had more information; a further 21 percent if they had a teaching kit, and 9 percent without further help. Those willing to teach about smoking included 82.4 percent of the men and 85.0 percent of the women. Based on their expressed wishes for cancer education methods, combined with the results of a concurrent survey of pupils' requirements, a teaching resources package was designed for testing. (Auth. Abs. Mod.)

80-1757. Creighton, D. E.; Lewis, P. H. **The Effect of Different Cigarettes on Human Smoking Patterns.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 289-300.

Frequent measurements of the smoking patterns were recorded for 16 subjects who were changed from a control brand of cigarettes to either a higher or lower delivery brand. Each cigarette brand was smoked exclusively for about 4 weeks. Representative smoking patterns (including puff volume, puff duration, and average butt length) for each subject were duplicated by machine to measure deliveries of total particulate matter (TPM), nicotine, and carbon monoxide (CO). Analyses of these data showed that on average smokers increased the intensity with which they smoked the lower delivery brand and decreased the intensity of smoking the higher delivery brand when compared with the control brand. The number of cigarettes smoked per day did not change significantly. The amounts of TPM, nicotine, and CO taken from the medium- and high-delivery brands by the smokers were similar, although the deliveries of these two brands differ considerably when smoked by machine under standard conditions. Although they smoked the low-delivery brand more intensely, the smokers did not equalize the deliveries taken from the low- and medium-delivery brands. A further analysis showed that the modified smoking patterns adopted to smoke the brands with changed deliveries were maintained for the period of smoking those brands. The smokers reverted to their former smoking patterns during the last phase of the experiment when they again smoked the control brand. (Auth. Abs. Mod.)

80-1758. Creighton, D. E.; Noble, M. J.; Whewell, R. T. **Instruments to Measure, Record and Duplicate Human Smoking Patterns.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 277-288.

This paper describes the structure, functions, and operation of a smoking analyzer and data logger designed to monitor the way human subjects smoke cigarettes, and a puff duplicator which smokes cigarettes to the recorded smoking patterns. The data may be displayed in numeric form and stored in digital form on magnetic tape. The magnetic tape may be read in several forms so that printouts of the pressures, flows, volumes, durations, and intervals between the puffs may be made in a number of ways. Paper tapes are made to control the functions of the puff duplicator which smokes cigarettes in the same pattern as the human smoker, enabling the tar, nicotine, and gas phase components to be collected for analysis. This instrument was used to duplicate the smoking patterns of a number of subjects who participated in a study of the effects of brand changes. It was found that the accuracy of volume duplication is generally within 1.0 percent, and the timing of both puff durations and intervals between puffs is within 0.02 sec.

80-1759. Dunn, P. J. **The Effects of a Reduced Draw Resistance Cigarette on Human Smoking Parameters and Alveolar Carbon Monoxide Levels.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 203-207.

Smoking parameters and alveolar resting carbon monoxide (CO) levels were measured in seven heavy smokers when they switched from smoking their normal brand of cigarette to cigarettes with 30 percent reduced draw resistance. There were no trends or significant changes with regard to butt length, puff number, or interval between puffs. The smokers, however, took puffs of 9.5 percent shorter duration and of 20 percent greater effort, which resulted in an increase of 28 percent in relative velocity. An average increase in relative volume per puff of 9 percent was observed for six of the seven smokers studied. An increase of 7 percent in total relative volume per cigarette was noted for five of the seven smokers as a result of changes in puff number. These changes in smoking parameters resulted in a significant increase of 56 percent in mouth nicotine intake. Despite the substantial increase in nicotine to the mouth with the cigarette of lower draw resistance, the alveolar resting levels decreased by an average of 16 percent, which is markedly (29 percent) less than the level predicted using mouth nicotine as an indicator of total smoke concentration. This difference is quite possibly due to all smokers inhaling less deeply with the increased nicotine concentration in the mouth. The ease of availability of nicotine with the cigarette of lower draw resistance (more nicotine per unit effort) did not cause a decrease in total amount of smoke taken from the cigarette; in fact the smokers appeared to show increased effort when smoking this cigarette. There was no significant change in total daily cigarette consumption or other smoking parameters. In experiments designed to measure how a smoker responds to cigarettes of different types or to cigarettes smoked under varying experimental conditions, it is essential to estimate inhalation.

80-1760. Dunn, P. J.; Freiesleben, E. R. **The Effects of Nicotine-Enhanced Cigarettes on Human Smoking Parameters and Alveolar Carbon Monoxide Levels.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 195-202.

A study was conducted to test the assumption that smokers modify their smoking to regulate nicotine intake. A change in inhalation is one means of smoke compensation, i.e., a smoker unconsciously inhales cigarette smoke of higher nicotine concentration to a lesser extent than smoke of lower nicotine concentration, resulting in a lower body carbon monoxide (CO) level. Four subjects who were heavy smokers were presented with cigarettes of higher nicotine delivery and were monitored for alveolar CO levels and smoking parameters. The study found no direct relationship between the cigarette smoke yield of CO obtained under standard smoking conditions and the actual alveolar uptake of CO during smoking. The resting levels of alveolar CO were significantly lower for all smokers when cigarettes of unchanged CO yield but 30 percent higher nicotine yield were smoked. The smokers did not compensate for the 30 percent increase in nicotine delivery by significantly altering puff number, interval, duration, or butt length. No decrease in total daily cigarette consumption was noted with the increased nicotine availability. With the nicotine-enhanced cigarette, all smokers reduced, to varying degrees, the amount of smoke inhaled, as evidenced by the lower alveolar CO resting levels. This greater availability of nicotine to the smoker may be an effective means of reducing the intake of tar and CO.

80-1761. Eiser, J. R.; Sutton, S. R.; Wober, M. **Smoking, Seat-Belts, and Beliefs About Health.** *Addictive Behaviors* 4(4): 331-338, 1979.

Questionnaires concerned with cigarette smoking and seat-belt use were completed by 378 respondents (28.8 percent were smokers, 44.4 percent never-smokers, and 26.7 percent ex-smokers) who were taking part in a survey of audience reactions to 1 week's television programs. Controlling for sex, age, and social class, self-reported seat-belt use was lower among smokers than nonsmokers ($p<0.01$). Smokers tended to hold less negative beliefs about the consequences of smoking than did nonsmokers. Notably, only 49.5 percent of smokers (as against 88.7 percent of never-smokers) believed smoking to be "really as dangerous as people say," and 13.8 percent of smokers (as against 29.2 percent of never-smokers) realized that smoking caused more deaths than road accidents. Similarly, nonwearers of seat-belts were more skeptical of the benefits of seat-belt use (51.6 percent of nonwearers believed "seat-belts really make driving much safer" compared with 92.2 percent of wearers). Respondents who asserted that people have the right to put their own health at risk, as opposed to a moral responsibility not to risk their health, were more likely to smoke and less likely to wear seat-belts. It is concluded that both smoking and the nonuse of seat-belts may partly reflect more general attitudes about safeguarding health, as well as more specific beliefs which may be susceptible to informational influence. (Auth. Abs. Mod.)

80-1762. Frederiksen, L. W.; Martin, J. E.; Webster, J. S. **Assessment of Smoking Behavior.** *Journal of Applied Behavior Analysis* 12(4): 653-664, Winter 1979.

The nature of smoking risk is first reviewed, and a classification of procedures for assessing smoking behavior is presented. Areas requiring assessment include not only the traditionally measured smoking rate, but also the substance used and topography of consumption. Each of these areas may be assessed through a variety of self-report, observational, or indirect techniques. These techniques as well as some of their advantages and disadvantages are presented. Recently published (1975 to mid-1978) data-based smoking research appearing in four behavioral journals is then reviewed with respect to measurement reliability and the use of multiple measures. Results show a strong tendency to assess only the risk area of smoking rate and a low frequency of appropriate measurement reliability checks, especially during baseline and treatment phases. Some of the implications of these results are discussed. (Auth. Abs.)

80-1763. Friedman, H. H.; Dipple, W. S., Jr. **The Effect of Masculine and Feminine Brand Names on the Perceived Taste of a Cigarette.** *Decision Sciences* 9(3): 467-471, July 1978.

The purpose of the current study was to determine whether brand names can affect the public's evaluation of a product.

All subjects (100 males, 100 females) smoked identical cigarettes. One group of subjects, however, knew the cigarettes by the name of "Frontiersman," a masculine name, while the other group knew the cigarettes as "April," a feminine name. Male and female subjects were asked to rate the cigarette on seven measures. Results show that women gave a more positive evaluation to the cigarette purportedly named "April," while men gave a more positive evaluation to the identical cigarette when they thought it was named "Frontiersman." In addition, women reacted more strongly than did men to brand name influence. The women rated "Frontiersman" as being of blander flavor, hotter and stronger taste, harsher, less enjoyable, and more masculine than "April." (Auth. Abs. Mod.)

80–1764. Greenwood, S. G. **Warning: Cigarette Smoking is Dangerous to Reproductive Health.** *Family Planning Perspectives* 11(3): 168-172, May/June 1979.

Against the background of findings that smoking presents a particular danger for women of reproductive age, 252 Planned Parenthood clinics were surveyed in 1978 to determine whether educational campaigns about smoking were in progress, and what the clinics' smoking policies for patients and staff were. Responses from 197 clinics indicated that there was a strong interest in informing patients about the risks of smoking. Half the clinics had banned smoking among patients. Only about one-fifth, however, had banned smoking in the clinic by staff (although another fifth had restricted staff smoking to certain areas). Banning or restricting staff smoking was seen as a thorny issue involving individual rights and job efficiency. The issue is being worked out in different ways, depending on the balance of opinion at individual clinics. Six out of 10 respondents said no visual materials on the hazards of smoking were used, although more than 8 out of 10 said they saw a need for such materials. Almost 9 out of 10 clinics provided patients with written material on smoking hazards, and almost all dealt with the subject in counseling sessions. (Auth. Abs. Mod.)

80–1765. Guillerm, R.; Radziszewski, E. **Analysis of Smoking Pattern Including Intake of Carbon Monoxide and Influences of Changes in Cigarette Design.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 361-370.

This paper combines three techniques (i.e., measurement of puff parameters, analysis of the smoker's ventilatory behavior, measurement of carbon monoxide (CO) intake) to analyze the behavior of eight smokers voluntarily changing their usual cigarette (UC) for a less irritant cigarette (EC) over a 7-day period. The cigarettes were similar in nicotine contents, but EC had an active charcoal filter. Puff parameters were measured by use of a flowmeter and a pyrometer. Measurements of ventilatory behavior were made with impedance pneumography, whereas blood carboxyhemoglobin (COHb) level was monitored for CO intake. The experimental schedules had a morning and an afternoon session. The morning sessions took place after cigarette deprivation, the second after unrestricted smoking. (Subjects were allowed to smoke the UC to 23 mm, and the EC to 29 mm.) The location of the puff in the ventilatory cycle was during an expiratory pause for 80 percent of the subjects. Changing from the UC to the EC resulted in several significant changes in the smoking pattern. With the EC, the puff peak fell by 31 percent ($p=0.001$), the number of puffs increased by two ($p=0.001$), the puff duration was 27 percent longer ($p=0.001$), and total smoking duration increased from 390 to 455 sec ($p=0.01$). The COHb level at the end of each day's session was significantly lower with EC than with UC ($p=0.001$).

80–1766. Hayashi, T. **Recent Trends in Smoking in Japan.** *World Smoking and Health* 5(2): 40-42, Summer 1980.

A 1979 market survey disclosed that despite an increase in the adult population in Japan, the estimated total number of regular adult smokers dropped to 34.8 million in 1979, compared to 35.3 million in 1978. The percentage of adult male (mostly older) smokers declined from an estimated 83.7 percent in 1966 to an estimated 73.1 percent in 1979; comparable figures for adult female smokers were 18.0 and 15.4 percent, respectively. The number of current regular smokers in the 20-29 year age group, however, increased. Among physicians and dentists the percentage of smokers is much lower than among the general population, ranging from 33 to 36 percent for physicians and from 44 to 46 percent for dentists in the Tokyo metropolitan area.

80–1767. Henningfield, J. E.; Griffiths, R. R. **Effects of Ventilated Cigarette Holders on Cigarette Smoking by Humans.** *Psychopharmacology* 68(2): 115-119, May 1980.

Heavy cigarette smokers individually attended daily 3-hour test sessions which were run in specially designed cigarette smoking evaluation rooms. Subjects were required to use the cigarette holder provided and to extinguish each cigarette 4 min after the first puff on the cigarette. Other than these restrictions, subjects were allowed to smoke ad libitum. The concentration of delivered tobacco product was varied from 100 to 10 percent across sessions by using graded commercially available ventilated cigarette holders. As concentration of tobacco product was decreased, rate of puffing and total number of puffs showed robust compensatory increases ($p<0.01$). Number of cigarettes increased only moderately in response to decreases in tobacco product concentration. There was little change in subjective ratings of strength on smoking satisfaction. Finally, expired air carbon monoxide values and cigarette butt weights were relatively stable across the four ventilation conditions. These later findings suggest that a significant degree of compensation had occurred in response to the concentration manipulations. (Auth. Abs.)

80–1768. Hrbek, J.; Komenda, S.; Navratil, J.; Siroka, A. **Comparative Effects of Smoking Marihuana (With Tobacco) in Men and Women.** *Agressologie* 19(D): 197-199, 1978.

The acute effect of smoking marihuana with tobacco on the learning process in men and women was studied with the help of a laboratory language method. The experiments were performed immediately before, and 15 and 75 min after smoking eight different doses of marihuana combined with tobacco. Of the doses, four contained denicotinized tobacco. In women, all the higher doses of marihuana decreased the test performance (number of correct reactions). The performance results in men were better. (Auth. Abs. Mod.)

80–1769. International Union Against Cancer. **Evaluation of the Educational Programme in the Fight Against Nicotine Addiction.** In: Hobbs, P. (Editor). *Public Education About Cancer. Recent Research and Current Programmes.* UICC Technical Report Series, Volume 45, Geneva, 1979, pp. 36-40.

This is an evaluation of a 2-year intensive educational program undertaken by the French Health Educational Council in order to combat the noxious effects of tobacco on health. The program was carried out in three different stages. The first stage intended to disseminate information to the whole population about the health hazards of tobacco, the second stage was more concerned with sensitizing opinion to the new legal measures taken to protect the rights of nonsmokers, and the third stage involved launching an educational program intended for young people and adolescents. The campaign results showed (1) a 3 percent reduction in the number of smokers and a 15 and 16 percent reduction in tobacco consumption by adults and young people, respectively, and (2) an acknowledgement by both smokers and nonsmokers that they were aware of the new regulations restricting smoking in some public places.

80–1770. Kelson, S. R.; Pullella, J. L.; Fulton, G. B. **The Control of Smoking: The Physician's Role.** *Ohio State Medical Journal* 76(6): 359-362, June 1980.

Surveys in 1964 and 1971 of Toledo, Ohio, area public school students showed increased smoking in 1971 by boys and girls in every grade. Higher percentages of boys smoked both years in all grades except 8 and 9 in 1971. A 1976 survey showed further increases in the percentages of girl smokers — exceeding boys in every grade except 12. The percentages of boys who smoked increased over 1971 in grades 7, 8, and 9, but decreased in 10, 11 and 12. Evidence indicates the predominant antismoking influence of physicians. Intensified antismoking education in medical and osteopathic school curricula is stressed, as is the formation of separate national, state, and local groups in the cancer, heart and lung organizations, or in interagency agencies, solely to spur antismoking efforts by physicians. (Auth. Abs.)

80–1771. Knott, V. J. **Personality, Arousal and Individual Differences in Cigarette Smoking.** *Psychological Reports* 45: 423-428, October 1979.

This study attempted to examine physiologically based personality correlates of "low-arousal smokers" and "high-arousal smokers." The former were smokers who generally experienced their strongest need to smoke in low-arousal situations, characterized by, e.g., monotony or boredom, while the latter experienced their strongest need to smoke in high-arousal situations, characterized by, e.g., anxiety or excitement. Members of each group completed the Eysenck Personality Inventory and the Vando Reduction-Augmentation Scale. High-arousal smokers exhibited significantly greater scores on extraversion ($p<0.05$), neuroticism ($p<0.05$), and reduction ($p<0.025$) than low-arousal smokers. (Auth. Abs. Mod.)

80–1772. Kumar, R.; Cooke, E. C.; Lader, M. H.; Russell, M.A.H. **Is Tobacco Smoking a Form of Nicotine Dependence?** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 244-258.

Twelve paid volunteers who were moderate to heavy smokers were given comparable doses of nicotine either by tobacco smoke inhalation (Experiment 1) or by intravenous injection (Experiment 2). The dependent variable in both cases was the amount and rate of ad libitum puffing of medium strength, filter-tipped cigarettes following each nicotine dose. Smoking behavior was monitored by measuring puff number, interpuff intervals, and duration and depth of each puff. Heart rate, skin temperature, skin conduction, and electroencephalogic (EEG) determinations were made for each subject. Experiment 1 showed that inhaled doses of smoke systematically postponed and reduced subsequent puffing and that this was a function of the content of the smoked doses. In the second experiment, however, the intravenous doses were without effect on smoking behavior. The physiological tests showed that the doses of nicotine clearly modified the heart rate and also the EEG (β activity) and very comparable effects were seen in the two experiments. Thus, the lack of effect of intravenous nicotine on smoking cannot be ascribed either to inadequate dosage or to a failure of the drug to enter the blood and the brain. These negative findings reopen the question whether physiological dependence upon nicotine really is the basis for the tobacco smoking habit.

80–1773. Lamontagne, Y.; Annable, L. **Acupuncture for Smokers: Effect of Expectation on Outcome.** *American Journal of Acupuncture* 7(3): 255-259, July-September 1979.

This study investigates the effects of the subject's own expectation of the outcome on the efficacy of acupuncture as a treatment for cigarette smoking. Twenty-five healthy male smokers were recruited for a high-expectancy group and 25 smokers for a low-expectancy group. The subject's mean age was 31.7 years, average cigarette consumption was 30.1/day, and mean addiction duration was 16.5 years. All subjects received two weekly 20-min acupuncture sessions; needles were placed at the "Ear 0" and "Lung" points. Results showed that at no time during the 6-month followup were there any statistically significant differences between the two groups as regards the number of subjects who quit smoking. The significant improvement in smoking rate achieved by subjects who did not stop smoking gradually declined during followup in both groups. However, the gains made by high-expectancy subjects were more prolonged than those of the low-expectancy subjects. Thus, although the subject's own confidence in the efficacy of acupuncture did not influence the likelihood of his quitting smoking, it did appear to have some effect for those who reduced their smoking rate following treatment. It is concluded that a number of psychological and practical questions still need to be answered by controlled experimentation before acupuncture can be considered an efficacious treatment for smokers. (Auth. Abs. Mod.)

80–1774. Lasogga, F. **Personlichkeitsmerkmale und Rauchverhalten Jugendlicher. [Personality Traits and Smoking Behavior of Adolescents.]** *Zeitschrift fur Klinische Psychologie—Forschung und Praxis* 7(3): 162-171, 1978, German.

All the students in the seventh grades in Cuxhaven (n = 726) were subjects in an experiment which attempted to establish correlations between the use of tobacco and personality variables. It was found that 69 percent of the students had smoked at some time. Young smokers were shown to be more neurotic than young nonsmokers. The lower values in scales testing truthfulness obtained by the young smokers suggested a greater readiness to contravene social norms. No connection between smoking and the degree of extraversion was found, a result which contradicts the evidence from adults. Furthermore, the amount of pocket money available proved to be significant. Young smokers had considerably more pocket money at their disposal than young nonsmokers. (Auth. Abs. Mod.)

80–1775. Leathar, D. S. **Smoking Among Student Nurses. An Exploratory Study Into the Reasons Underlying Nurses' Smoking Habits.** *Nursing Times* 76(14): 589-590, April 3, 1980.

This study, funded by the Scottish Health Education Unit, investigated potential causes of the high percentage of smoking among nurses. Some of the problems identified were general atmosphere problems such as leaving home, new working environment, and adjusting to the shift system that young nurses have to cope with in their first training period. Other problems were more directly related to the complexity of the nursing job itself: the ability to cope with death, long night duties with a large number of patients and little assistance, complex sophisticated medical machinery, and the rigid authority structure of the medical hierarchy. In order to cope with these problems, nurses formed informal friendship groups where smoking facilitated a relaxed atmosphere. The study suggests the development of a counseling support system outside the existing authority in the hospital as a way to reduce the smoking habit among nurses.

80–1776. Leon, G. R.; Kolotkin, R.; Korgeski, G. **MacAndrew Addiction Scale and Other MMPI Characteristics Associated With Obesity, Anorexia and Smoking Behavior.** *Addictive Behaviors* 4(4): 401-407, 1979.

Groups of persons with substance abuse problems (obesity, smoking, and anorexia) and a normal control group of persons without habit problems were evaluated on the MacAndrew Scale (MAC) of the MMPI (originally developed to measure proneness to alcoholism). Two point code types were calculated for those persons for whom complete MMPI information was available. The male smokers demonstrated a mean MAC score (24.864) close to the addictive range. The female smokers and the moderately obese groups, as well as the anorexic group, showed no significant differences in MAC score (19.617, 20.356, and 20.1, respectively) in comparison to the normal controls (21.595 for males and 19.617 for females). The smokers and obese subjects showed a tendency toward depression and impulsivity, and the smokers also showed a trend toward somatization. It appears from the present findings as well as those of other investigators that an elevated score on the MacAndrew Scale can be interpreted as an indication of an addiction problem or addiction proneness. However, not all persons with substance abuse patterns have an elevated score on this measure. (Auth. Abs. Mod.)

80–1777. Mangan, G. L.; Golding, J. **An 'Enhancement' Model of Smoking Maintenance?** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 87-114.

Data from the five experiments reported in this chapter: (1) effects of external manipulation of nicotine delivery on smoking rate; (2) effects of smoking on measures of arousal, response suppression, and excitation/inhibition balance; (3) psychophysiological effects of smoking under conditions of stress and mild sensory isolation; (4) effects of smoking on vigilance performance; and (5) effects of smoking on acquisition and retention in verbal learning tasks add support to the arousal modulation rather than the addiction theory of smoking maintenance. Subjects of the first experiment, for instance, smoked only three brands of anonymized cigarettes which did not differ in taste or draw resistance, but did differ in nicotine content (0.9, 1.1, 1.3, and 1.7 mg/cigarette). Subjects were told that they were participating in a research market exercise judging taste of three cigarette brands. Consumption, taste, and mood were recorded, and unused cigarettes were returned for credit as an incentive toward an equal number of the smoker's preferred brand. Both light and heavy smokers maintained a stable rate of consumption, unrelated to the cigarette brand's nicotine content. These smoking rates did not differ significantly from mean rates of preceding study days. The results from all five experiments demonstrate that smoking has beneficial effects in both the short and long term, such as in coping with extremes of arousal or in enhancing long-term memory.

80–1778. McKillip, J.; Vierke, S. **College Smokers: Worried, Sick But Still Puffing.** *Journal of the American College Health Association* 28(5): 280-282, April 1980.

This study reports results of a cross-sectional survey of college students (n = 370) at a large midwestern university concerning cigarette smoking, other health-related personal habits, and use of a university health service. On the average, smokers reported smoking slightly over half a pack a day (17 percent smoking ≥ 1 pack/day). Smokers were slightly older than the average respondent (median age 20.7 year) and the amount smoked was reliably related to age ($p<0.01$). When asked if they were "worried" if smoking would affect their health, 25 percent said "no" or "a little," 45 percent reported being somewhat worried, and 30 percent reported being greatly worried. Only 22 percent had not stopped smoking in the past, while 43 percent reported having quit once or twice, and 36 percent having quit three or more times. Since 75 percent were at least somewhat worried about the effect of smoking on their health, and 78 percent had tried to quit at least once, it appears that these college smokers had the desire to quit, but lacked effective techniques. Those who reported having quit smoking were about the same age as the total sample (median age 20.4). Some 19 percent had quit within the last year, 17 percent between 1 and 2 years ago, and 65 percent more than 2 years ago. About one-third (34 percent) of quitters reported living with a smoker. Those who never smoked were slightly younger than the general sample (median age 20.1 years), and 25 percent of this group reported living with a smoker. Self-reported illness was related to alcohol and coffee consumption, use of the university health service, and smoking status. Smokers rated themselves as significantly less healthy than nonsmokers, with quitters somewhat less healthy than those who never smoked ($p<0.03$). Controlling for sex and class, smoking status was reliably related to self-reported health service use ($p<0.05$). Seventy-one percent of those who never smoked did not use the student health service for illness or injury while this percentage was 66 and 58 percent for ex-smokers and smokers respectively. Only 12 percent of those who never smoked used the health service two or more times, while 18 percent of the ex-smokers and 26 percent of the smokers did so. (Auth. Abs. Mod.)

80–1779. Merbaum, M.; Avimier, R.; Goldberg, J. **The Relationship Between Aversion, Group Training and Vomiting in the Reduction of Smoking Behavior.** *Addictive Behaviors* 4(3): 279-285, 1979.

The effectiveness of strong aversion joined with various group training procedures was compared with strong aversion and mild aversion treatments in the reduction of smoking behavior. Fifty-two male and female smokers were assigned to strong aversion plus self-control group, strong aversion plus temptation control group, strong aversion, and mild aversion groups. In addition, the effect of vomiting during the aversive training was related to followup cigarette consumption rates. In general, these data suggest that strong aversion alone is as effective as a combination of strong aversion plus self-control training. Vomiting during treatment appears to be related to lowered cigarette consumption. (Auth. Abs.)

80–1780. Mills, I. H. **Coping Mechanisms in the Brain.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 1-11.

In this chapter, the arousal mechanism, interaction between arousal and depression, purposely stimulated arousal, and drugs and the arousal level are discussed in terms of everyday life. It is stated that many people are aware that stimulants in tea and coffee facilitate mental tasks, and that some individuals are aware that smoking serves the same purpose.

80–1781. Myrsten, A.-L.; Andersson, K. **Effects of Cigarette Smoking on Human Performance.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 156-167.

Results obtained in experimental studies on the effects of tobacco smoking on physiological and psychological functions are presented. Research was conducted on (1) immediate effects of smoking on sensorimotor and cognitive functions, (2) interactions of smoking with alcohol, and (3) symptoms and reactions during smoking deprivation. Habitual, but not heavy, smokers were used as subjects under smoking and no-smoking conditions. In studies on the effect of smoking on sensorimotor functions it was found that the stimulant effect of smoking raised the general arousal (measured primarily by changes in heart rate) to a level where the subjects maintained better standards of efficiency. In examining whether nicotine administered through tobacco smoking would influence human learning, there appeared to be adverse effects on short-term memory, but smokers had better recall in the long term. In tests with reaction-time and arithmetic tasks, smoking counteracted the depressant effects of alcohol. A sudden break in a long-lasting smoking habit induced pronounced changes and disturbances in both physiological and psychological functions. It was concluded that smoking induces an increase in physiological arousal which is often accompanied by changes in mental efficiency. Subjects claimed that smoking made them more alert and at the same time relaxed.

80–1782. National Institute on Drug Abuse. **Technical Review on Cigarette Smoking as an Addiction.** Final Report of the

Task Force Meeting of the National Institute on Drug Abuse, Division of Research, August 23-24, 1979, Rockville, Maryland, September 18, 1979, 17 pp.

This report summarizes the contents of the National Institute on Drug Abuse-sponsored task force meeting on cigarette smoking as an addiction. The purpose of this meeting was to evaluate the scientific evidence that exists for and against cigarette smoking as an addiction, and to use this evidence to arrive at a consensus on this question. An agenda and list of participants is included.

80–1783. Pertschuk, M. J.; Pomerleau, O. F.; Adkins, D.; Hirsh, C. **Smoking Cessation: The Psychological Costs.** *Addictive Behaviors* 4(4): 345-348, 1979.

To assess the psychological effects of smoking cessation, 21 subjects in a smoking treatment program were evaluated at the beginning of therapy and 4 months later. Mean subject age was 40 years, mean number of years smoking was 21.5 years, and mean smoking rate was 34.7 cigarettes/day. The treatment schedule consisted of 8 weekly group sessions and 5 followup meetings. Smoking was gradually reduced through a variety of stimulus control and contingency management techniques. No differences were observed in reported use of mental health facilities, psychotropic medications, or alcohol. A statistically ($p<0.005$) but not clinically significant weight increase was observed. It is concluded that within some contexts smoking cessation need not be as stressful as popularly depicted. (Auth. Abs. Mod.)

80–1784. Poulton, P. **The Combination of Smoking With Psychological and Physiological Stress.** *Ergonomics* 20(6): 665-670, 1977.

The combination of smoking cigarettes and the anticipation of a written examination was investigated in 10 teenage girls. Both stresses alone increased pulse rate ($p<0.005$ for both). The effect of the combined stresses was found to be additive. In a second experiment a step test was performed by eight teenage girls with and without previous smoking. The increase in the respiration rate was found to be greater after previous smoking than without ($p<0.05$). (Auth. Abs. Mod.)

80–1785. Rawbone, R. G.; Murphy, K.; Tate, M. E.; Kane, S. J. **The Analysis of Smoking Parameters: Inhalation and Absorption of Tobacco Smoke in Studies of Human Smoking Behaviour.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 171-194.

The techniques that may be used to determine how people smoke and what constitutes smoke uptake and retention, and the relationship between them, are discussed. The results of experiments to study differences between habitual middle-tar and low-tar smokers are described, as well as the effects of switching between middle- and low-tar products. Various smoking parameters are measured, including cigarette characteristics, puff parameters, inhalation, and carbon monoxide (CO) in expired air. It was found that there was no correlation between the dose of tobacco smoke presented to the smoker (estimated from butt nicotine analysis) and the amount of smoke absorbed by the smoker (measured by increments in alveolar CO levels). A significant correlation ($p<0.05$) was found between the amount of smoke absorbed by the smoker and the smoke exposure index derived from the volume and time periods of inhalation following the puffing of a cigarette. No demonstrable differences in inhalation patterns were found between habitual smokers of middle- and low-tar cigarettes. Some evidence was found in switching studies that smokers adjust their smoking to maintain a constant dose of cigarette smoke. The smoker can and may modify smoking parameters and hence modify the quality and/or the quantity of smoke when smoking a particular product.

80–1786. Redican, K. J.; Olsen, L. K.; Stone, D. B.; Wilson, R. W. **Cigarette Smoking Attitudes of Lower Socioeconomic Sixth Grade Students.** *Journal of Drug Education* 9(1): 55-65, 1979.

Little research has been done to assess the cigarette smoking attitudes of lower socioeconomic status black students. In addition, there is a lack of information with respect to assessing the impact of health education programs on lower socioeconomic groups. A 9-week study was conducted to determine the effects of the School Health Curriculum Project Heart Unit in three lower socioeconomic elementary schools in a midwestern and eastern metropolitan area. All students involved in the study completed the attitude inventory. Both a pretest/posttest design and a Solomon Three Group Design were used to assess the cigarette smoking attitudes of lower socioeconomic sixth grade students after exposure to a special health program. The findings revealed that students exposed to the special health program developed more positive attitudes toward not smoking than students receiving their regular health instruction. (Auth. Abs. Mod.)

80–1787. Schaer, M. **Prinzipien und Probleme der Evaluation von Resultaten der Gesundheitserziehung. [Principles and Problems of Evaluation of Health Education Results.]** *Sozial- und Praeventivmedizin* 23(2): 101-103, June 1978, German.

The possibilities and problems of a meaningful evaluation of the short- and long-term effects of health education programs are discussed. It is presently not known how effective the usual methods of educational communication are in modifying unhealthy behavior and habits, e.g., smoking. Most health education programs do not make any provision for the scientific measurement of their success. Such measurements require long-term observation of comparable populations. Illustrations of the problems inherent in health education evaluation are provided. In the so-called Ruti study, 60 students in each of two small Swiss industrial cities were surveyed at the beginning of their studies (at the age of 16) and 3 years later, toward the end of school. Within the 3-year period, the students in one of the cities (Ruti) were subjected to an extensive health education program consisting of 36 lectures and colloquia, while the students in the other city served as controls. At the end of the 3-year period, 78 percent of the controls were smoking compared to 42 percent of the Ruti students. Of the smokers, 24 percent of the controls vs. 7 percent of the Ruti students smoked ≥ 20 cigarettes/day. Five years after the program ended, the number of smokers in the control group had decreased to 66 percent and in the Ruti group to 39 percent. However, the number of cigarettes consumed daily had increased significantly from 11 to 22 in the control group and from 5 to 20 in the Ruti group.

80–1788. Schinke, S. P.; Blythe, B. J.; Doueck, H. J. **Reducing Cigarette Smoking: Evaluation of a Multifaceted Interventive Program.** *Behavioral Engineering* 4(4): 107-112, 1978.

This study, focusing on a community-based target group, examined the efficacy of a multifaceted interventive package in reducing cigarette smoking. At an initial assessment session, eight subjects began monitoring daily cigarette-urge and cigarette-smoking levels. Six experimental condition subjects (seven females and one male who had been smoking from 1 to 29 years) participated in eight interventive sessions (which included rapid smoking, relaxation techniques, and assertive training); two control condition subjects continued reporting daily cigarette urges and cigarettes smoked. After the interventive period, and at 1-, 3-, and 6-month followup checks, experimental condition mean scores showed decrements for both cigarette urges and cigarettes smoked; control condition mean scores remained at preinterventive levels. Findings support the effectiveness of the interventive package in reducing and maintaining reduced cigarette smoking. Results support other research examining a multifaceted approach to smoking reduction. (Auth. Abs. Mod.)

80–1789. Stone, J. D.; Breidenbach, S. T.; Heimstra, N. W. **Annoyance Response of Nonsmokers to Cigarette Smoke.** *Perceptual and Motor Skills* 49(3): 907-916, 1979.

In three experiments, nonsmoking subjects rated their annoyance with task environments while either exposed or not exposed to ambient tobacco smoke. In experiments one and two, subjects rated annoyance with the environment while exposed to three intensities of noise, and in experiment three, subjects rated annoyance while performing multiplication problems under conditions of high- and low-task motivation. The source of the ambient smoke in experiment one was a smoke pump. For experiments two and three, the source of the smoke was confederate subjects. No differences in annoyance were obtained in experiments one and two as a function of exposure to smoke. Experiment three showed that nonsmokers' annoyance to a task environment is dependent upon task motivation and exposure to ambient smoke. In experiment three, nonsmokers exposed to smoke were more annoyed than those not exposed under low motivating conditions. When nonsmokers were highly motivated in the task environment, those exposed to smoke were significantly ($p<0.001$) less annoyed than those not exposed. (Auth. Abs.)

80–1790. Sutton, S. R.; Russell, M.A.H.; Feyerabend, C.; Saloojee, Y. **Smokers' Response to Dilution of Smoke by Ventilated Cigarette Holders.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences.* Edinburgh, Churchill Livingstone, 1978, pp. 330-335.

The study reported here was designed to examine the extent to which smokers who were not trying to stop smoking would compensate for the dilution of smoke a withdrawal aid produced. The aid consisted of four cigarette holders which produced a 20-80 percent progressive cigarette smoke dilution from holder one to holder four. Study subjects included 16 women and 2 men aged 23-55 years who smoked an average of 30 cigarettes/day. The subjects attended six times over a 3-week period. On the first day all subjects smoked normally without using a holder. They then spent 2 days on one holder followed by 2 days on the other, with the order of holders balanced. They continued using their second holder for a further 5 days before switching again to their first holder for the following 7 days. For the remaining 5 days they reverted to smoking without a holder. Subjects smoked their usual brand of cigarette and were instructed throughout to smoke as much or as little as they felt inclined. Blood samples were analyzed for carboxyhemoglobin and for plasma nicotine. Mean plasma nicotine levels with holder one were significantly ($p<0.01$) lower than with no holder. The changes (as compared with no holder) in levels from 2 to 7 days were not significant for either holder one or two, but the differences between holders were significant at both times ($p<0.01$ and $p<0.001$, respectively). By monitoring observed and expected carboxyhemoglobin levels for evidence of compensation, it was found that subjects partially compensated, obtaining levels that were about 60 percent of their no-holder levels instead of the expected 42 percent.

80–1791. Tagliacozzo, R. **Smokers' Self-Categorization and the Reduction of Cognitive Dissonance.** *Addictive Behaviors* 4(4): 393-399, 1979.

Sixty percent of a sample of smokers ($n = 155$) who smoked between one and two packages of cigarettes per day considered themselves "moderate" smokers, while the remaining 40 percent considered themselves "heavy" smokers. This study compares the answers of these two groups of smokers to questions concerning the health risk of smoking. The results suggest that those respondents who assigned themselves to the lower risk category, i.e., "moderate," were more aware of the pathological, long-term effects of smoking than the smokers who classified themselves as "heavy" smokers. The smokers' tendency to minimize the extent of their smoking, as well as the avoidance of words which have threatening associations for the smoker, were interpreted as a means of reducing cognitive dissonance. (Auth. Abs.)

80–1792. Tagliacozzo, R.; Vaughn, S. **Women's Smoking Trends and Awareness of Health Risk.** *Preventive Medicine* 9(3): 384-387, May 1980.

National data on smoking cessation rates indicate that female smokers may have more difficulty than male smokers in quitting smoking. To test the hypothesis that awareness of risk of smoking and personal vulnerability may be not as strong in female as in male smokers, a series of telephone interviews dealing with smokers' awareness and knowledge of the recent Surgeon General's Report on Smoking and Health, and their perception of and attitudes toward personal risk was conducted. Of the 276 respondents, aged 18-72, 47.8 percent were male and 52.2 percent were female. Among these respondents were ≤ 10 cigarettes/day smokers (28 percent), 11-20 cigarettes/day smokers (39 percent), and >20 cigarettes/day smokers (33 percent). The results indicated no marked sex difference in awareness of the Surgeon General's Report, but showed that significantly more female (63.6 percent) than male (49.1 percent) smokers worried about what they heard and read about the risk of smoking ($p<0.05$). A larger percentage of female smokers attributed a greater health risk from smoking to men than to women (i.e., 40.6 vs. 18.8 percent), thus suggesting that women's feelings of personal vulnerability may be weaker than men's. (Auth. Abs. Mod.)

80–1793. Wesnes, K.; Warburton, D. M. **The Effects of Cigarette Smoking and Nicotine Tablets Upon Human Attention.** In: Thornton, R. E. (Editor). *Smoking Behaviour. Physiological and Psychological Influences*. Edinburgh, Churchill Livingstone, 1978, pp. 131-147.

A number of studies designed to determine the effects of nicotine upon human attention are presented in this paper. It was generally found that smokers performed more efficiently in the experimental situations with cigarettes or nicotine tablets than without. When comparing smokers with either nonsmokers or smokers of non-nicotine cigarettes, it was found that smoking significantly improved visual as well as auditory vigilance ($p<0.05$ and $p<0.0005$, respectively). In experiments with cigarettes containing 0.28, 0.7, and 1.65 mg nicotine, the only significant enhancement of performance was found between the low- and medium-nicotine cigarettes ($p<0.05$). Evidence for nicotine being the active agent in cigarette smoking was obtained from experiments in which the drug was administered orally. It was demonstrated that nicotine tablets enhanced performance relative to control. Nicotine also significantly reduced the Stroop effect ($p<0.05$) and enhanced information processing.

See also, **80–1625, 80–1705, 80–1739, 80–1826**

SMOKING CESSATION METHODS

80–1794. Abrams, D. B.; Wilson, G. T. **Self-Monitoring and Reactivity in the Modification of Cigarette Smoking.** *Journal of Consulting and Clinical Psychology* 47(2): 243-251, 1979.

The reactive effects of self-monitoring as a function of varying the specific nature of the target behavior and the perceived negative consequences of the behavior were investigated with chronic smokers as subjects. Forty subjects were assigned to one of the following four conditions from stratified blocks based on initial smoking rates: (a) self-monitoring nicotine plus health hazard information, (b) self-monitoring nicotine with no health hazard information, (c) self-monitoring cigarettes plus health information, and (d) self-monitoring cigarettes with no health information. Subjects self-monitored during a 4-week nondemand phase, during a 4-week treatment phase, or until they quit smoking. The two nicotine self-monitoring groups showed greater reactivity. There were no differences among groups as a function of exposure to health hazard information. Results are discussed in relation to models of self-control and previous investigations of other important parameters of reactive self-monitoring. (Auth. Abs.)

80–1795. Chen, J.Y.P. **Treatment of Cigarette Smoking by Auricular Acupuncture: A Report of 184 Cases.** *American Journal of Acupuncture* 7(3): 229-234, July-September 1979.

A standardized technique utilizing press needles implanted at the auricular "Shenmen" and "Lung" points for cessation of smoking is described. The efficiency of this technique is appraised in 184 cases (104 men, 80 women) in terms of quitting success for at least 6 months. Of the 184 cases treated with this method, 82 percent completely stopped smoking, 8 percent significantly reduced the number of cigarettes smoked, and 10 percent were unaffected. Auricular acupuncture as described in this report has proved a simple, safe, economic, and rapidly effective method to kick the smoking habit and can conceivably be employed in massive stop-smoking programs. (Auth. Abs. Mod.)

80–1796. Gmuer, M. **Die Raucherbehandlung des Handauflegers Hermano. 5-Jahres-Katamnese. [Treatment of Smokers by the Healer Hermano. 5-Year-Followup.]** *Schweizerische Medizinische Wochenschrift* 109(49): 1967-1973, December 22, 1979, German.

A study of a random sample of 532 cigarette smokers was made among the persons treated by the chirothesist Hermano in Seon, Switzerland, who had cured smokers of their nicotine addiction. They were examined immediately before the treatment ritual, then 4 months, 1 year, and 5 years after the treatment. The patients of the healer were chosen from all the strata of the population. They were heavy smokers with an average smoking rate of 31 cigarettes/day for males and 24 cigarettes/day for females. Four months after the treatment 40 percent of the treated persons, 12 months after the treatment 32.5 percent, and 5 years after it 20 percent of the treated persons were nonsmokers. Another 3.7 percent did smoke but only sporadically for up to 5 years. Two-thirds of the patients mentioned a suggestive effect which they consciously registered or which they remembered in the sense that their urge to smoke had diminished partly or disappeared completely due to the treatment. There were only a few cases of partial success (moderation of smoking or transitional stoppage). The methodical control of the followup study showed that recidivists showed a lesser readiness to answer during the catamnestic examinations. It is therefore necessary for all followup studies as well as for comparative therapeutical research to take into consideration the degree of completeness of the followup examination. (Auth. Abs. Mod.)

80–1797. Hyner, G. C. **Relaxation as Principal Treatment for Excessive Cigarette Use and Caffeine Ingestion by a College Female.** *Psychological Reports* 45: 531-534, October 1979.

A female graduate student with diagnosed tachycardia was the subject in an experiment to reduce consumption of caffeine and use of cigarettes. A relaxation procedure was used as the principal treatment. Reinforcement cards for nonsmoking and elimination of caffeine were also used. The subject was successful in reducing her caffeine intake and completely extinguished use of cigarettes. She reported that she was still practicing the relaxation procedure 1 year later. Apparently these procedures can be helpful in modifying use of cigarettes and caffeine. (Auth. Abs.)

80–1798. Lagrue, G.; Poupy, J. L.; Grillot, A.; Ansquer, J. C. **Acupuncture Anti-Tabagique. Resultats a Court Terme d'une Etude Comparative Menee a Double Insu. [Acupuncture Against Smoking. Short-Term Results of a Comparative Double-Blind Study.]** *Nouvelle Presse Medicale* 9(13): 966, March 15, 1980, French.

The results of a double-blind, experimental antismoking therapy study, using selected antismoking and placebo acupuncture points are reported. The acupuncture treatment was administered to 154 subjects. Each subject was punctured twice at 1-week intervals by inserting needles in the same location on each side of the face and leaving them in place for 25-30 min. The results are based on answers to a questionnaire filled out during the second treatment. After a single treatment, no difference appeared in the percentage of successful treatments as between the acupuncture of the standard antismoking and placebo points. Total cessation was obtained in 44 and 40 percent of the cases, and an 80 percent decrease was recorded in 18 and 23 percent of the cases after the first acupuncture of the actual antismoking and placebo points, respectively. The apparent failure of the treatment may be interpreted as a possible result of insufficient treatment, or the real absence of acupuncture effect, or as the result of insufficient experience of the practicing physician.

80–1799. Pederson, L. L.; Scrimgeour, W. G.; Lefcoe, N. M. **Variables of Hypnosis Which Are Related to Success in a Smoking Withdrawal Program.** *International Journal of Clinical and Experimental Hypnosis* 27(1): 14-20, 1979.

Sixty-five habitual smokers (28 men and 37 women, with a mean baseline smoking rate of 28.2 cigarettes/day) were randomly assigned to one of four groups: live-hypnosis plus counseling, videotape-hypnosis plus counseling, relaxation-hypnosis plus counseling, and counseling alone. The content and mode of presentation of the hypnosis session varied among the first three groups. At 6 months posttreatment, the live-hypnosis plus counseling group contained significantly more abstainers (53 percent abstinence rate) than the other three groups. The importance of the specific content of the hypnosis session and the presence of the hypnotherapist for the effectiveness of the procedure is discussed. (Auth. Abs. Mod.)

80–1800. Poole, A. D.; Stumbles, C. **Smoking Control: The Effectiveness of a Stop-Smoking Programme in a Community Health Centre Setting.** *Australian Psychologist* 14(2): 175-182, July 1979.

Persons under 40 years of age who indicated, at the Busselton Population Survey, that they smoked ≥ 15 cigarettes/day were invited to participate in a stop-smoking program involving rapid smoking. Fourteen persons (eight men and six women), all of whom met specified health criteria, completed the program. All underwent seven rapid smoking sessions which were carried out in groups of two to three subjects. Eight subjects were instructed to remain abstinent between sessions while six were permitted to smoke but instructed to do so rapidly. The instructions to refrain from smoking between sessions produced a significantly ($p<0.01$) greater number of subjects being abstinent at the end of treatment and 1 month following the program. The difference between the groups was no longer significant at 3 months. Overall, only three subjects, all from the group told to abstain between sessions, were not smoking 3 months after treatment. The remaining subjects were smoking, on average, 58.3 percent of their base rate daily consumption at the 3-month followup. (Auth. Abs.)

80–1801. Seibel, B. L. **An Evaluation of a Therapist-Administered Bibliotherapy and Spouse Smoking Habits on Smoking Behavior.** *Journal of Drug Education* 9(4): 327-335, 1979.

This study attempted to evaluate a readily available comprehensive bibliotherapy smoking cessation program and the impact of a spouse's smoking or nonsmoking behavior on the individual who attempts to stop smoking. The success rate for the therapist-administered program was 28.57 percent (two out of seven subjects) at the end of the program and 14.29 percent at the 3-month followup. Contrary to expectations, both subjects who stopped smoking had smoking spouses which could suggest that motivation is an important variable in smoking cessation. (Auth. Abs. Mod.)

80–1802. Shiffman, S. M.; Jarvik, M. E. **Withdrawal Symptoms: First Week is Hardest.** *World Smoking and Health* 5(1): 16-21, Spring 1980.

In a study of smoking cessation withdrawal symptoms, 40 participants in a cessation clinic completed questionnaires about four symptom clusters: alertness, craving, physical symptoms, and psychological symptoms. The questionnaires were completed daily for the first 2 weeks of abstinence. Results showed a strong diurnal variation in craving for cigarettes, with craving lowest early in the morning and rising to a peak around 7 P.M. The onset of the smoking withdrawal syndrome was rapid, with many of the symptoms appearing within 2 hours of withdrawal. All symptom clusters showed a similar pattern of decreasing severity in the first week followed by a leveling off or increase in the second week. The participants were divided into two groups: totally abstinent smokers for the entire period of the study, and partially abstinent smokers who reduced their smoking by an average of 60 percent. The two groups reported similar levels of symptom severity, but the totally abstinent group experienced a notable decrease in symptoms during the first week while the partially abstinent group maintained their withdrawal symptoms. This confirms previous results that the gradual reduction method of smoking cessation is not the most effective method because it precipitates a chronic state of withdrawal which will lead to relapse and return to baseline rates of smoking.

80–1803. Tongas, P. N. **The Kaiser-Permanente Smoking Control Program: Its Purpose and Implications for an HMO.** *Professional Psychology* 10(4): 409-418, August 1979.

Over the last 5 years, a number of specialized psychological services have been developed in the Los Angeles Kaiser-Permanente Medical Care Program designed to fulfill specific health care needs of Kaiser members. One of these services, the Anti-Smoking Service, is described in detail from an accountability point of view, in which specific goals are defined and implemented and their degree of attainment is evaluated. Baseline data on goals pertaining to effectiveness, efficiency, and cost-effectiveness are presented. The best procedure, involving 5 consecutive treatment sessions (each treatment including aversive and covert conditioning and behavior therapy) and 14 maintenance sessions over a 12-month period, produced a 62 percent complete abstinence at 24-month followup. The cost per session per patient was 1.60 dollars, and for the whole treatment and maintenance package per patient, it was 22.48 dollars. It is concluded that psychologists, because of their particular training and skills, can make important innovative contributions to the prevention of illness and the maintenance of health in health maintenance organizations. (Auth. Abs. Mod.)

80–1804. Wetherall, C. F. **Quit.** Minneapolis, Charles F. Wetherall and Co., Inc., 1979, 206 pp.

This cigarette pack-sized book offers a self-help, five-step method to kick the smoking habit. The steps are (1) determine why you want to quit, (2) rank the cigarettes you smoke for their importance in your lifestyle, (3) gradually reduce your smoking, (4) quit, and (5) guard against a resumption of smoking.

80–1805. World Smoking and Health. **How Successful Are Five-Day Plans for Smoking Cessation?** *World Smoking and Health* 5(1): 42, Spring 1980.

A 2-year followup was made of 158 persons who completed Five-Day Plans in England in 1975. Of the subjects, 83 percent stopped smoking during the 5 days. After 1 month, 64 percent of those who stopped (53 percent of the subjects) were still not smoking, a figure which dropped to 37 percent (31 percent of the subjects) after 1 year. After 2 years, there was a further drop to 33 percent of those who had stopped smoking (27 percent of the subjects). Stress, social pressures, lack of willpower, or increasing weight were given as the major reasons by those who started smoking again.

See also, **80–1773, 80–1783, 80–1790**

TOBACCO ADDITIVES

80–1806. Baldwin, A. D. **Pipe for Drawing Smoke Through and Extracting Juice From a Piece of Fruit.** United States Patent No. 4,181,136, January 1, 1980, 3 pp.

A pipe for smoking tobacco and similar substances while sucking a piece of fruit is described. The device is comprised of (1) a hollow first tube having a sharpened edge at one end and a sleeve at the other end, the sharpened edge capable of carving a substantially L-shaped passageway within the piece of fruit by a vertical first insertion meeting with a horizontal second insertion; (2) a hollow second tube having a larger inside diameter at one end, a smaller inside diameter at the other end, and an internal lip, the smaller diameter slidable over the sleeve of the first tube, and the second tube resting within the vertical insertion smaller diameter first; (3) a screen onto which tobacco is placed which is positionable across the cross section of the second tube resting on the lip and where suction applied to the sleeved end of the first tube causes smoke from the ignited tobacco to flow through the second tube, thereby contacting the pulp and the fruit's juice which accompany the smoke leaving the fruit via the first tube; and (4) means for connecting the first and second tubes when the second tube is positioned on the sleeve.

80–1807. Heim, E.; Imbery, D.; Kliemann, B. **Additive for Smoking Tobacco Products, Filter Elements Thereof and Process for the Preparation Thereof.** United States Patent No. 4,193,412, March 18, 1980, 8 pp.

An additive for smoking tobacco products and their filter elements consists of a mixture of at least two highly dispersed metal oxides or metal oxyhydrates of aluminum, calcium, magnesium, silicon, and titanium, or a mixture of different forms of the same metal oxide or metal oxyhydrate. The maximum average particle size in the additive is approximately 25 μm. The mixture exhibits liquid-like properties anomalous for a mixture of solids, and specific properties of the mixture, such as bulk density, surface area, and flowability, whereby the absorption capacity for toxic substances in the tobacco smoke is synergistically increased. (Auth. Abs. Mod.)

80–1808. Light, K. K.; Shuster, E. J.; Vinals, J. F.; Vock, M. H. **Flavoring With a Tricyclic Alcohol.** United States Patent No. 4,139,650, February 13, 1979, 12 pp.

A tricyclic alcohol which can be used in a flavoring composition for tobacco products ranging from cigarette to chewing tobacco is described. When added to cigarette tobacco in concentrations ranging from 10 to 20 ppm, these alcohols (e.g., octahydro-7A,8,8-trimethyl-1,5-methano-1H-inden-1-ol) result in a more aromatic, sweet bitter, green, richer, slightly less harsh, and more tobacco-like flavor than nonadditive containing control cigarettes.

80–1809. Wilson, R. A.; Mookherjee, B. D.; Hruza, A. S.; Vock, M. H.; Frederick, L. S.; Vinals, J. F. **Process for Augmenting or Enhancing the Flavor of Tobacco Using 1-(3-Methylthio)butyryl)-2,6,6-Trimethyl-Cyclohexene and the 1,3-Cyclohexadiene Analog Thereof.** United States Patent No. 4,209,025, June 24, 1980, 20 pp.

Described are methods for augmenting or enhancing the aroma or taste of a tobacco or tobacco flavoring composition by the addition of a substantially pure, synthetically produced 1-(3-(methylthio)butyryl)-2,6,6-trimethyl-cyclohexene or its 1,3-cyclohexadiene analog. Experimental cigarettes containing 500 or 100 ppm of the former are found to have a rich sweet flavor effect with lingering sweetness with fruity, blackcurrant-like, raspberry-like, dried fruit-like, and damascenone-like notes prior to smoking, and to have more tobacco-like, fruity-sweet, and damascenone-like notes, and more body than the control cigarettes on smoking.

80–1810. Withycombe, D. A.; Mookherjee, B. D.; Vock, M. H.; Vinals, J. F. **Isobutyl Substituted Heterocyclic Compounds for Augmenting or Enhancing the Organoleptic Properties of Smoking Tobaccos and Smoking Tobacco Articles.** United States Patent No. 4,207,905, June 17, 1980, 20 pp.

Isobutyl-substituted heterocyclic compounds for augmenting the organoleptic properties of smoking tobaccos are described. Included in the compound mixtures discussed are 2,4,6-triisobutyl-1,3,5-trioxane and 2-isobutyl dialkyloxazolines. In one tobacco formulation test, 500 or 1,000 ppm of 4-ethyl-2-isobutyl-5-methyl-3-oxazoline was added to experimental cigarettes. These cigarettes had chocolate-like, nutty, green, and burnt roasted-like aroma notes, and were more tobacco-like on smoking, as compared with control (no additive-containing) cigarettes. The tobacco of the experimental cigarettes had chocolate-like, nutty, and green notes prior to smoking.

80–1811. Zborowski, J.; Procak, M.; Obroniecka, L.; Kuter, E. **Ocena Technologicznej Przydatnosci Niektorych Polioli i ich Mieszanin w Procesie Uszlachetniania Krajowych Tytoni. [Evaluation of the Technological Usefulness of Certain Polyols and Their Mixtures in Improving the Quality of Native Tobacco.]** *Biuletyn Centralnego Laboratorium Przemyslu Tytoniowego* (3/4): 29-44, 1979, Polish.

The specific volume, sorption, and desorption of cut native cigarette tobacco impregnated with aqueous solutions of different glycols or their mixtures and desorption of the cigarettes made from the tobacco were determined. Statistical analysis of the data provided the basis for the evaluation of the economic effect of polyols as humectants in tobacco compositions. Addition of a mixture of 2 percent of a

diethylene glycol and 1,2-propanediol in equal parts had the best economic effect. Other mixtures were also rated. The 2-6 percent doses of the glycol compositions in tobacco appeared to be the optimum which would permit thermal processing of tobacco at temperatures up to 130°C. Desorption tests of cigarettes made from glycol-impregnated tobacco confirmed the beneficial effect of glycols in maintaining a given moisture content of the cigarettes during storage.

***See also,* 80–1820**

TOBACCO MANUFACTURING AND PROCESSING

80–1812. Albo, J.-P.; Deletang, J.; Chouteau, J. **Influence de Certaines Caracteristiques Propres au Tabac sur la Production de Goudrons. [Influence of Some Characteristics Proper to Tobacco on Tar Production.]** *Annales du Tabac Section 2* (13): 133-147, 1976, French.

Removing samples from three leaf levels (lower, median, and upper) of two tobacco varieties, P.B. and Burley, from the curing barn at three distinct stages of curing (i.e., harvest, yellowing, end of curing), a sampling with a wide range of physical, chemical, and smoking characteristics is obtained. The anhydrous condensates of these tobaccos can be predicted through an equation derived from the following data (in decreasing order of importance): ash hydrosoluble alkalinity, total nitrogen content, and weight of the cigarettes. This regression equation accounts for 90 percent of the variations of anhydrous condensates. Experimental data also verified that by decreasing the total nitrogen content and increasing the hydrosoluble alkalinity of the upper leaves, it is possible to markedly reduce anhydrous condensate production. (Auth. Abs.)

80–1813. Epstein, S. S.; Lijinsky, W. **Smoke Processing.** United States Patent No. 4,156,431, May 29, 1979, 7 pp.

This smoke filter contains silicic acid for removing secondary amines and a second material capable of removing nitrosamines. The second material is preferably an unsulfonated cross-linked polystyrene. The silicic acid and unsulfonated cross-linked polystyrene are mixed together to form a granular porous mass having 90-50 percent by weight silicic acid and 10-50 percent by weight unsulfonated cross-linked polystyrene. About 3-45 percent by weight of the mixture of a nonreactive binder is incorporated in the mass. Small amounts of the filter material of this invention are effective in removing all secondary amines and nitrosamines from the amount of tobacco ordinarily used in a commercial cigarette. The filter material is useful for removing basic nitrogen compounds from a variety of differently constituted smokes such as smoke generated by cigarettes burning, cigar burning, pipe smoking, and food processing.

80–1814. Gumushan, O. H. **Tobacco Refining Process.** United States Patent No. 4,183,364, January 15, 1980, 4 pp.

A process for refining tobacco for cigarettes is described which leaves the end product free of resin and tar and virtually free of nicotine, and which affords economics in the processing procedure. The tobacco is initially steeped in a hot solution of potassium metabisulfite ($K_2S_2O_5$) or potassium sulfate and then boiled in a solution of potassium sulfate and potassium nitrate. The tobacco is then air dried and sprayed with flavoring material. Tobacco processed in this manner was found to have no tar or resin content and between 0.01 and 0.02 percent nicotine. (Auth. Abs. Mod.)

80–1815. Kelley, S. A. **Water Pipe or Bong.** United States Patent No. 4,183,365, January 15, 1980, 5 pp.

A water pipe or bong for smoking tobacco is comprised of a base to which one end of an upright, hollow cylinder is affixed leaving an open end or mouthpiece so that smoke can be inhaled by applying a suction action to the interior of the cylinder. In this design, a revolving member in the form of an annulus is axially aligned with the longitudinal centerline of the hollow cylinder with the cylinder being telescopingly received within the annulus and with the annulus being rotatably positioned around a medial portion of the cylinder. A plurality of radially spaced combustion chambers is formed within the annulus in which tobacco can be stored and combusted. The combustion chambers include an open end into which tobacco is charged and an outlet end through which smoke can flow. A passageway is formed between the outlet of the combustion chamber and the interior of the hollow cylinder so that air can be drawn into the combustion chamber, thereby burning the tobacco, so that smoke flows through the outlet into the interior of the chamber and into the smoker's mouth. (Auth. Abs. Mod.)

80–1816. Mohapatra, S. C.; Johnson, W. H. **Post-Harvest Physiology of Bright Leaf Tobacco. 1. Comparative Biochemical Changes During the Yellowing and Drying Phases of Curing.** *Tobacco International* 182(7): 87-89, April 4, 1980.

Analysis of several tobacco leaf constituents showed that each changed quantitatively during both yellowing and drying phases of bright leaf curing. Nearly 10 percent of the sugar, 9 percent of the amino acids, and 13 percent of the polyphenols found in the cured leaf were generated during the drying phase alone. These along with other changes indicate that the drying phase of bright leaf curing is not as inert as is generally believed. (Auth. Abs.)

80–1817. Rainer, N. B.; Feins, I. R. **Filter Material for Selective Removal of Aldehydes for Cigarette Smoke.** United States Patent No. 4,182,743, January 8, 1980, 7 pp.

A gas-permeable substrate (e.g., inorganic substances such as silica gel, alumina, and magnesia; or organic substances such as polyvinyl chloride, polymethacrylates, and polyolefins), particularly adapted for the selective removal of aldehydes from gases is comprised of a granular carrier containing concentrated hydrogen peroxide, water, and a hydrophilic stabilizer for the hydrogen peroxide. Granules of this mixture are used in cigarette filters. Using a filter of this example, acetaldehyde reduction in gas phase cigarette smoke was 78 percent. (Auth. Abs. Mod.)

80–1818. Riedesser, W. **Mouthpiece Paper for Cigarettes.** United States Patent No. 4,185,643, January 29, 1980, 3 pp.

In the preparation of synthetic mouthpiece paper for cigarettes which simulates cork, it is found that the similarity to cork is improved by applying a coating of lacquer over the entire surface of the paper. The paper has randomly distributed depressions formed by stamping the paper, which leaves it more dense in the depressed areas. The luster of the depressed portions of the paper is increased by the lacquer, while the matt luster of the remaining portions of the paper is retained. (Auth. Abs. Mod.)

80–1819. Schepartz, A. I.; Ellington, J. J.; Schlotzhauer, W. S. **Destruction of Precursors of Polynuclear Aromatic Hydrocarbons in Tobacco by Treatment With Ozone.** *Tobacco International* 182(10): 25-27, May 16, 1980.

The neutral lipid fraction of the tobacco leaf contains the major precursors of the polynuclear aromatic hydrocarbons (PAH) in cigarette smoke. A method was developed for the treatment of tobacco with ozone to effect decomposition of major components in the lipid fraction. Ozonization of tobacco in an aqueous or methanol suspension produced decreases in solanesol (C_{45} isoprenol) content from 86 to 100 percent; in unsaturated C_{18} acids from 81 to 97 percent; and in major sterols from 46 to 78 percent, depending on the tobacco used. These decreases, in turn, resulted in a reduction in the amount of the PAH fraction of the pyrolyzate of treated tobacco by 36 to 70 percent. Phenols and nicotine are also significantly reduced. These findings indicate that it should be feasible to ozonize tobacco on a large scale and utilize the treated tobacco in the production of cigarettes yielding decreased amounts of smoke PAH. (Auth. Abs.)

80–1820. Seehofer, F.; Kausch, E. **Removal of Nitric Oxide and Carbon Monoxide From Tobacco Smoke.** United States Patent No. 4,182,348, January 8, 1980, 3 pp.

A method for the catalytic removal of nitric oxide and carbon monoxide from tobacco smoke is described which consists of contacting the smoke with a compound of the formula $M_2M'RuO_6$, where M is a divalent metal, M′ is a trivalent rare earth metal, and Ru has a valence of 5, M and M′ being such that their ions are capable of forming a perovskite lattice with the Ru ions. The catalyst can be mixed with tobacco, or incorporated into the cigarette paper or filter. Optionally, suitable inorganic or organic adsorbing materials are desirable, e.g., carbon and calcium carbonate. (Auth. Abs.)

80–1821. Steinau, P. **Cigarette Filter.** United States Patent No. 4,182,350, January 8, 1980, 4 pp.

This patent describes a cigarette filter made of a fibrous adsorption agent which contains weakly hydrophilic polyolefin fibers made by a flash evaporation process. The filter was found to have excellent adsorption properties for tar and nicotine. With less than half the quantity of filter material used in a conventional cellulose acetate filter, the new filter had the same or better efficiency. (Auth. Abs. Mod.)

80–1822. Swanson, G. T. **Cigarette Filters.** United States Patent No. 4,175,571, November 27, 1979, 6 pp.

A cigarette filter includes a barrel, a mouthpiece at one end, and a cigarette-receptive tip at the other. A restricted orifice extends through a transverse wall within the barrel. An air inlet extends through the barrel wall at a location toward the mouthpiece from the transverse wall. Within the barrel is a stem that defines a passage for the flow of smoke, and the inlet also communicates with that passage. The stem includes a nose which is spaced from the outlet end of the orifice and defines a collection area for particulate matter included in the smoke. A system concept includes a combination of a number of such filters in which the orifice of each successive one is of a respectively decreased diameter and the air inlet is of a respectively increased diameter. The relationship of those diameters is such that each of the filters establishes a substantially constant impedance to the drawing of smoking and air through the mouthpiece. (Auth. Abs.)

80–1823. Tobacco International. **Ozoned Tobacco May Lead to Safer Smoke.** *Tobacco International* 182(7): 60, April 4, 1980.

Treating tobacco with ozone produced by an electrical discharge of oxygen was investigated as a possible means of reducing the lipid content of tobacco leaves. Ozonization decreased solanesol content from 44 to 100 percent, fatty

acids from 45 to 93 percent, and sterols from 21 to 83 percent. These decreases represent a reduction of 21 to 70 percent in the polyaromatic hydrocarbon content of cigarette smoke. Phenols and nicotine content were also significantly reduced.

See also, **80–1767, 80–1807, 80–1811**

TOBACCO ECONOMICS

80–1824. Ahmad, M. **Pakistan Tobacco: The Boom Continues.** *Tobacco Reporter* 107(7): 33-35, July 1980.

Cigarette production in Pakistan registered a 9.1 percent increase for the last quarter of 1979 compared to the same quarter a year earlier. Production of domestic leaf fell marginally short of industry requirements. The overall production picture and price pattern of cigarette-type tobacco during 1979 are presented. There was an official increase of 10 percent in minimum prices of various flue-cured grades. Government revenue from excise taxes on cigarettes surpassed 2.03 billion dollars. While exports of raw tobacco declined, manufactured tobacco exports increased. Domestically, the popularity of filter-tip cigarettes grew rapidly, a number of new brands were launched, a new cigarette factory was commissioned, and existing small units were modernized. A health warning inscription ordinance was deferred to September 1980, and the gradual shift to cigarette smoking from other modes of tobacco consumption continued.

80–1825. Leu, R. **The Effects of Cigarette Price and Anti-Smoking Publicity on Cigarette Consumption in Switzerland. (Letter).** *Revue d'Epidemiologie et de Sante Publique* 27: 359-362, 1979.

Time series of cigarette consumption per capita (population aged 15 and over) in Switzerland have been analyzed for the period 1954-1977. Multiple regression models in linear and nonlinear specification were used. The following variables were tested: real income per capita, changing smoking habits, cigarette price (tobacco tax), antismoking publicity, and lagged cigarette consumption. Linear model results are given and include these facts: (1) cigarette consumption can be effectively influenced by tobacco tax and antismoking campaigns, (2) smoking habits increased, on average, 3 percent/year, and (3) cigarette price and cigarette consumption are negatively related (-20.9821). It is suggested that radio and television antismoking publicity as well as tobacco taxes be increased.

80–1826. Schouten, M. **Tobacco Advertising Effects on the Consumption of Tobacco Products. A Report to the Canadian Council on Smoking and Health.** Canadian Council on Smoking and Health, Ottawa, Ontario, August 1978, 152 pp.

The topic of the effects of tobacco advertising on the consumption of tobacco products is highly emotional and partisan. This project has sought to compile and assess some of the reputable research of the past, and to suggest areas for future investigation. More research is required to clarify the tobacco advertising/consumption interaction to the satisfaction of government, industry, lobbyists, and consumers. Information is supplied on the health and economic consequences of smoking; cigarette consumption demographics; influences on smoking behavior; the structure of the tobacco industry; economics, themes, and effects of tobacco advertising; and tobacco advertising regulation. A 74-page appendix contains references and abstracts of selected research on tobacco advertising. (Auth. Abs. Mod.)

80–1827. Tobacco International. **High MH Residues Threaten Foreign Sales; Six-Point Containment Program Proposed.** *Tobacco International* 182(11): 43-44, May 30, 1980.

German cigarette manufacturers have been using less U.S. tobacco in their blends in order to keep maleic hydrazide (MH) levels down. The unofficial agreement between German public health authorities and the cigarette industry, which may become law in 1981 when current pesticide residue regulations are reviewed, is that the MH residual level in a finished tobacco product cannot exceed 80 ppm. A six-step program to chemically control suckers and to maintain acceptable MH residue levels is described.

80–1828. Tobacco International. **Leaf Exports Fell by 13 Percent in 1979.** *Tobacco International* 182(11): 46, May 30, 1980.

U.S. exports of unmanufactured leaf tobacco declined 13 percent from 1978 levels to 1.2 billion dollars in 1979. European market shipments were down 29 percent; however, some Asian country (Taiwan, Thailand, South Korea, Hong Kong) shipments rose 5 percent, offsetting shipment reductions to Japan and the Philippines (6 and 16 percent, respectively). Duty-paid leaf imports rose 12 percent from 1978 levels, to 17,000 tons. The import value rose 9 percent to 412 million dollars. Manufactured exports, mainly cigarettes, were 964 million, 26 percent above 1978 sales.

TOBACCO ECONOMICS

80-1829. Tobacco International. **Chewing Leads Off in U.S. Sales Gains.** *Tobacco International* 182(7): 64, 67, April 4, 1980.

Chewing tobacco production and sales surpassed the 100-million-lb mark in 1979, for the first time since 1946. Sales increased 9.3 percent in 1979, a gain of 8.6 million lbs. Snuff production and sales declined in 1979 by 4.5 and 1.6 percent, respectively. Domestic sales of smoking tobacco continued to decline, 8.2 percent in the case of pipe tobacco, and 10.2 percent for roll-your-own tobacco, compared to 1978.

GENERAL

80-1830. Adair, V. M. **Air Moving Simulated Cigarette Device.** United States Patent No. 4,184,496, January 22, 1980, 4 pp.

While there have been many devices proposed for enabling a smoker to gradually give up the smoking habit, there is nothing available to the individual who has given up smoking which would provide a substitute for the manual and oral manipulation of a cigarette, and at the same time provide the user with the ability to disperse cigarette smoke imparted into the vicinity of the user's mouth and nose by others. It is an object of the present invention to provide a device which will fulfill the above-mentioned need. The device comprises a mouthpiece member having an exterior simulating a cigarette holder, and a tubular member having an exterior simulating the exterior of a cigarette. The mouthpiece and tubular member have a continuous passage extending from the mouth-engaging end of the mouthpiece to the free end of the tubular member. Through the latter, a substantial flow of air can be established by the user blowing into the end of the mouthpiece so that a substantial current of air flows outwardly from the end of the tubular member. The mouthpiece has air-aspirating openings extending from the exterior into aspirating relation with the main passage so that when a substantial flow of air is established within the passage, the flow will aspirate additional air through the air-aspirating openings. (Auth. Abs. Mod.)

80-1831. Blizard, J. **Print Readership and Cigarette Usage. (Letter).** *Medical Journal of Australia* 2(1): 30, 32, July 1, 1978.

With the phasing out of radio and television advertising of cigarettes in Australia, cigarette advertising in the print media has increased. The relationships between print media readership and cigarette usage from 1973 to 1978 were examined. The results show apparent inconsistencies as to whether the presence or absence of cigarette advertising and antismoking articles affect the number of readers who smoke.

80-1832. British Medical Journal. **Which is More Harmful to Health: Smoking a Cigarette Down to the Filter in a Holder or Extinguishing it 2.5 cm From the Filter Without a Holder?** *British Medical Journal* 2(6154): 1773, December 23-30, 1978.

In order to answer which of the following two methods of smoking a cigarette results in a lesser smoke exposure of the smoker: (1) smoke a cigarette down to the filter in a holder or (2) smoke a cigarette down to 2.5 cm from the filter without a holder, the amount of smoke obtained by a particular individual would need to be measured. The second method is the probable answer for most people as the holder is an inefficient filter. By comparison, the unburnt part of the tobacco provides an efficient filter, and the amount of smoke produced per puff increases approximately logarithmically as the cigarette burns down.

80-1833. Dartnell's Business 1980. **Smoking Still Not Policy Issue in Most Offices.** *Dartnell's Business 1980* 1(4): 37-40, June 1980.

The 1980 Dartnell Survey on office smoking policies showed a decline in the number of companies with such policies from 30 percent in 1977 to 23 percent in 1980. Companies with antismoking campaigns also declined from 11 percent in 1977 to 9 percent in 1980, while those offering incentives to quit remained at 3 percent. Smoking was restricted to certain areas in 18 percent of the companies. Approximately 43 percent of the managers thought that productivity was adversely affected by smoking.

80-1834. Denoix, P. **Tobacco—Current French Legislation.** In: Davis, W.; Rosenfeld, C. (Editors). *Carcinogenic Risk Strategies for Intervention.* IARC Scientific Publications No. 25, 1979, pp. 9-19.

A recent (1975-1976) French antismoking campaign to bring about a reduction in tobacco consumption is described. A number of facts were used to convince the public that smoking is dangerous: of all cancers, those in which tobacco plays an important role are increasing most rapidly; and smoking reduces life expectancy. A study of smoking habits in France showed that smoking begins earlier and earlier; another study showed that 60,000 deaths/year could be linked to smoking. The cost of medical services related to smoking was calculated to be 22,000 million francs. By comparison, the budget of the antismoking campaign was 3 million francs: this was sufficient to reduce the risk by 7 percent. The second objective of the program was to give nonsmokers the possibility to assert their rights. The main target of the campaign was young people. The social pressure on them to begin smoking could be reduced if the number of smokers were reduced, if nonsmokers asserted their rights, and if the image of the smoker was made less attractive. The campaign was supported by antismoking legislation which became effective in July 1976. Smaller campaigns were planned to follow annually. The main targets of the law were advertising and publicity; in addition, smoking was prohibited in public places, public means of transportation, and educational establishments. The effectiveness of the antismoking campaign was measured by sampling changes in behavior and in sales volume. In December 1976, after the end of the campaign, there was a 5 percent decrease in the number of smokers and a 15 percent decrease in the consumption of those who continued to smoke. (Auth. Abs. Mod.)

80–1835. Emboden, W. **Narcotic Plants.** New York, Macmillan Publishing Co., Inc., 1979, 220 pp.

The disciplines of botany, chemistry, anthropology, and archeology are combined in this book to explain the various origins and uses of plant narcotics throughout history. Hypnotica, the sedatives and tranquilizers, and tobacco, hallucinogens (e.g., marihuana), stimulants, and inebriants are discussed. Many drawings and color plates accompany the discussion.

80–1836. Flamm, H.; Kunze, M.; Kunze, M. J. **Rauchen und Gesundheit in Osterreich. 2., erweiterte Auflage. [Smoking and Health in Austria. 2nd Expanded Edition.]** Austrian Federal Institute for Health, Vienna, August 1975, 71 pp., German.

The 1974 report *Smoking and Health in Austria* contained suggestions for reducing the adverse effects of smoking on health. This second edition of the report provides updated information on the activities since 1974 and indicates that many of the suggestions have been implemented in the 15 months since the original report was issued. The new edition also contains a reprint of the original report.

80–1837. Flamm, H.; Kunze, M.; Kunze, M. J. **Rauchen und Gesundheit in Osterreich. Ungekurzte Fassung. [Smoking and Health in Austria. Unabridged Version.]** Austrian Federal Institute for Health, Vienna, 1975, 113 pp., German.

This unabridged version of the 1974 report *Smoking and Health in Austria* contains a comprehensive examination of the smoking problem in that country. The topics discussed include diseases associated with smoking; smoking during pregnancy; passive smoking; epidemiology of smoking in Austria; and health costs related to smoking. Possibilities and methods for reducing the adverse effects of smoking on health are discussed and suggestions for antismoking campaigns in Austria are made.

80–1838. Gerson, L.; Wingard, D. **Fire Deaths and Drinking: Data From the Ontario Fire Reporting System.** *American Journal of Drug and Alcohol Abuse* 6(1): 125-133, 1979.

Data on recorded alcohol impairment of 264 victims of fatal fires in Ontario in 1976 are analyzed. Male victims were twice as likely to be impaired as females, and impairment was more common among those under 50 years of age. Alcohol impairment was most often noted in victims of fires at night and on weekends. Overall, 31 percent of adult victims were reported as alcohol impaired. Sixteen percent (3,653/23,109) of fires in Ontario in 1976 were caused by smoking; 68 people died and 350 were injured in these. Neither men nor women appear to be overrepresented among victims of fires caused by smoking; 34 percent (39/115) of adult men and 32 percent (22/68) of adult women who die in fires die in smoking-related fires. In fires caused by smoking, persons over 30 years were found to have a greater risk of dying. Among the four time periods, in the 6 a.m. to 12 noon period where smoking was heavily implicated, 28 percent (17/60) of people died in smoking fires. Only 5 percent (3/60) were impaired by alcohol. The Ontario data suggest that alcohol involvement in victims was not associated with smoking as a cause of the fire. (Auth. Abs. Mod.)

80–1839. Markle, G. E.; Troyer, R. J. **Smoke Gets in Your Eyes: Cigarette Smoking as Deviant Behavior.** *Social Problems* 26(5): 611-625, June 1979.

This article focuses on the long-standing, but recently intensified controversy over cigarette smoking. In the late 1960's and 1970's a variety of laws and regulations were implemented to regulate smoking and the smoker. Initially these strictures were what has been called assimilative, but more recently they have become coercive. In 1978, for example, a California referendum to ban smoking in most public places was narrowly defeated. Many view this controversy using a medical model or emphasizing the conflicting rights of smokers and nonsmokers. In this study it is analyzed as a status battle between prosmoking and antismoking vested interests. Using data from a variety of primary and secondary sources, such as government statistics, corporate reports, State codes, marketing reports, and public opinion polls, the study focuses on the political, economic, and organizational forces which have militated for or against changes. At stake is the symbolic definition of a behavior as socially approved or illegitimate and the consequent denigration of the losers. More and increasingly militant confrontations between prosmoking and antismoking forces, both at the individual and collective level, are predicted. (Auth. Abs.)

80–1840. Medical Journal of Australia. **Cigarettes—What To Do? (Letter).** *Medical Journal of Australia* 2(7): 301, September 23, 1978.

A conference was convened by the New South Wales State Cancer Council in 1978 to bring together a number of organizations, including the Australasian Colleges of Physicians and of Surgeons, the Thoracic Society, and the National Heart Foundation, for the purpose of coordinating their separate antismoking policies. The feeling at the conference was to get on with the job of influencing the community's opinion leaders and of countering the tobacco industry's concerted lobbying as well as penetrating the marketplace with the antismoking campaign.

80–1841. National Institute on Drug Abuse. **Marijuana and Health. Eighth Annual Report to the U.S. Congress From the Secretary of Health, Education, and Welfare—1980.** U.S. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, DHEW Publication No. (ADM) 80-945, 1980, 54 pp.

In this eighth edition of the Marijuana and Health Report several areas of recent developments in marihuana research are highlighted together with a summary of the scientific research accumulated through the end of 1979 concerning the drug's possible health implications. The topics discussed are nature and extent of use; chemistry; acute, long-term, and psychopathological effects; and therapeutic uses.

80–1842. O'Malley, B. **Cigarettes and Sofas. How the Tobacco Lobby Keeps the Home Fires Burning.** *Mother Jones* 4(6): 56-63, July 1979.

About 40 percent of home fire deaths in the United States are caused by cigarettes, and about 30 percent of all home fires start either in upholstery or bedding. There is an existing patent (U.S. patent No. 4,044,778) for the manufacture of a cigarette that would self-extinguish when not puffed for 5 min (and would therefore not set fire to most upholstery). Safety officials, however, are promoting efforts to fireproof home furnishings instead of advocating legislation for the manufacture of a self-extinguishing cigarette. This is attributed to the strong tobacco lobby in Congress. Even the National Safety Council has failed to endorse such legislation because of the financial support it receives from industry. The history of attempts at legislation for safety standards for cigarettes in the United States is described. Some suggestions are made for forcing Congress to take a new look at legislating safety standards for cigarettes. (See also 80-0494)

80–1843. Ross, W. S. **Let's Stop Exporting the Smoking Epidemic.** *Reader's Digest* 116(697): 143-147, May 1980.

The United States and other Western countries are exploring new markets in Africa and Asia for export of their tobacco products. The U.S. Government exports unmanufactured tobacco to Third World countries under the Food for Peace program. Health warnings are not required for cigarettes marketed overseas, and cigarettes in these countries usually contain higher levels of tar than cigarettes in the United States. The World Health Organization has reported the increase of smoking-related diseases in developing countries. Multinational tobacco companies have persuaded foreign governments that the economical benefits of growing tobacco crops results in foreign exchange. Exporting tobacco as a form of bilateral aid and governmental subsidy of tobacco farming should be discouraged.

80–1844. Slater, P. **Cigarette Smoking: The Dentist's and Hygienist's Role.** *Dental Hygiene* 52: 437-438, September 1978.

Dentists and hygienists should join physicians in educating their patients on the health hazards of smoking, particularly on the risk of oral carcinoma. Upon discovery of any abnormality, the dentist should emphasize treatment, refer the patient to a specialist, and inform the patient of possible causes for his condition (e.g., tobacco is recognized as an etiologic factor in leukoplakia development in the oral cavity).

80–1845. Spillman-Thulin, I. **A Polite Campaign for No-Smoking Areas in Restaurants.** *World Smoking and Health* 5(2): 32-33, Summer 1980.

The Swiss Association for Nonsmoking (SAN) is engaged in a campaign for no-smoking areas in restaurants. The campaign involves having nonsmokers politely ask for tables free from smoke. As part of the campaign, SAN has produced a table card reading "Thanks for not smoking here." The aim of the campaign is to motivate restaurants to provide nonsmoking areas and politicians to make them obligatory.

80–1846. Tso, T. C. **Progress in Producing Tobacco for Less Harmful Cigarettes.** *World Smoking and Health* 5(1): 30-36, Spring 1980.

Recent findings and work in progress on the production of less hazardous tobacco are discussed. Simple correlations and multiple regressions for the physical and chemical characteristics of leaf tobacco, smoke constituents of experimental cigarettes, and biological activities of cigarette smoke condensate are examined. Markers of leaf tobacco to predict its smoke condensate's biological response (expressed as the Pf value, the proportion of animals surviving without tumors) were identified. Agricultural techniques can be used to modify the levels of markers negatively associated with Pf value (nicotine, total volatile bases, phytosterols, polyphenols, petroleum ether extractables, waxes, lipid residues, and certain organic and fatty acids).

80–1847. United States Tobacco Journal. **$6 Million Granted for Smoke-Health Studies.** *United States Tobacco Journal* 208(8): 3, April 17, 1980.

More than 6 million dollars were granted in 1979 by the Council for Tobacco Research—U.S.A., Inc., for studies on smoking and health. Study subjects funded included research into genetic susceptibility to bladder cancer and the biochemical basis of predisposition to chronic obstructive pulmonary disease.

80–1848. World Smoking and Health. **The Canadian Cancer Society Brings *Fresh Air* to Youth.** *World Smoking and Health* 5(1): 44, Spring 1980.

The Canadian Cancer Society has adopted a tabloid format for publicizing the no-smoking message to young people. The new publications are "Tobacco Road" for students in grades 8 and under and "Fresh Air" for high school students. The tabloids have enjoyed high appeal among students, with circulation reported to be around 500,000.

80–1849. World Smoking and Health. **Anti-Smoking Measures Proposed in Australia.** *World Smoking and Health* 5(1): 43-44, Spring 1980.

In view of the overwhelming evidence of the harmful effects of smoking, a member of the Australian Parliament has proposed a ban on tobacco advertising and an addition to the present cigarette package imprint. Other possible antismoking policies suggested include (1) the achievement of lower smoking rates through taxes, no-smoking areas, and health warnings on packages; (2) the encouragement of young nonsmokers to remain nonsmokers; (3) the cessation of all forms of tobacco promotion; and (4) the encouragement of smokers to reduce their exposure to the harmful components of tobacco smoke.

80–1850. World Smoking and Health. **Chinese Government Begins Anti-Smoking Program.** *World Smoking and Health* 5(1): 42-43, Spring 1980.

A six-measure antismoking program in the Republic of China is described. Measures include (1) dissemination of scientific knowledge on the health hazards of smoking, (2) launching of an audiovisual antismoking campaign, (3) production of less hazardous cigarettes, (4) smoking restrictions in public places, (5) health education programs for youth, and (6) involvement of all health department clinics in antismoking campaigns.

CUMULATIVE AUTHOR AND ORGANIZATIONAL INDEX

Aasen, A. J., 80-0211, 80-0871
Abboud, R. T., 80-0333
Abdullaev, R. A., 80-1277
Abe, Y., 80-0914
Abedinzadeh, Z., 80-0011
Abel, E. L., 80-1609
Abelmann, W. H., 80-1210, 80-1640
Abelson, H. I., 80-0399
Ablov, V.I.N., 80-0671
Abraham, J. L., 80-0181
Abrams, D. B., 80-1794
Abrams, N., 80-0554
Abramson, J. H., 80-1749
Abratt, R., 80-1560
Abuhay, M., 80-0995
Achilova, A. B., 80-1226
Adair, V. M., 80-1830
Adamczyk, B., 80-1631
Adamo, C., 80-0660
Adams, B. E., 80-0095
Adams, L., 80-1693
Adams, P. I., 80-0400, 80-1750
Adams, W. G., 80-0401
Adams, W.G.F., 80-1487
Adamsson, E., 80-0250
Adey, H., 80-0958
Adir, J., 80-1091
Adkins, D., 80-1783
Advisory Council on the Environment of Quebec, 80-0847
Aerts, C., 80-1655
Afanasova, L. N., 80-1182
Agrawal, K. P., 80-1680
Agrawal, V. P., 80-0062
Ahfeldt, P.-E., 80-0783
Ahlenius, T., 80-1383
Ahmad, M., 80-1824
Ahmed, Z., 80-0995
Aikman, L. N., 80-0482
Akbarkhanzadeh, F., 80-1681
Akgun, N., 80-0956
Akin, F. J., 80-0740
Akinkugbe, O. O., 80-1491
Akiya, T., 80-0004
Akkermann, S., 80-0381
Alam, M., 80-0190
Alam, S. M., 80-0121
Albertson, P., 80-0449
Albo, J.-P., 80-0237, 80-1262, 80-1593, 80-1812
Aleksandrov, V. N., 80-1092
Alert Silva, J., 80-0295
Alexander, H., 80-1526
Alexander, J., 80-0120
Alexander, L. L., 80-1390
Alexander, P., 80-0760
Alexandrov, K., 80-1097
†Alfohn Corp., 80-0666
Ali, H., 80-1613
Allegrante, J. P., 80-1492
Allen, D. L., 80-1243
Allen, M. D., 80-1108
Almqvist, S.-O., 80-0483
Alomar, A., 80-0138
Alpern, H. P., 80-0048
Althoff, J., 80-1360
Altland, P. D., 80-0879
Alton, I. R., 80-0130
Alvarez Alva, R., 80-0343
Alvarez, W., 80-1715
American Automatic Merchandiser, 80-1042, 80-1267
†American Brands, Inc., 80-0499
American Cancer Society, 80-0078
American Chemical Society, 80-0867
American College of Obstetrics and Gynecology, 80-1222
†American Filtrona Corp., 80-0487, 80-0488, 80-0489, 80-0490, 80-0491, 80-0492
American Health Foundation, 80-0831
American Hospital Association, 80-1165
American Medical Association, 80-1278
†AMF Inc., 80-0497, 80-0498, 80-0534, 80-0546, 80-0549, 80-1545
Amindzhanov, S. A., 80-1226
Amit, Z., 80-0832
Ammann, A., 80-1538
Anastacio, R. V., 80-0784
Andersen, R. A., 80-0012, 80-1074
Anderson, A. J., 80-1205
Anderson, D., 80-1487
Anderson, H. R., 80-0322
Anderson, P. S., Jr., 80-1417
Anderson, R. C., 80-0628, 80-0629, 80-0630
Anderson, R. L., 80-1387
Andersson, K., 80-1781
Andresen, B. D., 80-0797
Angersbach, D., 80-1227
Annable, L., 80-1537, 80-1773
Anschel, K. R., 80-1019
Ansquer, J. C., 80-1798
Antman, K. H., 80-1146
Appleton, D. R., 80-0835
†Aquafilter Corp., 80-0673
Aracil, B., 80-0329
Ardelean, I., 80-1753
Arenella, L., 80-1640
Aresini, G., 80-1696
Aresini, G. A., 80-0292
Argon, S., 80-1013
Arledter, H. F., 80-0484
Armstrong, B. K., 80-0073
Armstrong, W. R., 80-0390
Arndorfer, R. C., 80-1100
Arnott, M. S., 80-0900
Aromaa, A., 80-1213
Aronov, D. M., 80-0959
Aronow, W. S., 80-0782
Arqueros, L., 80-0276
Arrendale, R. F., 80-0038, 80-0709, 80-0874
Artho, A., 80-0483, 80-0544
Ashikaga, T., 80-0955
Ashley, M. J., 80-1410
Ashton, H., 80-1610, 80-1751
Ashworth, J. T., 80-0499
Asmussen, I., 80-1468, 80-1469, 80-1732
Asp, S., 80-0751, 80-1666
Astrand, I., 80-1716
Astrup, P., 80-1117
Atanasov, B., 80-1258
Atanasova, D., 80-0799
Atanasova, Zh., 80-0799
Athanasou, J. A., 80-1391
Atkinson, W. O., 80-1585
Aubry, F., 80-0092
Auer, C., 80-1686
Auerbach, O., 80-0079, 80-0321
Augsten, M., 80-1098
Augustin, J., 80-1138

---

†Patent assignee.

Austin, H., 80–1407
Austin, M., 80–0112, 80–1709
Austin, P. R., 80–0631
†Austria Tabakwerke Aktiengesellschaft vorm., 80–0484
Autrup, H., 80–0039, 80–0265
Avimier, R., 80–1779
Awokoya, F. O., 80–0287
Ayad, W., 80–1720
Aycock, M. K., Jr., 80–1313
Ayers, R., 80–0182
Azerad, R., 80–1097
Azzam, A.M.H., 80–0095

†B.A.T. Cigaretten-Fabriken GmbH, 80–1820
Babar, S. A., 80–0190
Babbott, F. L., Jr., 80–0936
Bachman, J. G., 80–0414
Backman, H., 80–0366
Bader, M., 80–1147
Badre, R., 80–0219
Baer, D. S., 80–1334
Baer, P. E., 80–0163
Baettig, K., 80–1093
Bagga, O. P., 80–1133
Bagnato, A., 80–0763
Baicoianu, S., 80–1684
Baird, M. D., 80–1421
Bakaj, J., 80–1664
Bake, B., 80–0946, 80–1179
†Baker-Alpha Corp., 80–0485, 80–0486
Baker, E. L., Jr., 80–0880
Baker, J. C., 80–0485, 80–0486
Baker, J. L., 80–1319
Baker, M. D., 80–0093
Baker, R. R., 80–0868
Balavadze, M. B., 80–1723
Baldwin, A. D., 80–1806
Bale, C. R., 80–0528
Balenko, N. V., 80–1120
Ball, J. B., 80–1676
Ball, K., 80–0013, 80–0183
Ball, L. M., 80–0881, 80–0908
Ballantyne, D. A., 80–0958
Bane, A. L., 80–0408
Banks, J. F., 80–0567
Banks, M. H., 80–0402, 80–1754
Bansal, R. C., 80–1433
Barbenel, J. C., 80–0788
Barbey, S., 80–1630
Barbooti, M. M., 80–0036
Barboriak, J. J., 80–1205
Baretta, E. D., 80–0014
Barfknecht, C. F., 80–0282
Baric, L., 80–1752
Barnea, E., 80–1753
Barnea, M., 80–1753
Barnett, G. D., 80–1171, 80–1682
Baron, A., 80–1641
Barr, J. E., 80–1654
Barrada, M. I., 80–0130
Barranger, J. A., 80–1424
Barrett-Connor, E., 80–0112, 80–1561, 80–1709
Bartelik, S., 80–1611
Barth, W., 80–1279
Barton, T., 80–1466
Bartsch, G. E., 80–0885
Baskies, A. M., 80–0934
Basrur, P. K., 80–0251, 80–0882, 80–0901, 80–1094
Bass, R. L., III, 80–1649
Bass, R. T., 80–0028
Bassano, J.-L., 80–0255
Bassfield, R. L., 80–0233
Bastiaan, R. J., 80–0988
Bastian, P., 80–0344
Basu, P. K., 80–0882
Bateman, J.R.M., 80–0339
Batsch, F., 80–1493
Battard, J.-C., 80–1317
Battegay, R., 80–1494
Baudoux, A., 80–1650
Bauermann, E., 80–1010
Baulac, L., 80–1199
Baum, H., 80–1354
Baumgaertner, R., 80–1463
†Baumgartner Papiers SA, 80–0527
Bayer, L. W., 80–0123
Baylin, S. B., 80–1162
Bayliss, D. L., 80–0931
Bayne, C. K., 80–0252
Beaglehole, R., 80–0345
Beale, C. L., 80–1020
Beaman, B. L., 80–0101
Beard, C. M., 80–0756
Beaumatin, J., 80–1132
Beck, G. J., 80–1425
Beck, J. S., 80–0971
Beck, K. H., III, 80–0810
Becklake, M. R., 80–0092, 80–0937
Beems, R. B., 80–1345
Behr, D., 80–0211, 80–0212, 80–0213, 80–0688, 80–0689, 80–1318
Beilin, L. J., 80–1437
Belanger, C., 80–1215
Bell, H., 80–1335
Bellamy, D., 80–1354
Bellanca, J. J., 80–1280
Benacerraf, A., 80–0346
Benarie, M., 80–0718
Benatar, S. R., 80–0141
Bend, J. R., 80–0881, 80–0908
Benedict, W. F., 80–1122
Benevolenskii, A. V., 80–1240
Benfari, R., 80–1715
Benfari, R. C., 80–1703
Benfield, J. R., 80–1658
Bengtsson, C., 80–1485, 80–1704
Benignus, G., 80–0253
Benignus, V. A., 80–0253
Bennett, D., 80–1495
Bensilum, S., 80–1337
Benson, M. K., 80–0949
Berendes, H., 80–0133, 80–1739
Berenson, G. S., 80–0870
Berg, J.-E., 80–0211
Berg, K., 80–0254
Berger, R. M., 80–0487, 80–0488, 80–0489, 80–0490, 80–0491, 80–0492
Bergstermann, H., 80–1166
Bergstrand, A., 80–1383
Bergstrand, R., 80–1434
Bergstrom, R., 80–1380
Beringer, M., 80–0544, 80–0632, 80–0633
Berkson, D. M., 80–0113, 80–1730
Berman, L. B., 80–0047
Bernacca, G., 80–1646
Bernadou, M., 80–1000
Bernard, A., 80–0334
Bernard, J. P., 80–0757
Bernard, M., 80–0286
Bernfeld, P., 80–0883
Berntorp, K., 80–1629
Berntson, G. G., 80–0906
Berry, C. N., 80–0711
Bersier, A.-L., 80–1435
Bertrand, M., 80–1451
Best, F. W., 80–0611
Bettelheim, R., 80–0760
Beurret, L., 80–1435
Bevan, C., 80–1689
Beven, E. G., 80–0118
Bewley, B. R., 80–0402, 80–1754
Bezuidenhout, B. N., 80–0093
Bhatnagar, R. S., 80–1627
Bianchi, G., 80–0111
Bias, W. B., 80–1169
Bibbo, M., 80–0281
Bidou, S., 80–1184
Biener, K., 80–0403
Billiet, L., 80–0097
Billings, J. H., 80–0172

---

†Patent assignee.

Binder, C., 80–1232, 80–1745
Binder, V., 80–0806
Bing, O.H.L., 80–1640
Bingham, S., 80–0294
Binnie, C. D., 80–1496
Birchler, R., 80–1482
Birkett, D. J., 80–0263
Birnbaum, R. S., 80–1162
Birrell, J., 80–0822
Bischoff, D. P., 80–0127
Bishop, H., 80–1382
Bishop, Y.M.M., 80–0098, 80–0952
Biswas, N. M., 80–1336
Bixler, E. O., 80–0807
Bjarnason, O., 80–0806
Bjartveit, K., 80–0450
Bjornheden, T., 80–1434
Black, G. W., 80–1106
Black, I.H.C., 80–1106
Blackburn, C., 80–1431
Blackburn, C.R.B., 80–1691, 80–1701
Blackburn, H., 80–0752, 80–1212
Blackwell, M. J., 80–1281
Blair, S., 80–1715
Blanco, G. G., 80–0323
Bland, J. M., 80–1754
Blankenhorn, D. H., 80–1706
Blezard, P. N., 80–1530
Blizard, J., 80–1831
Bloch, A., 80–1435
Bloom, S., 80–1718
Blot, W. J., 80–0919
Bluethgen, A., 80–1594
Blum, A., 80–1562
Blum, A. L., 80–1482
Blum, R. H., 80–1146
Blumberg, H.-H., 80–1471
Blumberg, S., 80–1703
Blumenthal, J. A., 80–1705
Blumlein, S. L., 80–1205
Blundell, G., 80–0342
Blythe, B. J., 80–1788
Bodendorfer, T. W., 80–0129
Boeger, M., 80–0472
Bogat, G. A., 80–0415
Bognacki, N., 80–1172
Boguslawska, M., 80–1075
Boisvert, J.-M., 80–0168
Bond, C. A., 80–1328
Bondjers, G., 80–1434, 80–1440
Bonham, G. S., 80–0996
Boobis, A. R., 80–0712
Boos, R., 80–0357
Bordeaux, A. F., Jr., 80–1021
Borg, K., 80–0895
Borgmeyer, L., 80–1648
Borhani, N., 80–1715
Borhani, N. O., 80–1448
Bornstein, P. H., 80–0434
Boroski, H. M., 80–0661
Boroski, S. T., 80–0661
Boross, M., 80–0805
Borresen, A.-L., 80–0254
Borthwick, J., 80–0634, 80–0635
Bos, R. P., 80–1140
Bostock, M. I., 80–0958
Botha, A. H., 80–1589, 80–1591
Bottiger, L. E., 80–0783
Botvin, G. J., 80–0430, 80–0811, 80–1234
Bouhuys, A., 80–1411, 80–1425
Boulton, T.J.C., 80–0802
Bourcier, F., 80–0217
Bourke, G., 80–1204
Bouvrain, Y., 80–1451
Bowker, T., 80–1707
Bowman, E. R., 80–1613
Boxall, R. R., 80–1337
Boxer, P., 80–0923
Boyd, D., 80–0636
Brackmann, W. A., 80–0493
Bradford, G. L., 80–1022
Bradley, D.S.G., 80–1106
Brady, R. O., 80–1424
Braendli, O., 80–0957
Braeuer, R., 80–1192
Brahime-Reteno, O., 80–1399, 80–1409
Brailey, A. G., Jr., 80–1043
Brand, G., 80–0713
Brand, M. J., 80–0705
Brandange, S., 80–1612
Brandt, K. R., 80–1122
Branellec, A., 80–1449
Brannon, R. H., 80–1019
Brantl, V., 80–0668
Brass, A., 80–0848
Braun, L., 80–1645
Braybrook, K. J., 80–1638
Brazier, J. L., 80–1099
Breart, G., 80–0382
Breidenbach, S. T., 80–1789
Bremsak, F., 80–0056
Brengelmann, J. C., 80–0431
Breslow, L., 80–1392
Bressler, R., 80–0803
Bridbord, K., 80–0312
Briddell, D. W., 80–1755
Bridges, B. A., 80–0714
Briggs, G. G., 80–0129
Brille, D., 80–1181
Brinkhoff, D., 80–1351
Brinkmann, G., 80–0839
Briskin, T. S., 80–0637
Brisse, B., 80–1351
†British-American Tobacco Co., Ltd., 80–0474, 80–0512
British Medical Journal, 80–1044, 80–1832
Brito, M., 80–0950
Brock, L. L., 80–0188
Brockert, J. E., 80–1221
Brockway, B. S., 80–0164
Brod, G., 80–0191
Broda, S., 80–0976
Brodie, H.K.H., 80–0819
Brodie, M. J., 80–0712
Brodnitz, M. H., 80–0608
Brody, A., 80–1039
Brody, B., 80–1039
Bromberg, P. A., 80–0331
Brooks, R. R., 80–1595
Brooks, S. H., 80–1706
Bross, I.D.J., 80–0082
†Brown and Williamson Tobacco Corp., 80–0495, 80–0513, 80–0518, 80–0532, 80–0535, 80–0550, 80–0551, 80–0553, 80–0558, 80–0559, 80–0560, 80–0561, 80–0567, 80–0605, 80–0640, 80–0647, 80–0648
Brown, D., 80–0096
Brown, D. G., 80–0716
Brown, G. W., 80–0207
Brown, J. J., 80–0360
Brown, P., 80–0764
Brown, R. A., 80–1012
Brown, R. D., 80–0079
Browning, J.H.D., 80–0623
Bruce, J., 80–1686
Bruce, N., 80–1338
Brunnemann, K. D., 80–0015
Bruppacher, R., 80–1193
Bruschke, A.V.G., 80–0369
Bryant, H. G., Jr., 80–0545, 80–0562, 80–0563
Bryson, R., 80–0144
Buchbinder, B., 80–0451
Buchet, J.-P., 80–0334, 80–0386
Buchmann, P., 80–0544, 80–0632
Bucholc, B., 80–0105
Bucknall, T. E., 80–1707
Buerger, S. B., 80–1436
Buhl, F., 80–1235
Buisson, J., 80–1259
Buist, A. S., 80–0094, 80–0095
Buist, S., 80–0938

†Patent assignee.

Bulpitt, C. J., 80–1103, 80–1437
Burch, J. D., 80–1152
Burch, P.R.J., 80–0080, 80–0347, 80–0920, 80–1148
Burge, A., 80–0096
Burge, P. S., 80–0096
Burghard, G., 80–0405, 80–1412
Buring, J. E., 80–1158
Burrows, B., 80–0767
Burton, H. R., 80–1320
Busbee, D., 80–1095
Busch, F. W., 80–0715
Bush, L. P., 80–0192, 80–1381
Bustamante, C. R., 80–0638
Butler, D. P., 80–0808, 80–0992
Butler, J., 80–1168
Butts, N. K., 80–1708
Byckling, T., 80–0416
Byington, R., 80–1194
Byrne, A. R., 80–0692
Bystrom, G., 80–0984

Cabaniss, J. S., 80–1032
Caddy, G. R., 80–1755
Cady, B., 80–0452
Cady, L. D., 80–0127
Cahannes, M., 80–1244
Caille, E.-J., 80–0255
Caille, J. E., 80–1625
Calabrese, E. J., 80–0921
Caldwell, E., 80–0833
†Calgon Corp., 80–0537
Calienno, A., 80–0779
California Fire Incident Reporting System, 80–0453, 80–0849
Calkins, B. M., 80–1718
Callmer, E., 80–0359, 80–1455
Calsini, P., 80–1372
Camacho, T. C., 80–1309
Camarasa, G., 80–0138
Camm, A. J., 80–1438
Campbell, J. M., 80–1480
Campbell, J. S., 80–1015
Campbell, K. I., 80–0877
Campbell, M. J., 80–0337, 80–0716, 80–1689
Campbell, R. G., 80–1541
Camps Mestre, J. M., 80–0662
Canadian Council on Smoking and Health, 80–0869
Canellos, G. P., 80–1146
Cantrell, E. T., 80–1095
Capdevila, J., 80–1130
Caputi, A. P., 80–0717
Card, J. P., 80–0256
Carey, A. E., 80–0193
Cargill, R. L., 80–0473
Carley, D. H., 80–1023
Carlsen, J. E., 80–1465
Carlson, L. A., 80–0359, 80–0783
Carney, R. E., 80–0812
Carp, H., 80–0053, 80–0266
Carr, J., 80–0771
†Carreras Rothmans Ltd., 80–0644, 80–0645
Carreras Ruiz, O., 80–0295
Carter, P. K., 80–0296
Cartwright, W. F., 80–0568, 80–0569
Carugno, N., 80–0034, 80–1602
Casey, W. J., 80–0546
Cash, G. A., 80–1615
Castell, D. O., 80–0389
Castelli, W. P., 80–1444
Castillo-Fenoy, A., 80–0346
Castro, A., 80–1613
Catena, E., 80–0717
Caton, J. E., 80–0719
Cazamajour, F., 80–0237, 80–0244
Center for Disease Control, 80–0813
Cerreta, J. M., 80–0047
Chahidi, H., 80–0960
Chakaravarthy, S., 80–0842
Chakarvarti, S. K., 80–1596
Chakraborty, M. K., 80–0031, 80–0690, 80–0840, 80–1084, 80–1314, 80–1325, 80–1326
Challberg, K., 80–0997
Chalmers, D. M., 80–0961
Chalon, J., 80–0758
Chamberlain, W. J., 80–1319
Chamberlin, R. I., 80–1698
Chambers, L. W., 80–1152
Chambliss, C., 80–1497
Chan-Yeung, M., 80–0333, 80–1167, 80–1410
Chanarin, I., 80–0961
Chance, M.R.A., 80–0425
Chance, W. T., 80–0040
Chaplin, J. F., 80–0194, 80–0709, 80–1069, 80–1071, 80–1074
Chapman, R. A., 80–0703
Chappell, B. V., 80–1208
Chapuis, L., 80–1637
Charan, N. B., 80–1168
Charlewood, G. P., 80–0850
Charlton, A., 80–1756
Charnay, N., 80–1396
Chase, G. A., 80–1169
Chase, S., 80–1268
Chaturvedi, M. K., 80–0785
Chauvette, C., 80–1317
Chavis, M. K., 80–0028
Chazova, L. V., 80–1723
Check, W. A., 80–1547, 80–1563
Chen, C. B., 80–0722
Chen, C.H.B., 80–0873
Chen, C. J., 80–1674
Chen, J.Y.P., 80–1795
Chen, K., 80–1607
Chen, M. K., 80–1195
Cheraskin, E., 80–1339
Cherniack, R. M., 80–1432
Chernichenko, I. A., 80–1120
Chernow, B., 80–0389
Chernozemski, I., 80–0313
Chernozemsky, I. N., 80–1644
Cherry, N., 80–1498
Chia, K. B., 80–1677
Chien, C.-P., 80–0142
Chilvers, C., 80–0738
Chin, H. P., 80–1706
Chinoy, N. J., 80–1340
Chisholm, A. W., 80–0968
Chortyk, O. T., 80–0038, 80–0740, 80–0874, 80–1088, 80–1319
Chouteau, J., 80–0195, 80–0242, 80–0243, 80–0244, 80–1812
Chovil, A. C., 80–0081
Chretien, J., 80–1630
Chretien, P. B., 80–0934
Christen, A., 80–1214
Christen, A. G., 80–0165, 80–0390, 80–1045
Christensen, B., 80–0379
Christensen, J., 80–1100
Christensen, M. S., 80–1232, 80–1745
Christensen, N. J., 80–1465
Christiansen, C., 80–1232, 80–1745
Christiansen, J. S., 80–0804
Christianson, R. E., 80–0391
Christie, D.G.S., 80–1726
Chumney, R. D., Jr., 80–0499
Chuong, B. T., 80–0718
Chuttani, H. K., 80–1133
†Ciba-Geigy Corp., 80–0472
Cisin, I., 80–0399
Claflin, W. E., 80–0017, 80–0669
Clairmonte, F. F., 80–1548
Clappison, B. H., 80–0140, 80–0259
Clark, R. R., 80–0143, 80–0404
Clark, T.J.H., 80–0102, 80–0738, 80–0768, 80–1177, 80–1687
Clarke, S. W., 80–0339
Classen, W., 80–1093

†Patent assignee.

Claude, J., 80–0962
Claude, J. R., 80–1364, 80–1614
Claus, D., 80–0851
Clay, F. R., 80–0702
Clemmesen, J., 80–0714
Clifton, P., 80–1437
Cobey, C., 80–0845
Coe, R. M., 80–0814
Cohen, A. H., 80–1658
Cohen, B. H., 80–1169
Cohen, B. L., 80–1393
Cohen, B. S., 80–0257, 80–1597
Cohen, F. L., 80–1236, 80–1566
Cohen, G. M., 80–0060, 80–0741, 80–0884, 80–1096
Cohen, J. D., 80–0814, 80–0885
Cohen, S. B., 80–0184
Cohn, C. C., 80–0494
Cohn, S. H., 80–1105
Coin, E. J., 80–1358
Colbourne, M. J., 80–1670
Cole, T. J., 80–0294
Coleman, C., 80–0880
Coles, E. C., 80–1437
Collan, Y., 80–0751, 80–1666
Colle, B., 80–0763
Collen, M. F., 80–0074
Collette, P., 80–1731
Colletti, G., 80–1499
Collins, D., 80–0109
Collins, G. B., 80–0196
Collins, J. V., 80–0337, 80–1176, 80–1415
Collins, P. F., 80–0691
Collins, R., 80–1426, 80–1697
Comber, R., 80–0474
Comberg, H. U., 80–1138
Comer, A. K., 80–1496, 80–1500
Comstock, G. W., 80–0767
Congleton, W. F., 80–1586
†Consolidated Cigar Corp., 80–0554
Conway, J. B., 80–1248
Cook, G. M., 80–1152
Cook, L. C., 80–0639
Cooke, A. R., 80–0475
Cooke, E. C., 80–1772
Cooksley, W.G.E., 80–1615
Cooper, K. H., 80–0165
Cooreman, J., 80–0405, 80–1412, 80–1420
Coq, F., 80–0442
Corberand, J., 80–0258
CORESTA, 80–0214
Corey, P., 80–1410
Corman, W., 80–0663
Cornford, E., 80–1645
Corrao, G., 80–1621
Correman, J., 80–1184
Corson, M. A., 80–0900
Cosemans, J., 80–0097
Costa, E., 80–0896
Cotes, J. E., 80–1689
Cotton, D. J., 80–1171, 80–1682
Coudray, P., 80–1000
Coulet, B., 80–1435
Court, W. A., 80–0016, 80–0197
Courtois, D., 80–1132
Coussirat, J.-C., 80–0238
Couto, M. R., 80–0002
Cowell, M. J., 80–0290
Cox, M., 80–1218
Cramer, J. A., 80–0148
Crawford, J., 80–0139
Crawley, J.C.W., 80–0770
Creighton, D. E., 80–1500, 80–1501, 80–1757, 80–1758
Crellin, R. A., 80–0495, 80–0512
Cremaschi, D., 80–1260
Cresanta, J. L., 80–1450
Criqui, M. H., 80–0112, 80–1709
Crittin, J., 80–0348
Croog, S. H., 80–0824
Cropp, G.J.A., 80–0070
Crossen, P. E., 80–1616
Crost-Deniel, M., 80–0382
Crosthwaite, L., 80–1320
Crow, R. S., 80–1212
Croy, R. G., 80–0039
Cruise, J. S., 80–0432
Cruise, R. J., 80–0432
Crystal, R., 80–1118
Crystal, R. G., 80–0327, 80–1424
Cumming, A. M., 80–0371
Cumming, A.M.M., 80–0360
Cumming, G., 80–1341
Cunliffe, S., 80–0933
Curran, T. L., 80–0454
Curren, R. D., 80–1122
Curvall, M., 80–0248, 80–0249
Cusack, B., 80–1617
Czajkowski, S., Jr., 80–1126, 80–1642

Da Silva, A., 80–1196
Daftary, D. K., 80–0989
Dagenais, G. R., 80–1214
Dagle, G. E., 80–0284
Dahlen, G., 80–0254
Dahoma, U., 80–0336
†Daicel Ltd., 80–0533, 80–0538, 80–0539, 80–0659
Dalbey, W. E., 80–0719
Dalen, J., 80–1712
Dales, L. G., 80–0074
Dalferes, E. R., Jr., 80–0870
Daly, L., 80–1204
Danaher, B. G., 80–0834, 80–1249
Danel, C., 80–1630
Daniel, H., 80–0730
Daniele, R. P., 80–0324
Daniels, L. M., 80–1197
Danner, S. A., 80–0349
Dansette, P. M., 80–1097
Darcy, D., 80–0760
Dartnell's Business 1980, 80–1833
Dauber, J. H., 80–0324
Davidovski, F., 80–0329
Davies, D.M., 80–1198
Davies, D. S., 80–0712
Davies, J. K., 80–1241
Davies, J. M., 80–0720
Davies, P., 80–0893, 80–1618
Davies, S., 80–0941
Davies, S. F., 80–0940
Davis, H. T., 80–0123
Davis, J. W., 80–0350
Davis, N., 80–0750
Davis, R. F., 80–0350
Dax, E. M., 80–0259
Day, H. E., 80–1369
Day, N., 80–0312, 80–1609
Day, N. E., 80–1396
Dean, G., 80–0109
Dearing, R., 80–0053
Deasy, M. J., 80–0393
Deb, C., 80–1336
De Backer, G., 80–0969, 80–1447
DeBaets, A., 80–0001, 80–0198
DeBardeleben, J. F., 80–0020, 80–0693
DeBardeleben, M. Z., 80–0017
de Beaumont, M.-J., 80 0349
De Beer, M. C., 80–1589, 80–1590, 80–1591
DeBrunn, A. W., 80–0547, 80–0548
DeCamilla, J., 80–0123
Decherf, D., 80–1413
DeCicco, S. G., 80–0287
Deckner, C. W., 80–0171
Decoufle, P., 80–0082, 80–0312
de Gennes, J. L., 80–1199
deGroot, I., 80–0364
de Hamel, F. A., 80–1170
Dehnen, W., 80–0052
DeJong, D. W., 80–1071, 80–1261
Dekker, A., 80–1157

---

†Patent assignee.

del Castilho, P., 80–0749
Del Soldato, P., 80–1372
De Leacy, E. A., 80–1342
Deletang, J., 80–1262, 80–1812
Delman, F., 80–1549
De Marchi, D., 80–0763
Demedts, M., 80–0097
Demole, E., 80–0215, 80–0216, 80–1256
Demole, E. P., 80–0570, 80–0571, 80–0572, 80–0573, 80–0574, 80–0575, 80–0576, 80–0577, 80–0578
Demone, H. W., Jr., 80–0158
Dempsey, D. A., 80–0886
de Neuman, M. P., 80–0974
Denis, P., 80–1619
Dennison, D., 80–0998
Denoix, P., 80–1834
Dent, J., 80–1100
Denyer, M., 80–1744
Depersdorff, J., 80–1098
Dermelj, M., 80–0692
De Roo, M., 80–0097
Desai, M. V., 80–1316
Desai, R., 80–1354
De Salles de Hys, L., 80–0018
Descotes, J., 80–1099
de-The, G., 80–1396
Deubner, D. C., 80–1710
Devreux, M. G., 80–0217
Dewar, A., 80–1415
deWolfe, V. G., 80–0118
Dezso, Z., 80–0246
Dharmarajan, V., 80–0771
Dhiman, J., 80–1596
Di Costanzo, F., 80–0664
Di Ianni, D., 80–0493
Diamond, E. L., 80–1169
Dichter, M., 80–0554
Dicken, C., 80–0144
Dickerson, J. P., 80–0207, 80–0579, 80–0602
Dickey, D. W., 80–0070
Diem, J. E., 80–0771
Dietrich, A., 80–0397
Dietrich, P., 80–0580, 80–0581
Digenis, G. A., 80–0841
Dillinger, A., 80–1494
Dimitrov, At., 80–1068
Dinah, F., 80–0109
Dintcheff, B. A., 80–1609
Dipple, W. S., Jr., 80–1763
Dirimanov, M., 80–1068
Djukic, V., 80–0734
Do, A. H., 80–0258
Dobordzhginidze, L. M., 80–1723
Dobrowolski, L. A., 80–1439
Dockerty, M. B., 80–0756
Dockery, D., 80–0098
Dodds, A. M., 80–1502
Dodds, W. J., 80–1100
Dodson, R. A., 80–1101
Doggart, J. R., 80–1106
Dohi, T., 80–1102
Doi, K., 80–0523
Dolgintsev, V. I., 80–1163
Doll, R., 80–0297
Dollery, C. T., 80–1103, 80–1437
Domir, S. C., 80–0260
Donahoo, K. K., 80–0014
Dones, J.-P., 80–1000
Dorken, E., 80–1167
Dosman, J. A., 80–1171, 80–1348, 80–1682
Dossing, M., 80–1104
Doubrawa, R., 80–0406
Doueck, H. J., 80–1788
Douglas, W.H.J., 80–0742
Downes, J. E., 80–0932
Doyle, N. C., 80–1046
Drabek, J., 80–0472
Draganov, I., 80–0313
Drage, C. W., 80–0940
Dramaix, M., 80–0969, 80–1447
Drath, D., 80–0893
Drath, D. B., 80–0041, 80–0042, 80–1343
Drew, J. H., 80–1228
Drew, R. T., 80–0266
Drouet, D., 80–1181
Drummond, M. M., 80–0788
Drwiega, J., 80–0218
Dubey, R. S., 80–0685
Dubin, N. H., 80–1472
Dubois, G., 80–1413
Ducimetiere, P., 80–0962, 80–1229
Dufour, A., 80–0455
Dugan, W. M., Jr., 80–1047
Duhamel, Y., 80–0731
Dulac, G.-L., 80–0922
Dumas, C., 80–0219
Dumery, B., 80–1593
Dumont, E., 80–0370
Dundee, J. W., 80–1106
Dunger, M., 80–0032
Dunn, N. J., 80–1755
Dunn, P. J., 80–1759, 80–1760
Dunnill, M. S., 80–0939
Dunning, A. J., 80–0349
Dupree, E. A., 80–1741
Durakovic, Z., 80–0918
Durand, J. L., 80–0803
Durda, M., 80–1683
Durding, B., 80–0429
Durlacher, L., 80–0894
Durocher, D. F., 80–0019
Dutartre, D., 80–0852
Dutau, G., 80–0258
Dutu, S., 80–1684
Duval, C., 80–1619
Dweck, J. S., 80–0287
Dwivedi, S.S.L., 80–0686
Dyer, A., 80–1730, 80–1731
Dyer, A. R., 80–0113, 80–1142, 80–1194

Eadie, M. J., 80–1342
Eaker, E., 80–1703
Eaton, J. W., 80–0353
Ebing, W., 80–1327
Ecklin, G., 80–1435
Edleson, J., 80–0164
Edling, C., 80–1149
Edmondson, N. A., 80–0732
Edwards, L. E., 80–0130
Edwards, W. B., III, 80–0221, 80–0233
Effert, S., 80–0363
Ehrly, A. M., 80–1344
Eilerman, R. G., 80–0625
Einolf, W. N., 80–0020, 80–0693
Einstein, S., 80–1503
Eisenberg, S., 80–0116
Eisenbud, M., 80–1597
Eiser, J. R., 80–0145, 80–0146, 80–0147, 80–0407, 80–1504, 80–1761
Ek, S., 80–1505
El-Gamasy, A. M., 80–0684
El-Gohary, M., 80–1720
El-Sawy, M., 80–1720
Elgerot, A., 80–0043
Elgrishi, I., 80–0962
Elia, V. J., 80–0877
Elias, E. G., 80–0934
Elinder, C.-G., 80–0261
Elkind, A. K., 80–1237
Ellenberg, S. S., 80–1718
Ellington, J. J., 80–1819
Elliot, J. M., 80–0197
Elliott, J., 80–0456
Ellis, K. J., 80–1105, 80–1373
Ellis, R. L., 80–1321
Elmenhorst, H., 80–1076, 80–1598
Elomaa, I., 80–0366
Elovainio, L., 80–1048
Emboden, W., 80–1835

Emerson, P. A., 80–0351
Emley, G. S., 80–0821
Emslander, H.-P., 80–1166
Eng, A., 80–0430, 80–0811, 80–1234
Enggist, P., 80–0215, 80–0216
Enggist, P. F., 80–0578
Englund, F., 80–0057
Ensor, J. E., 80–0670
Enstrom, J. E., 80–0915, 80–1392
Enzell, C. R., 80–0211, 80–0212, 80–0213, 80–0248, 80–0249, 80–0483, 80–0688, 80–0689, 80–0871, 80–1318, 80–1333
Enzmann, J., 80–0199
Epler, G. R., 80–0777
Epstein, A., 80–1503
Epstein, F. H., 80–0114
Epstein, S. S., 80–1813
Erb, L., 80–0797
Erdreich, L. S., 80–1490
Erickson, L.G.W., 80–0148
Ericson, A., 80–0131
Eriksen, H. M., 80–0392
Eriksson, C.-G., 80–0457
Eriksson, S., 80–0325
Erinoso-Francis, A. O., 80–1574
Eschwege, E., 80–0962, 80–1451
Essigmann, J. M., 80–0039
Estel, C., 80–1470
Estes, R. S., 80–1109
Esteve, J., 80–1152
Estrada, M. L., 80–0784
Etnier, E. L., 80–1385
Ettle, S. N., 80–1197
Evans, H. E., 80–1172
Evans, H. J., 80–0723
Evans, J., 80–0109
Evans, K. E., 80–1438
Evans, R. I., 80–0408
Everitt, B. J., 80–0887
Evers, W. J., 80–0582, 80–0615
Evreux, J.-C., 80–1099
Eyer, J., 80–0075
Eyles, E., 80–0345
Eyssen, G. E., 80–1668

Faber, O. K., 80–1232
†Fabriques de Tabac Reunies SA, 80–0633
Fager, G., 80–1440
Fagerstrom, K.-O., 80–0044, 80–1535
Fainstein, V., 80–1685
Fair, G. D., 80–0496
†Fairmount Chemical Co., Inc., 80–0477
Fairshter, R. D., 80–1173
Falek, A., 80–0271
Falkson, J. L., 80–0185
Farebrother, M.J.B., 80–0835
Farinaro, E., 80–0963
Farkas, J., 80–0246, 80–0263
Farley, M. L., 80–0943
Farm Index, 80–1282
Farman, A. G., 80–1742
Farmer, W., 80–1185
Farone, W. A., 80–0707
Farquhar, J. W., 80–0120
Farrell, G. C., 80–1615
Farrell, T. R., III, 80–1357
Faucher, G., 80–1449
Fayeulle, J.-P., 80–1628
Fazey, C., 80–0458
FDA Drug Bulletin, 80–0352
Federal Trade Commission, 80–0220, 80–0448
Federspiel, C. F., 80–1686
Fedorchenko, I. M., 80–0671
Fee, J.P.H., 80–1106
Fee, W., 80–1011
Feinleib, M., 80–0117, 80–0787, 80–1203, 80–1444
Feins, I. R., 80–1817
Feist, G. R., 80–0547, 80–0548
Feldman, J. G., 80–0923
Feldman, J. J., 80–1207
Fells, G. A., 80–0327
Ferguson, R. N., 80–0020, 80–0693
Fergusson, D., 80–0111
Feron, V. J., 80–0721, 80–1345
Ferrans, V. J., 80–1424
Ferretti, R., 80–0945
Ferris, B. G., 80–0767
Ferris, B. G., Jr., 80–0098
Ferris, B., Jr., 80–0952
Feyeraband, C., 80–1687
Feyerabend, C., 80–1137, 80–1246, 80–1790
Feyerband, C., 80–0102, 80–0768
Field, G. B., 80–0766
Figueroa, P., 80–0002
Filio, A. K., 80–1620
Filipy, R. E., 80–0284
Finelli, V. N., 80–0877
Finlay-Jones, L. M., 80–0892
Finnegan, L. P., 80–1223
Fiore, J. V., 80–0497, 80–0498, 80–0549
†Firmenich SA, 80–0570, 80–0571, 80–0572, 80–0573, 80–0574, 80–0575, 80–0576, 80–0577, 80–0578, 80–0580, 80–0581, 80–0609, 80–0612, 80–1256
First, M. W., 80–1116, 80–1626
Fischbach, E.-D., 80–0191
Fischbein, A., 80–0326, 80–0954
Fischer, T., 80–0809, 80–1388, 80–1748
Fischetti, F., Jr., 80–0595, 80–0625
Fishburne, P. M., 80–0399
Fisher, C., 80–1752
Fisher, E. B., Jr., 80–0999
Fisher, F., 80–0432
Fisher, M. L., 80–0433
Fitzgerald, J., 80–0838
Flachsbart, H., 80–1323
Flamant, R., 80–0924, 80–1679
Flamm, H., 80–1836, 80–1837
Flaxman, J., 80–0166
Fleischl, P., 80–0958
Fleischman, R. W., 80–1651
Fleming, S. M., 80–0091
Fletcher, C. M., 80–0099, 80–0459, 80–0853, 80–0949
Fletcher, D. R., 80–0712
Flower, R.L.P., 80–0270, 80–1639
Flowerdew, G., 80–1146
Flowers, N. C., 80–1370
Flynn, C., 80–0583
Fodor, G., 80–1152
Fodoreanu, L., 80–1375
Fontanilles, A. M., 80–0258
Food and Drug Administration, 80–0815
Forbes, C. D., 80–0788
Forbes, W. F., 80–0829
Foreign Agriculture, 80–0176
Foreyt, J. P., 80–0163
Formicola, A. J., 80–0393
Forrest, D. W., 80–0149
Fortson, J. K., 80–0759
Fortson, P. J., 80–1088
Foss, O. P., 80–0385
Foster, F., 80–1671
Fox, A. F., 80–0665
Fox, E.R.W., 80–1058
Fox, R. B., 80–1115
Foxx, R. M., 80–1012
Foy, C. L., 80–0260
Francis, R. W., 80–1323
Franti, C. E., 80–1448
Fraser, H. S., 80–1103
Fraumeni, J. F., Jr., 80–0312, 80–0919

†Patent assignee.

Frayssinet, C., 80–1097
Frazier, T. C., 80–0499
Frear, D. S., 80–0045
Frecker, R. C., 80–1568
Frederick, L. S., 80–1809
Fredericks, G. R., 80–0583
Frederickson, J. D., 80–0500
Frederiksen, L. W., 80–0816, 80–1762
Freiesleben, E. R., 80–1760
Frelinger, A. L., 80–1162
French, J. G., 80–1281
Freour, P., 80–1000
Fridman, J. Y., 80–0671
Friedl, H. P., 80–1150
Friedlander, J., 80–0116
Friedman, G. D., 80–0074, 80–0293, 80–1441, 80–1711
Friedman, H. H., 80–1763
Frishman, W. H., 80–0964
Fritschler, A. L., 80–0854
†Fritzsche Dodge and Olcott Inc., 80–0595, 80–0625
Froberg, J., 80–1505
Froeb, H. F., 80–1190
Froh, F., 80–1171, 80–1682
Frost, J. K., 80–0329
Fu, P. C., 80–1658
Fujii, S., 80–0539
Fujikawa, K., 80–0476
Fujimori, T., 80–0240, 80–0694, 80–0695, 80–1605
Fujiwara, M., 80–0911
Fukuda, K., 80–1672
Fukuda, Y., 80–1216
Fukuzumi, T., 80–0240
Fuller, J. H., 80–1200
Fuller, R.G.C., 80–0149
Fullton, J. M., 80–0331
Fulmer, J. D., 80–1424
Fulton, G. B., 80–1770
Fung, K. H., 80–0046
Furst, A., 80–0886
Fuxe, K., 80–0887

Gachie, J.-P., 80–1000
Gadek, J., 80–1118
Gadek, J. E., 80–0327
Gaensler, E. A., 80–0777
Gagnon, M.-A., 80–0168, 80–1537
Gaines, T. P., 80–1315
Gajdusek, D. C., 80–0764
Galdston, M., 80–0100
Gallagher, H., 80–1175
†Gallaher Ltd., 80–0636
Gallais, M., 80–1449
Galloway, W. D., 80–0622
Gannon, W. F., 80–0017
Ganyard, M. C., 80–1328
Garcia, A. G., 80–1361
Gardner, J. W., 80–0119
Gardner, M. J., 80–0268
Garfinkel, L., 80–0079, 80–0150, 80–0916, 80–0917
Garg, J. P., 80–0785
Garisch, J.A.M., 80–1744
Garn, S. M., 80–1481
Garrett, N. E., 80–1620
Garrett, R.J.B., 80–1346, 80–1620
Garrison, R. J., 80–1444
Garrow, J. S., 80–1143
Garside, D., 80–1142, 80–1194, 80–1731
Gart, J. J., 80–1283
Gartside, P. S., 80–0364
Garvey, J., 80–1443
Gautam, R. K., 80–0062
Gavett, E. E., 80–1031
Gayle, T. M., 80–0023
Gear, J.S.S., 80–1437
Geary, M., 80–0941
Gebri, P., 80–0200
Geddes, D. M., 80–0925
Geddes, S., 80–0329
Gee, J.B.L., 80–1112, 80–1174
Geiss, V. L., 80–0550, 80–0551, 80–0553, 80–0559, 80–0560, 80–0561
Gelb, A. F., 80–0328
Gelbart, S. M., 80–1347
Gelboin, H. V., 80–1371
Gellatly, G., 80–0552, 80–0555
Gentry, L. O., 80–1430
George, L. K., 80–0819
George, P. F., 80–0672
Gergely, I., 80–0805
Gerrard, J. W., 80–1348
Gersel-Pedersen, N., 80–1743
Gerson, L., 80–1838
Gervois, M., 80–1413
Geser, A., 80–1396
Ghanayem, B., 80–1095
Ghezzo, H. R., 80–1411
Ghosh, J. J., 80–1127
Giacino, C., 80–0627
Giambiagi, N., 80–0002
Gibbs, G. W., 80–0083, 80–0092, 80–1668
Gibel, W., 80–1279, 80–1471
Gibson, G. J., 80–1688
Gibson, J., 80–1175
Gibson, J. A., 80–0628
Gielen, J. E., 80–0912, 80–1107
Giger, M., 80–1482
Gilbert, D. G., 80–1001, 80–1002
Gilbert, M., 80–0987
Gillam, D., 80–0775
Gillespie, P., 80–0139
Gilli, G., 80–1621, 80–1622, 80–1623
Gillum, R., 80–0752
Gillum, R. F., 80–0115
Gilmour, W. H., 80–1445
Gilpin, G. R., 80–1337
Giorguieff-Chesselet, M. F., 80–1349
Girod, M., 80–1435
Giudice, J. C., 80–1188
†Givaudan Corp., 80–0583
Glatt, H. R., 80–0912
Glazunov, I. S., 80–1723
Gleich, G. J., 80–0262
Gleizes, B., 80–0258
Glenn, D. F., 80–0221
Glick, J. H., 80–0271
Glindmeyer, H., 80–0771
Glover, E. D., 80–1506
Glowinski, J., 80–1349
Glueck, C. J., 80–0364, 80–0379
Gmuer, M., 80–1796
Gnana Kumar, D., 80–0840
Goavec, E., 80–0501
Godbold, J. H., Jr., 80–0753
Godfrey, F. D., Jr., 80–0502
Godsey, F., 80–0370
Goeringer, G. C., 80–0274
Goffinet, D., 80–0346
Gofin, J., 80–1749
Gofin, R., 80–1749
Goh, E. H., 80–1677
Going, R. E., 80–0696
Golaszewski, T., 80–0998
Gold, J., 80–0383
Goldberg, J., 80–1507, 80–1779
Goldberg, M. S., 80–0671
Goldbourt, U., 80–1624
Golde, D. W., 80–0769, 80–1114
Golding, J., 80–1777
Golding, L. A., 80–1708
Goldstein, B., 80–0413
Goldstein, J. J., 80–0673
Golf, S., 80–1049
Golli, V., 80–1508
Gonvers, J.-J., 80–1482
Gooden, D. T., 80–1315
Goodenberger, D., 80–1643
Gooding, L., 80–1050

---

†Patent assignee.

Gopalachari, N. C., 80–0003, 80–0021, 80–0030, 80–0460, 80–0681, 80–0697, 80–1077, 80–1085, 80–1263, 80–1542
Gopalam, A., 80–0690, 80–0697, 80–1077, 80–1263
Gordon, P., 80–1152
Gordon, R., 80–1188
Gordon, Y. B., 80–0383
Gori, G. B., 80–0443, 80–0464, 80–1283, 80–1321, 80–1565
Gorstein, F., 80–0758
Gorynski, P., 80–0994
Gossop, M., 80–1504
Gotestam, K. G., 80–0044
Gotsman, M. S., 80–0116
Gottlieb, N. H., 80–0158
Goubran, E. Z., 80–1350
Goujon, F., 80–1107
Gourgon, R., 80–1451
Gowen, J. A., 80–0193
Graff, S. A., 80–0014
Graham, B. L., 80–1171, 80–1682
Graham, S., 80–0088, 80–0089
Grandjean, E., 80–0809, 80–1388, 80–1748
Granerus, G., 80–0772
Grassino, A., 80–0946
Graudenz, M., 80–1355
Gravely, L. E., 80–0550, 80–0551, 80–0553, 80–0559, 80–0560, 80–0561
Graves, C. G., 80–1169
Gray, N. J., 80–1502
Great Britain Laboratory of the Government Chemist, 80–0698, 80–0699, 80–0700, 80–0855, 80–0856
Gredler, B., 80–0926, 80–1570
Green, C. R., 80–0222, 80–0611
Green, K. G., 80–0965
Green, L. W., 80–1191
Green, M., 80–0096
Green, M. R., 80–0047
Greenberg, S. D., 80–0943
Greenberger, J. S., 80–1146
Greenblatt, D. J., 80–1108
Greene, F. E., 80–0888
Greene, G. E., 80–0180
Greenwood, S. G., 80–1764
Greer, C. A., 80–0048
Gregory, C. F., 80–0550, 80–0551
Gregory, F. H., 80–0444
Greig, M., 80–1201, 80–1489
Griesemer, R., 80–0719
Griesemer, R. A., 80–1660, 80–1661
Griest, W. H., 80–0223
Griffin, A. C., 80–0900
Griffiths, R. R., 80–1767
Grillot, A., 80–1798
Grimm, J., 80–1463
Grindel, B., 80–0381
Grishina, L. G., 80–1182
Grosgogeat, Y., 80–1451
Grossman, H., 80–0503, 80–0504, 80–0505, 80–0506, 80–0507
Grosz, H. J., 80–0817
Grubbs, H. J., 80–0584, 80–0585, 80–0586, 80–0610, 80–0614
Gruenewald, K., 80–0164
Grufferman, S., 80–1712
Grune, M., 80–1630
Grygiel, J. J., 80–0263
Grylls, D. G., 80–1525
Grzybowski, S., 80–1167
Gsell, O., 80–1202
Guberan, E., 80–0076
Guelker, H., 80–1351
Guerin, M. R., 80–0223, 80–0719
Guerra, R. E., 80–0666
Guetg, R., 80–1400
Guidotti, A., 80–0896
Guillerm, R., 80–0219, 80–0409, 80–1625, 80–1765
Guimard, J., 80–0889
†Gulf and Western Corp., 80–0502
Gulsvik, A., 80–1414
Gump, D. W., 80–0936
Gumushan, O. H., 80–1814
Gunning, J. E., 80–0129
Gunnlaugsson, O., 80–0806
Gupta, P. C., 80–0989
Gupta, P. K., 80–0329
Gupta, S., 80–1739
Guyatt, A. R., 80–1341
Guz, A., 80–1527, 80–1693
Guzowski, K., 80–1442

Haack, D. G., 80–0100, 80–0330
Haas, H., 80–1360
Haas, J. F., 80–0132
Habib, J., 80–1749
Hackett, B. J., 80–1667
Hackett, D. R., 80–0818
Hackett, G., 80–0836, 80–1536
Haenszel, W., 80–0927
Haga, P., 80–0385
Hagedorn, B., 80–0264
Hagerup, L., 80–1729
Haire, M., 80–1106
Hakama, M., 80–1145
Hakanson, E. Y., 80–0130
Hakkila, J., 80–0356
Hall, D., 80–1171
Hall, J. B., 80–0587, 80–0588, 80–0589, 80–0590, 80–0591, 80–0592, 80–0596, 80–0616, 80–0617, 80–0618, 80–0619
Hall, R. A., 80–0629
Hall, R. G., 80–0786, 80–0838
Hall, S. M., 80–0170, 80–0786
Halter, H. M., 80–0174
Hamalainen, H., 80–0356
Hamann, J., 80–1594
Hamashima, T., 80–0872
Hamburg, D. A., 80–1051
Hamby, R. I., 80–1443
Hames, C. G., 80–0085, 80–1710
Hamid, A., 80–0701
Hamilton, D., 80–0102, 80–0768
Hamilton, P.J.S., 80–1200
Hamilton, S. B., 80–0434
Hammad, Y. Y., 80–0771
Hammarsten, J. F., 80–1417
Hammer, R. E., 80–0049
Hammond, E. C., 80–0917, 80–1394, 80–1397, 80–1405
Hamosh, M., 80–0890
Hamosh, P., 80–0890
Hamza, J., 80–0461
Hanashiro, P. K., 80–1706
Hannan, M. A., 80–1109
Hansen, B. A., 80–0167
Hansen, W. B., 80–0408
Hansson, L., 80–0366
Hanst, P. L., 80–0702
Harada, T., 80–0901, 80–1094
Hardes, G., 80–1526
Harel, S., 80–0053
Harenberg, J., 80–1138, 80–1206
Harlap, S., 80–0133, 80–1739
Harley, N. H., 80–1597
Harmatz, J. S., 80–1108
Harper, E. D., 80–0500
Harper, G. S., 80–1437
Harries, P. G., 80–0776, 80–1695
Harris, C. C., 80–0039, 80–0050, 80–0265
Harris, C. R., 80–0703
Harris, D. E., 80–1208
Harris, I. R., 80–0513
Harrison, K. L., 80–1480, 80–1733
Harrup, T., 80–0167
Harvey, B.A.M., 80–0800
Harvey, W. R., 80–0704
Hashimoto, T., 80–1216

---

†Patent assignee.

Hasin, Y., 80–0116
Haslam, P. L., 80–1176, 80–1415
Hasmall, D.L.A., 80–0496
Hassanein, K., 80–0798
Hathaway, D. E., 80–1061
Haugh, L. D., 80–0696
Hause, L. L., 80–1389
Havis, J., 80–0408
Havlik, R. J., 80–1444
Hawkins, C. F., 80–1382
Hawkins, R., 80–0096
Hawley, R. J., 80–0101
Hawthorne, V. M., 80–1445
Hay, D. R., 80–1509
Hay, I., 80–1201
Hayashi, M., 80–0893
Hayashi, S., 80–1110
Hayashi, T., 80–1766
Hayem, A., 80–1111
Hayes, D. A., 80–0331
Haynes, S. G., 80–0117, 80–0787, 80–1203
Health and Welfare Canada, 80–0298, 80–0410
Health Insurance Institute, 80–1052
Heard, B., 80–1176
Heath, J. R., 80–1695
Heath, R. B., 80–0337
Hebbel, R. P., 80–0353
Hecht, S. S., 80–0022, 80–0024, 80–0722, 80–0873, 80–0891
Heckman, C. A., 80–0729, 80–1121
Heczko, P., 80–1702
Hedegard, B., 80–0396
Hedge, R. W., 80–0640
Heeschen, W., 80–1594
Heggestuen, J. E., 80–0674
Heim, E., 80–1807
Heimstra, N. W., 80–1789
Heinemann, G., 80–1656
Heiner, D. C., 80–1348
Heinsohn, H. H., Jr., 80–0615
Heiss, G., 80–0085, 80–0379
Heit, P., 80–1510
Helgeland, A., 80–0966, 80–1714
Hell, D., 80–1494
Heller, R. F., 80–1726
Helms, M. J., 80–1710
Helsing, K. J., 80–0767
Helzer, S. G., 80–0451
Hendel, J. G., 80–0016
Henderson, B. E., 80–0084
Henderson, P. T., 80–1140
Henderson, R. M., 80–0207
Hendtlass, J., 80–1238
Hengen, N., 80–1138, 80–1206
Henin, J. P., 80 0730
Hennekens, C. H., 80–1215, 80–1712
Hennequin, J.-F., 80–0382
Henningfield, J. E., 80–1767
Henry, C. J., 80–1122
Hensleigh, P. A., 80–0798
Hepper, N.G.G., 80–0940
Herber, R.F.M., 80–0749
Herberg, D., 80–1570
Herd, A. J., 80–0630
Herman, C. P., 80–0151, 80–1568
Herman, J. B., 80–1624
Hernandez, A. C., 80–0508
Hernberg, S., 80–0318, 80–0751, 80–1666
Herning, R. I., 80–1357
Hersey, P., 80–0462
Hertzer, N. R., 80–0118
Herzog, C., 80–0354
Hess, H., 80–1647
Hettler, W., 80–1247
Heyden, F., 80–0085
Heyden, S., 80–0085
Hickey, N., 80–1204
Hickman, E. S., 80–0500
Hidaka, H., 80–0911
Hidmark, A., 80–0057
Hieger, L. R., 80–0943
Hieronymi, U., 80–1524
Higenbottam, T., 80–0102, 80–0768, 80–1177, 80–1687
Higgins, C. E., 80–0023
Higginson, J., 80–1151
Higman, H. C., 80–0037, 80–0038, 80–0874
Hildebrandt, J., 80–1168
Hilfrich, J., 80–0891
Hill, D., 80–1269
Hill, D. J., 80–1502
Hill, P., 80–0430, 80–1352
Hiller, J. M., 80–0705
Hillerdal, G., 80–0103
Hillhouse, A., 80–1050
Hillman, E., 80–0265
Hills, E. A., 80–0941
Hind, J. D., 80–0509
Hinds, M. W., 80–0086
Hinds, W. C., 80–1116, 80–1626
Hinman, L. M., 80–1112, 80–1174
Hirayama, T., 80–0087, 80–1674
Hirohata, T., 80–1208, 80–1672
Hirono, I., 80–0744, 80–0902
Hirota, N., 80–1113
Hirsh, C., 80–1783
Hirst, B. L., 80–0290
Hirvonen, L., 80–0411
Hites, R. A., 80–0054
Hjermann, I., 80–0966, 80–1446, 80–1714
Hjern, L. O., 80–0483
Ho, H. C., 80–1396
Ho, J.H.C., 80–1670
Hobbs, M.S.T., 80–0073
Hoch, P., 80–1494
Hochstetler, A. R., 80–0583
Hockett, R. C., 80–1053
Hocking, W. G., 80–0769, 80–1114
Hodous, T. K., 80–0942
†Hoechst Aktiengesellschaft, 80–0511, 80–1821
Hoeffler, D. F., 80–1564
Hoel, D. G., 80–0312
Hoelzel, C. B., 80–0510, 80–0564
Hofer, H. O., 80–1400
Hoff, F. L., 80–1024
Hoffman, I., 80–1443
Hoffman, W. K., 80–1197
Hoffmann, D., 80–0015, 80–0022, 80–0024, 80–0187, 80–0708, 80–0722, 80–0873, 80–0891, 80–1332, 80–1565
Hoffmann, K., 80–1206
†Hoffmann-La Roche Inc., 80–0557
Hoffmann, R. G., 80–1205
Hofstetter, J.-R., 80–1482
Hogetveit, A. C., 80–1408
Hoidal, J. R., 80–1115
Hokfelt, T., 80–0887
Holbrook, J. H., 80–1054
Holdbrook, M. J., 80–1709
Hollenweger, V., 80–1399, 80–1409
Hollinshead, W. H., III, 80–0985
Hollnagel, H., 80–1713
Hollo, I., 80–0805
Holly, R. C., 80–0936
Holma, B., 80–1178, 80–1416
Holme, I., 80–0966, 80–1714
Holmes, M. A., 80–1341
Holst, A., 80–0511
Holt, P. G., 80–0892
Holtz, B., 80–1179
Holtz, J. L., 80–0880
Homan, K., 80–1255
Homburger, F., 80–0883
Hood, B., 80–1629
Hoop, K. A., 80–1657
Hoover, D. M., 80–1025, 80–1550
Hoover, R. N., 80–0312, 80–1398
Hopkin, J. M., 80–0723

†Patent assignee.

Hopkins, J., 80–1734
Hopkins, W. C., 80–0509
Hopp, C., 80–1749
Horan, J. J., 80–0836, 80–1536
Hornbrook, K. R., 80–1136
Horowitz, C., 80–0554
Horowitz, I., 80–0083
Horowitz, M. J., 80–1715
Horrobin, D. F., 80–0967
Horsch, A., 80–1206
Horsewell, H. G., 80–0512, 80–0513
Horster, M., 80–0724
Horton, F. O., III, 80–1417
Hosoda, Y., 80–1216
Hosokawa, T., 80–0914
Hospitals, 80–1284
Hoult, J.R.S., 80–0711
Houpt, J. L., 80–0819
Hourigan, W. W., 80–1021
Houten, L., 80–0082
Howard, G., 80–0795
Howe, E. E., 80–0393
Howe, G. R., 80–1152
Howe, M., 80–0820
Howland, K. E., 80–0177
Hrbek, J., 80–1768
Hruza, A. S., 80–1809
Hruza, D. E., 80–0587, 80–0588
Hruza, D. E., Sr., 80–0596, 80–0616, 80–0617, 80–0618, 80–0619
Hseih, R. H., 80–0025
Hsieh, T.C.Y., 80–0224
Hsu, I. C., 80–0050
Hsu, S. C., 80–0696
Huber, G. L., 80–0041, 80–0042, 80–0893, 80–1116, 80–1210, 80–1343, 80–1618, 80–1626, 80–1640
Huber, S. A., 80–0289
Hubermont, G., 80–0386
Hubert, P., 80–0565
Hughes, J., 80–0288
Hugod, C., 80–1117, 80–1353
Hulands, G., 80–0770
Hulanicka, K., 80–1442
Hull, C. L., 80–0412
Hulley, S., 80–1715
Hulshoff, A., 80–0051
Humberd, D. R., 80–1551
Humble, C., 80–0098
Humphries, B., 80–0186
Hunninghake, G., 80–1118
Hunt, J., 80–1489
Hunt, J. H., 80–0139
Hunt, P. T., 80–0152
Hunter, D., 80–0342, 80–0949, 80–1688
Hurley, L. H., 80–1109
Hurst, G. A., 80–0943
Husain, M. M., 80–0052
Hussain, M. Z., 80–1627
Hussain, T., 80–0202
Husum, B., 80–1119
Hutchinson, C. R., 80–0231
Hutchinson, R. R., 80–0821
Hutchison, D.C.S., 80–1354
Hutt, P. B., 80–0463
Huy, N. D., 80–0355
Hyatt, R. E., 80–0773
Hyland, M., 80–0822
Hynd, G. W., 80–0435
Hyner, G. C., 80–1797

Iammarino, N., 80–1510
Ianysheva, N. Ia., 80–1120
Ibraeva, B. Sh., 80–0273
Ihrig, A. M., 80–0514
Ikeda, M., 80–0302
Imbery, D., 80–1807
†Imperial Chemical Industries Ltd., 80–0628, 80–0629, 80–0630, 80–0634, 80–0635, 80–0643, 80–0658
†Imperial Group Ltd., 80–0528
Imura, H., 80–0748, 80–1665
Infante, F., 80–1399, 80–1409
Infante, P. F., 80–0931
Ingenito, A. J., 80–0725, 80–0894
Ingervall, B., 80–0396
Ingram, C. G., 80–1427
Ingram, R., 80–0413
Inoki, M., 80–1363
†International Flavors and Fragrances Inc., 80–0582, 80–0587, 80–0588, 80–0589, 80–0590, 80–0591, 80–0592, 80–0596, 80–0597, 80–0598, 80–0599, 80–0600, 80–0601, 80–0603, 80–0606, 80–0607, 80–0608, 80–0615, 80–0616, 80–0617, 80–0618, 80–0619, 80–0620, 80–0621, 80–0626, 80–0627, 80–1808, 80–1809, 80–1810
International Union Against Cancer, 80–1769
Ippolito, R. A., 80–1511
Irwig, L. M., 80–0093
Isacsson, S.-O., 80–1055
Ishiguro, S., 80–0026, 80–0027, 80–0876, 80–1322
†Ishihara Sangyo Kaisha Ltd., 80–0476, 80–0479
Ishii, K., 80–0538
Ishizaki, H., 80–0004
Isles, C., 80–0360
Israel, M., 80–1026
Ito, T. I., 80–0174
Itskovitz, H. D., 80–1197
Ivanov, N., 80–0225
Iwase, H., 80–0914
Izard, C., 80–1512, 80–1628
Izquierdo, I., 80–1355

Jackson, H., 80–0104
Jackson, M. A., 80–1346, 80–1620
Jacob, H. S., 80–0353
Jacobs, D. R., Jr., 80–1212
Jacobsen, E. L., 80–0675
Jain, D. K., 80–0299
Jakobsson, S. W., 80–1383
Jaloustre, H. S., 80–1250
James, W. B., 80–0207
James, W. G., 80–1513
James, W.P.T., 80–0294
Jamison, R. N., 80–1514
Janardhana Rao, G., 80–0726
Janfaza, P., 80–0759
Janoff, A., 80–0053, 80–0266
Janousek, K., 80–0543
Januszkiewicz, A., 80–0950
Janzon, L., 80–1629
†Japan Monopoly Corp., 80–0539
†Japan Tobacco and Salt Public Corp., 80–0523, 80–0641, 80–0659, 80–1265
Jarrett, R. J., 80–1200
Jarvik, M. E., 80–1802
Jarvinen, E., 80–0318
Jarvis, L. R., 80–1356
Jarvis, M. J., 80–1137
Jaskolka, D., 80–1631
Jaubert, F., 80–1630
Jayasundar, S., 80–0267
Jedrychowski, W., 80–0105, 80–0332, 80–1418, 80–1631
Jeffery, R. W., 80–0066
Jefferys, D., 80–0903
Jeffrey, A. M., 80–0265
Jenkins, A., 80–1527, 80–1693
Jenkins, C. R., 80–0495
Jenkins, R. W., Jr., 80–0028, 80–1323
Jenkins, W. T., 80–0900
Jerina, D. M., 80–0068
Jernstrom, B., 80–0888

†Patent assignee.

Jewell, J. N., 80–0559, 80–0560, 80–0561
Jienescu, Z., 80–1684
Jimenez, S. M., 80–0226
Jitsukawa, H., 80–1216
Jodl, R., 80–0706
Joffe, J. M., 80–1224
Johansen, A., 80–1175
John, V., 80–1368
Johns, E. W., 80–0760
Johns, P. A., 80–0436
Johnson, B., 80–0377
Johnson, B. F., 80–1437
Johnson, B. L., 80–1131
Johnson, D. E., 80–1634, 80–1649
Johnson, D. H., 80–0393
Johnson, E. E., 80–1384
Johnson, J.W.C., 80–1472
Johnson, M.R.D., 80–0402
Johnson, P. R., 80–1027
Johnson, R. R., 80–0605, 80–0648
Johnson, W. E., 80–1101
Johnson, W. H., 80–1816
Johnson, W. R., 80–0227
Johnson, W. R., Jr., 80–0585, 80–0586, 80–0610, 80–0614
Johnston, C. I., 80–0140, 80–0259
Johnston, H.M.L., 80–1106
Johnston, J. R., 80–0093
Johnston, L. D., 80–0414
Johnston, S. B., 80–1106
Joigny, C., 80–1599
Jolly, D. T., 80–1180
Jonas, S., 80–0187
Jonasson, K., 80–0806
Jonasson, T. A., 80–0806
Jones, D. P., 80–0337
Jones, J. G., 80–0720, 80–0770
Jones, J. L., 80–0201
Jones, J. W., 80–0415, 80–0823
Jones, M. L., 80–0010
Jones, R. N., 80–0771
Jones, R. T., 80–1357
Jonnard, R., 80–1153
Jonsson, B. G., 80–1716
Jorgovic-Kremzer, J., 80–0682
Journal of the National Medical Association, 80–1285
Joyce, P. J., 80–0515
Judson, F. N., 80–0188
†Julius Glatz GmbH, 80–1818
Jusko, W. J., 80–0268
Jussawalla, D. J., 80–0299

Kaden, D. A., 80–0054
Kadner, J., 80–1470
Kaemmerer, W. F., 80–0157
Kagan, A., 80–1505
Kagawa, J., 80–1429
Kahl, G. F., 80–0727
Kahler, R. W., 80–0676, 80–0677, 80–0678
Kahn, G. C., 80–0712
Kaijser, L., 80–0359, 80–1455
Kaji, T., 80–0476
Kakande, I., 80–0300
Kales, A., 80–0807
Kales, J. D., 80–0807
Kalimo, E., 80–0416
Kallen, B., 80–0131
Kallianos, A. G., 80–0568, 80–0569, 80–0593, 80–0594, 80–0622
Kallio, V., 80–0356
Kallman, M. D., 80–0040
Kamath, V., 80–0596, 80–0621
Kameswara Rao, B. V., 80–0021, 80–0840, 80–1542
Kanarek, D. J., 80–1698
Kane, S. J., 80–1785
Kaneko, H., 80–0240, 80–0694, 80–0695, 80–1605
Kanemitsu, T., 80–1699
Kannel, W. B., 80–0117, 80–1203
Kansakoski, I., 80–0411
Kantanen, M., 80–1245
Kaplan, R., 80–1510
Karageorgos, A. C., 80–1018
Karagezov, G. S., 80–1067
Karba, R., 80–0056
Karki, N. T., 80–0067, 80–1225
Karlsson, C.-G., 80–1505
Karlsson, K., 80–0249
Karmazyn, M., 80–0967
Karnik, A. S., 80–1358
Karnovsky, M. L., 80–0041, 80–0042
Karoum, F., 80–0896
Karr, R. M., 80–0899
Karvonen, M. J., 80–1227
Kasperbauer, M. J., 80–0012
Kassman, A. J., 80–0707
Kasuga, R., 80–0694, 80–0695
Kaswan, J., 80–0516
Kato, K., 80–0659, 80–1605
Kato, R., 80–0275
Kato, Y., 80–0748
Katz, J. S., 80–0758
Katz, T., 80–0693
Kauffmann, F., 80–1181
Kaufman, D. W., 80–1230, 80–1727
Kaufman, J. D., 80–0187
Kaufman, J. L., 80–1188
Kaul, K., 80–0224, 80–1600
Kauppila, A., 80–1225
Kaur, H., 80–0153
Kausch, E., 80–1820
Kavanagh, T., 80–0968
Kawabata, M., 80–0641
Kawamoto, J., 80–0538
Kawano, M. M., 80–1003
Kawashiro, T., 80–1359
Kazemi, H., 80–1698
Keast, D., 80–0892
Keeling, C. A., 80–1527
Keen, H., 80–1200
Keith, C. H., 80–1078
Kekki, P., 80–1056
Kelble, D. L., 80–0188
Kelledjian, A. J., 80–1253
Kelley, S. A., 80–1815
Kelly, J. G., 80–1617
Kelly, K., 80–0364
Kelly, N. R., 80–1566
Kelly, T. K., 80–0497
Kelson, S. R., 80–1770
Kemel, M. L., 80–1349
Kennedy, J., 80–0968
Kenney, E. B., 80–1635
Kerker, M., 80–0707
Kershnar, R. K., 80–0127
Ketkar, M. B., 80–1360
Keyl, K., 80–1515
Keys, A., 80–1717
Khadim, M. I., 80–0394
Khalil, J. K., 80–0202
Khalkhali, Z., 80–0011
Khan, M. Y., 80–1153
Khan, P., 80–0202
Khariton, M. Sh., 80–1546
Khosla, T., 80–1567
Khoury, P., 80–0364
Kiefer, J. E., 80–0029
Kiernan, K. E., 80–1498
Kiessling, K.-H., 80–0277
Kiger, H. C., 80–1270
Kilgore, A., 80–1653
Kilpio, J., 80–0366
Kimberling, W. J., 80–1454
Kimura, F., 80–0476
King, B. E., 80–0159
King, D. A., 80–0644, 80–0645
Kirkscey, K. A., 80–1385
Kirpekar, S. M., 80–1361
Kisaki, T., 80–1543
Kish, C. J., 80–0477
Kiss, I., 80–0728

†Patent assignee.

Kitchens, C. S., 80–0973
Kite, G. F., 80–0555
Kittel, F., 80–1447
Kiviluoto, R., 80–1145
Kiwala, J., 80–0599, 80–0600, 80–0601, 80–0615, 80–0616, 80–0617, 80–0618, 80–0619, 80–0620
Kizilbash, M., 80–0301
Kjaer, G., 80–1178, 80–1416
Kjellstrom, T., 80–0261, 80–0269, 80–0895, 80–1362
Klaiber, E. M., 80–0606, 80–0607
Klauber, M. R., 80–0119
Klaukka, T., 80–0416
Klaus, E., 80–0727
Klein, E., 80–0328
Klein, G. S., 80–0758
Klein, M., 80–1253
Klein-Szanto, A.J.P., 80–0729, 80–1121
Kleinman, J. C., 80–1207
Kleinmann, G., 80–0164
Klick, R., 80–0998
Kliemann, B., 80–1807
Knauth, P., 80–1227
Knekt, P., 80–1213
Knight, B. J., 80–0417
Knott, V. J., 80–1632, 80–1633, 80–1771
Knuesli, C., 80–1400
Kobayashi, N., 80–0891
Koch, A., 80–0357, 80–1206
Koch, R., 80–0483, 80–0544
Kochar, M. S., 80–1197
Kodlin, D., 80–1457
Koenig, T., 80–0724
Koepsell, T. D., 80–1231
Koeter, H.B.W.M., 80–1345
Koivisto, M., 80–1225
Koiwai, A., 80–0872, 80–1543
Kokko, S., 80–1245
Kolonel, L. N., 80–1208
Kolotkin, R., 80–1776
Komansky, H. J., 80–1188
Komenda, S., 80–1768
Kondo, F., 80–1264
Kondo, K., 80–1216
Kong, Y., 80–1705
Kono, S., 80–0302
Kopaczewski, A., 80–1111
Kopel, S. A., 80–1499
Kopitar, Z., 80–0056
Kopp, H. G., 80–1365
Korgeski, G., 80–1776
Kornegay, H. R., 80–1286, 80–1557
Kornhuber, H. H., 80–0851
Kornitzer, M., 80–0969, 80–1447
Korovina, O. V., 80–1182
Korsgaard, R., 80–0303
Koskela, K., 80–0367, 80–1220, 80–1255
Koskela, R.-S., 80–0318
Koslowsky, M., 80–0824
Kostenbauder, H. B., 80–0051, 80–0264
Kostrzewa, M., 80–0511
Kosuge, T., 80–0280
Kotani, M., 80–0533
Koup, J. R., 80–0268
Kouri, R. E., 80–1122
Kovats, E., 80–1256
Kowal, N. E., 80–1634
Koyama, Y., 80–0604
Kozak, P. P., Jr., 80–0055
Kozjek, F., 80–0056
Kozlowski, L. T., 80–0151, 80–0154, 80–1568
Kraal, J. H., 80–1635
Kraemer, D. F., 80–1634
Kramer, J. R., 80–0118
Krannich, W., 80–1279
Krantz, D., 80–0346
Krantz, D. S., 80–0970
Krasnegor, N. A., 80–1239
Kraus, J. F., 80–1448
Krawczyk, L., 80–0178
Kretzschmar, R. M., 80–1569
Kreyberg, L., 80–0304, 80–0305
Krishna Rao, A., 80–0726
Kritsikis, S., 80–1229
Kritskii, V. N., 80–1057
Krueger, D. E., 80–1718
Kruski, A. W., 80–1649
Krzyzanowski, M., 80–1183
Kubik, A., 80–0106
Kucek, P., 80–1004
Kudo, T., 80–0523
Kuebler, W., 80–0362
Kuehnle, J. C., 80–1005, 80–1519
Kuenstler, I., 80–1082
Kulka, K., 80–0595
Kuller, L. H., 80–1209, 80–1450
Kumakura, K., 80–0896
Kumar, A., 80–1680
Kumar, R., 80–1772
Kung, S. D., 80–0875
Kunze, M., 80–0090, 80–0306, 80–0307, 80–0926, 80–0930, 80–1570, 80–1836, 80–1837
Kunze, M. J., 80–1570, 80–1836, 80–1837
Kuo, K. C., 80–1674
Kurihara, H., 80–0604
Kurihara, T., 80–1216
Kurland, L. T., 80–0756
Kurogochi, Y., 80–0897, 80–1363
Kurt, T. L., 80–0070, 80–1210
Kurta, A. V., 80–0958
Kusano, S., 80–1216
Kuschner, M., 80–0079
Kuter, E., 80–0218, 80–1079, 80–1811
Kuznetsov, V. S., 80–0990
Kwok, L. W., 80–1452

La Voie, L., 80–0824
Labadie, J.-C., 80–1000
†Laboratoire Theranol, 80–0667
Lader, M. H., 80–1772
Lafitte, J. J., 80–1655
Lafuma, J., 80–1132
Lagrue, G., 80–1449, 80–1798
Laharrague, P., 80–0258
Laine, A., 80–1111
Laine, R. A., 80–0224
Lajtha, A., 80–0063
Lakartidningen, 80–0418
Lakha, A. S., 80–0336
Lakowicz, J. R., 80–0057
Laliberte, D., 80–0355
Lam, J. J., Jr., 80–1261
Lam, L.K.T., 80–0068
Lam, S., 80–0333
Lam, S.T.K., 80–1467
Lamarre, M., 80–1587, 80–1588
Lambert, J., 80–0310
Lambert, P. W., 80–1162
Lamontagne, Y., 80–0168, 80–1537, 80–1773
LaMothe, J., 80–0940
Lampman, J. H., 80–1516
Lamprecht, M. P., 80–1589, 80–1590, 80–1591
Lancet, 80–0358, 80–1154, 80–1287, 80–1571, 80–1572
Landecker, K. D., 80–0139
Landmann, H., 80–1279
Lando, H. A., 80–0437, 80–0825
Landrigan, P. J., 80–0880
Lang, W., 80–0903
Lange, H. F., 80–1335
Langenbach, R., 80–0743
Langer, A. M., 80–0326
Langley, R. B., 80–0958

†Patent assignee.

†Lantor of Australia Pty. Ltd., 80–0496
LaPorte, R. E., 80–1209, 80–1450
LaRiviere, W., 80–0308
Larme, P., 80–1629
Larsson, L., 80–0277
Lask, H., 80–0511
Lasogga, F., 80–1774
Laties, V. G., 80–0898
Latiff, A., 80–0903
Latto, I. P., 80–0720
Laurence, D.J.R., 80–0760
Lauwerys, R. R., 80–0334, 80–0386
Lavan, J., 80–1617
Lavrova, I. G., 80–1240
Lawler, P., 80–0770
†Lawrence Peska Associates, Inc., 80–0660
Lawther, P. J., 80–0309
Layne, J. T., 80–1686
Layward, L., 80–0800
Le Bouffant, L., 80–0730
Le Groumellec, A., 80–0731
Le Mesurier, S. M., 80–1123
Leaper, D. J., 80–1707
Leathar, D. S., 80–1241, 80–1775
Leaverton, P. E., 80–0996
Lebas, J., 80–1111
Lebowitz, M. D., 80–0767
Leclercq, J. F., 80–1451
LeCron, L. M., 80–1251
Lee, C. B., 80–0517
Lee, D., 80–0053
Lee, D. K., 80–0266
Lee, I.-S., 80–1393
Lee, J. J., 80–0911
Lee, P. N., 80–1144, 80–1288
Lee, S. D., 80–0877, 80–1627
Lee, W. R., 80–0932, 80–0933
Leeder, S., 80–1431
Leeder, S. R., 80–1526, 80–1691, 80–1701
Leese, C., 80–0760
Lefcoe, N. M., 80–1799
Lefrancois, R., 80–1619
Legg, P. D., 80–0196
Legraverend, C., 80–0727
Lehrer, S. B., 80–0899, 80–1636
Lehtovirta, E., 80–0975
Lehtovirta, P., 80–0384
Leidy, R. B., 80–0203
Leighton, M., 80–0383
Leighton, M. H., 80–0337
Leijdekkers, C.-M., 80–1140
Lellouch, J., 80–1181, 80–1364, 80–1614
LeMarbre, P. A., 80–1115
Lemoine, J. M., 80–0857, 80–1155, 80–1156, 80–1673
Lenfant, C., 80–1573
Lento, A. G., 80–1517
Leon, G. R., 80–1776
Leong, B.K.J., 80–0907
Lepper, M., 80–1142, 80–1730
Lepper, M. H., 80–0113
Leren, P., 80–0966, 80–1714
Lessard, R., 80–0310
Lester, R. L., 80–0224, 80–1600
Leu, R., 80–1825
Leuchtenberger, C., 80–1637
Leuchtenberger, R., 80–1637
Leucuta, S., 80–1375
Lever, A. F., 80–0360
Levi, A. J., 80–0961
Levi, L., 80–1505
Levine, S., 80–0117
Levins, R. J., 80–0228, 80–0693
Levitt, L. J., 80–0073
Levy, B. S., 80–1495
Levy, D. A., 80–1169
Lew, E. A., 80–0917
Lewis, B. S., 80–0116
Lewis, C. I., 80–0519
Lewis, D. J., 80–0732, 80–1638
Lewis, J. D., 80–0383, 80–1389
Lewis, J. L., 80–1466
Lewis, P. H., 80–1501, 80–1757
Li, K.Y.R., 80–1682
Li, R., 80–1171
Liard, R., 80–1184, 80–1420
Liberman, R. P., 80–0169
Lichtensteiger, W., 80–1365
Lichtenstein, E., 80–0834
Liddell, F.D.K., 80–0937, 80–1668
Lifshits, N. A., 80–1182
†Liggett and Myers Inc., 80–0521, 80–0541, 80–0542, 80–0562, 80–0563, 80–0568, 80–0569, 80–0622
†Liggett Group Inc., 80–0522, 80–0545, 80–0593, 80–0594
Light, K. K., 80–0596, 80–0597, 80–0598, 80–0599, 80–0600, 80–0601, 80–1808
Lijinsky, W., 80–1813
Lim, B. C., 80–0517
Lin, J. S., 80–0047
Lin, T. M., 80–1674
Lind, B., 80–0895
Lindall, A. W., 80–1162
Lindberg, H. A., 80–0113, 80–1730
Lindblom, L., 80–1612
Lindell, S.-E., 80–1629
Lindenschmidt, R. R., 80–0733
Lindental, D., 80–0974
Lindesmith, L. A., 80–0846
Lindgarde, F., 80–1629
Lindquist, O., 80–1704
Lindsay, D. W., 80–1337
Line, B. R., 80–1424
Line, W. B., 80–0207
Lingjaerde, O., 80–1366
Link, L. A., 80–1585
Linnman, L., 80–0261
Lipfert, F. W., 80–0291
Lippold, O.C.J., 80–1124
Lisboa, C., 80–0945
Lisitsin, V. Yu., 80–1723
Littleton, J. M., 80–0711
Liu, B. M., 80–1573
Liu, K., 80–1194
Lloyd, D., 80–1526
Locke, F. B., 80–0927
Lockerbie, L., 80–0359
†Loews Theatres, Inc., 80–0514
Loftus, J. J., 80–0858
Logan, R. L., 80–0359
Logue, E. E., 80–1466
Loizeau, E., 80–1399, 80–1409
Loke, J., 80–1185
Lombardo, C., 80–0283
Long, R. C., 80–0005
Longest, W. G., Jr., 80–1289
Longhini, E., 80–0779
Longnecker, D. S., 80–1157
Lonsdale, D., 80–1693
Lorenz, J., 80–1125
Lortscher, R. H., 80–1454
Loschenkohl, K., 80–1656
Loskant, H., 80–1227
Louie, E., 80–0084
Lovkova, M. Ia., 80–0273
Lovlin, R. E., 80–0971
Lowe, G.D.O., 80–0788
Lowe, R. H., 80–0012
Lowhagen, O., 80–0772
Lown, B., 80–1712
Lowrey, A. H., 80–1378
Luban-Plozza, B., 80–1538
Lubawy, W. C., 80–0051, 80–1367
Lubet, R. A., 80–1130
Lucas, A. O., 80–1574
Lucas, Z. J., 80–0289
Lucke, W., 80–1744
Lugliani, R., 80–0328
Luke, J. A., 80–0518
Luke, R.K.J., 80–0046
Lukoszek, A., 80–1176, 80–1415

---

†Patent assignee.

Lumnitz, J. S., 80–0187
Lund-Larsen, P. G., 80–0966, 80–1714
Lundgren, M., 80–0984
Lupien, P. J., 80–1214
Luria, S. M., 80–1483
Luscombe, D. K., 80–1368
Lutz, W., 80–0359
Luurila, O. J., 80–0356
Lykke, A.W.J., 80–1123
Lynch, C. J., 80–0464
Lynn, L. B., 80–0010
Lyon, J. L., 80–0119, 80–1221

Maatela, J., 80–1213
Mabry, L. C., 80–0943
MacArthur, C.G.C., 80–1688
Maccoby, N., 80–0120
Mackay, A., 80–0360, 80–0361
Mackenthun, A. V., 80–1417
MacKenzie, G., 80–1201
Mackenzie, J. S., 80–0270, 80–1639
Macklem, P. T., 80–1411
MacLean, L., 80–1167
MacLean, N., 80–1575
MacLeod, M. C., 80–1096
MacPherson, B. V., 80–0936
MacRae, K., 80–1693
Madden, J. J., 80–0271
Maddison, S. E., 80–0947
Madsbad, S., 80–1232, 80–1745
Maeda, K., 80–0523, 80–0539, 80–0641, 80–1264
Maeda, Y., 80–1216
Maeder, J.-P., 80–1435
Magnus, K., 80–0972
Mahendru, R. K., 80–0121
Maher, F. A., 80–1640
Maheux, B., 80–0310
Mahfouz, M., 80–1350, 80–1576, 80–1720
Maier, F., 80–0445
Majeski, J. A., 80–0794
Majewski, T. E., 80–0646
Makarem, M., 80–0684
Maki, J., 80–0367
Makler, A., 80–1749
Maksud, M. G., 80–1641
Makuch, R. W., 80–0934
Malaia, E. V., 80–1240, 80–1242
Malaty, H. A., 80–1350
Malcolm, S., 80–1518
Malik, I. A., 80–0204
Malinar, M., 80–0774, 80–0918
Maliza, C., 80–1718
Malkin, S. A., 80–1243
Malo, J.-L., 80–0946
Maltzman, B., 80–1153
Mancini, M., 80–0963
Mandl, I., 80–0047
Manes, M., 80–0537
Maness, A. T., 80–1221
Manfreda, J., 80–0937
Mangan, A. J., 80–0778
Mangan, G. L., 80–1777
Mangaraj, D., 80–0554
Mangione, A., 80–0268
Manil, L., 80–0912
Manku, M. S., 80–0967
Manole, G., 80–1080
Mansell, A., 80–0949
Manthey, J., 80–0362
Manz, T., 80–1400
Marc, F., 80–1435
Marcatili, S., 80–0717
Marcel, G. A., 80–1449
Marchok, A. C., 80–0741
Marcus, A. H., 80–1126, 80–1642
Marek, J., 80–0484
Markle, G. E., 80–1839
Marklund, U., 80–0419
Markose, V. T., 80–0006
Marks, I. N., 80–1744
Marmo, E., 80–0717
Marmot, M. G., 80–1452
Marquardt, H., 80–1352
Marquardt, J., 80–1730
Marsh, V. R., 80–1610
Marshall, J. P., 80–1061
Marshall, M., 80–0272, 80–1095, 80–1647
Marshall, M. V., 80–0900
Martell, E. A., 80–0737
Martin, A., 80–1438
Martin, D. C., 80–1473
Martin, D. D., 80–1369
Martin, D. P., 80–1231
Martin, J. C., 80–0730, 80–1369, 80–1473
Martin, J. E., 80–1762
Martin, J. R., 80–1093
Martin, R. H., 80–0556, 80–0642, 80–1737
Martin, R. R., 80–0900, 80–1430
Martinez, R., 80–0427
Marvit, R. C., 80–0826
Masden, R. R., 80–1370
Mashiko, K., 80–1265
Masironi, R., 80–1290
Mateeva, A., 80–1068
Matejevic, D., 80–0734
Matroos, A., 80–0972
Matsubara, N., 80–0902
Matsumura, K., 80–1658
Matsunaga, E., 80–1735
Matsushima, S., 80–0876
Matsushita, H., 80–1601
Matthay, R. A., 80–1112, 80–1174, 80–1185, 80–1643
Matthews, K. A., 80–0970
Mattina, C. F., 80–0019, 80–0175
Mattle, W., 80–1482
Matulionis, D. H., 80–0058, 80–0059
Matzdorff, F., 80–0789
Maxwell, J. C., Jr., 80–0843, 80–1028, 80–1271, 80–1552
Mayo, R., 80–1081
McAlister, A., 80–1255
McAloon, E. J., 80–0973
McCabe, K. D., 80–1481
McCants, C. B., 80–0008
McCartney, P., 80–1200, 80–1713
McCarty, S. W., 80–0642
McClearn, G. E., 80–1334
McClure, S., 80–0251
McClure, W. F., 80–0701
McCulloch, M. W., 80–0739
McDaniel, R. K., 80–0390
McDonald, A. D., 80–1668
McDonald, J. C., 80–0937, 80–1668
McDonald, J. R., 80–1106
McDonald, K. E., 80–0284
McEwan, J., 80–1719
McFadden, M., 80–1008
McFall, R. M., 80–1539
McGeady, J. C., 80–0519
McGill, A. M., 80–1291
McGill, H. C., Jr., 80–1453, 80–1649
McGrath, M. J., 80–0170
McHardy, J., 80–0109
McIlroy, P.D.A., 80–1106
McIntosh, J., 80–1489
McIntyre, K., 80–1703
McKay, C. L., 80–1483
McKean, H., 80–1142
McKee, C. G., 80–1313
McKennis, H., Jr., 80–1613
McKillip, J., 80–1778
McLarty, J. W., 80–0943
McLeay, C. D., 80–1342
McLemore, T., 80–1095
McLemore, T. L., 80–0900, 80–1675
McMahan, C. A., 80–1649

McMillan, G.H.G., 80–0107, 80–1419
McMillan, S. A., 80–1106
McNair, P., 80–1232, 80–1745
McNamara, P. M., 80–1444
McNeil, D., 80–1489
McNeill, H. G., 80–1106
McNicol, M. W., 80–1302
McNulty, J. C., 80–0073
McPherson, I. G., 80–0958
McPherson, K., 80–0377
Mead, J., 80–0335
Meade, P. D., 80–0901
Meade, T. W., 80–0961, 80–1464
Meadows, J. A., III, 80–0773
Means, R. E., 80–0568, 80–0569
Meberg, A., 80–0385
Mechanic, D., 80–0155
Mecoy, R., 80–1577
Medalie, J. H., 80–1624
Medical Journal of Australia, 80–1840
Medical Letter on Drugs and Therapeutics, 80–0395
Mehta, F. S., 80–1486
Mehta, R., 80–0060, 80–0884
Meisinger, R. H., 80–0478
Melissinos, C. G., 80–0335
Mellies, M., 80–0364
Mello, N. K., 80–1005, 80–1519
Melnick, E. L., 80–0100
Menapace, R. H., 80–0438
Mendelson, J. H., 80–1005, 80–1519
Menkes, H. A., 80–1169
Menser, H. A., 80–1069
Merbaum, M., 80–1779
Meredith-Brown, M., 80–0884
Mergens, W. J., 80–0557
Merigan, W. H., 80–0898
Merillon, J. P., 80–1451
Merker, J., 80–0864
Merrett, J. D., 80–1106
Merrill, W. W., 80–1643
Merx, W., 80–0363
Metha, F. S., 80–0989
Mettlin, C., 80–0088, 80–0089, 80–0301, 80–1578
Metz-Marcy, G., 80–1412
Meurman, L. O., 80–1145
Mewborn, C. R., 80–0171
Meyer-Lie, A., 80–0984
Meyer, M. B., 80–1169, 80–1741
Meyers, A., 80–1348
Meyre, R. W., 80–0499
Michael, L. W., 80–0877
Michaeli, D., 80–0904
Miettinen, O. S., 80–1230, 80–1727
Mihatsch, M. J., 80–1400
Mikami, T., 80–0230
Mikhail, M., 80–1720
Mikhail, M. M., 80–1350
Milbrath, J. R., 80–1389
Mild, F., 80–0595
Miles, J.R.W., 80–0703
Milla, M. H., 80–0336
Miller, A., 80–0954
Miller, A. B., 80–1152, 80–1401
Miller, C. W., 80–0602
Miller, G. H., 80–0464, 80–1252, 80–1292
Miller, H. C., 80–0798
Miller, H. N., 80–1371
Miller, J. A., 80–0948, 80–1692
Miller, P. M., 80–0827
Miller, R. H., 80–1029
Miller, R. P., 80–1091
Miller, R. R., 80–1058
Miller, S. F., 80–0794
Miller, S. I., 80–1740
Millman, J. E., 80–1610
Mills, I. H., 80–1780
Milvy, P., 80–0122
Mimica, M., 80–0774, 80–0918
Minei, L. J., 80–1384
Mink, J., 80–1348
Minozhedinova, N. S., 80–0273
Minty, B. D., 80–0770
Mitchell, C., 80–0991
Mitchell, D. M., 80–1676
Mitchell, J. A., 80–0049, 80–0256
Mitchell, R. S., 80–0188, 80–1421
Mitchell, T. G., 80–0558
Mitchell, W. G., 80–0193
Mitchem, A. R., 80–0035
Mitev, P., 80–0799
Mitra, G., 80–1127
Mittelmark, M. B., 80–0408
Mittman, C., 80–0944
Miyake, T., 80–0520
Miyano, M., 80–1601
Miyazaki, M., 80–1110
Mizgala, H. F., 80–0348
Modler, S., 80–0353
Modval, R., 80–1753
Moerl, H., 80–0357, 80–1138, 80–1206
Mohan, M. S., 80–0705
Mohapatra, S. C., 80–1816
Mohlin, B., 80–0396
Mohsenifar, Z., 80–1186
Moilanen, M.-L., 80–1128
Mokhnachev, I. G., 80–1324
Moldenhauer, W., 80–1082
Moldovan, O., 80–1375
Monch, W., 80–0052
Monji, N., 80–1613
Monk, M. A., 80–1207
†Monsanto Ltd., 80–0526
†Montclair Research Corp., 80–0503, 80–0504, 80–0505, 80–0506, 80–0507
Montgomery, R. T., 80–1016
Montiel, M. M., 80–1649
Moody, P. M., 80–1520
Moogalian, C. M., 80–0522
Mookherjee, B. D., 80–0596, 80–0603, 80–0620, 80–0621, 80–0626, 80–1809, 80–1810
Moolgavkar, S. H., 80–0762
Moore, C. J., 80–1096
Moore, J. I., 80–1136
Moore, L. D., 80–0007
Moore, P. K., 80–0711
Morgan, P.G.M., 80–1676
Morgan, W. D., 80–1083
Morgan, W. F., 80–1616
Morgan, W.K.C., 80–0108
Morgenstern, W., 80–0362
Mori, H., 80–0902
Moriarty, J., 80–1204
Morin, M., 80–1132
†Morinaga Milk Industry Co., Ltd., 80–0523
Morioka, M., 80–1216
Morita, N., 80–0897, 80–1363
Morman, J. F., 80–0634, 80–0635, 80–0643
Moroe, M., 80–0604
Morosco, G. J., 80–0274
Morozov, A. B., 80–0990
Morrill, C. G., 80–0070
Morris, L. E., 80–0919
Morrison, A. B., 80–1059
Morrison, A. S., 80–1158
Morrison, B., 80–1152
Morrison, J. A., 80–0364
Moscoso, M., 80–0427
Moser, R. L., 80–0321
Moss, A. J., 80–0123, 80–0420
Moss, D., 80–1194
Moss, E., 80–0933
Mott, G. E., 80–1649
Moulding, T. S., 80–0188
Mrhar, A., 80–0056
Mucklow, J. C., 80–1103
Muehlemann, R., 80–1494

---

†Patent assignee.

Mueller, G., 80–0229, 80–1159, 80–1493
Mueller, K.-M., 80–1159
Mueller, R., 80–1244, 80–1521
Muir, C. S., 80–1151
Mulcahy, R., 80–1204
Mundy, S. D., 80–1551
Munro-Faure, A. D., 80–1437
Munson, P., 80–0940
Muraki, T., 80–0275
Muralidhara Rao, N. K., 80–0844
Muramatsu, M., 80–0230
Murphy, B. P., 80–1219
Murphy, G. P., 80–0301
Murphy, H.B.M., 80–0421
Murphy, K., 80–1785
Murphy, R. D., 80–1511
Murray, E. J., 80–1497
Murray, J. E., Jr., 80–0679
Murray, J. L., 80–1395
Murray, R., 80–0292, 80–1696
Murty, K.S.N., 80–0003, 80–0030, 80–0205
Musher, D. M., 80–1685
Musk, A. W., 80–1689
Mussinan, C. J., 80–0620
Mustafa, K. Y., 80–0336
Mycinska, M., 80–1611
Mylonas, V. A., 80–0206
Mymin, D., 80–0375
Myrsten, A.-L., 80–1781

Naegel, G. P., 80–1643
Naeye, R. L., 80–1474, 80–1475, 80–1476
Nagakura, A., 80–0604
Nagao, M., 80–0280
Nagaraj, G., 80–0031, 80–1084, 80–1325
Nagashima, A., 80–1216
Nageswara Rao, C. R., 80–1085
Nagorski, B., 80–1075, 80–1544
Nagpaul, K. K., 80–1596
Naguib, M. R., 80–1553
Nagy, J., 80–0938
Nagy, J. M., 80–0094
Naik, R. S., 80–0124
Naik, S. R., 80–1133
Naito, N., 80–0230
Nakadate, T., 80–0275
Nakane, M., 80–0231
Nakanishi, Y., 80–1363
Nakashima, T., 80–0897, 80–1363
Namikoshi, H., 80–0659
Naquira, D., 80–0276
Narasayya, K. L., 80–0205
Narasimha Rao, C. V., 80–1326
National Academy of Sciences, 80–0311
National Cancer Institute, 80–0312, 80–1060, 80–1160, 80–1402
National Center for Health Statistics, 80–1211, 80–1477
National Clearinghouse for Smoking and Health, 80–1522
National Heart, Lung, and Blood Institute, 80–1060, 80–1721
National Institute for Occupational Safety and Health, 80–0312, 80–1690
National Institute of Environmental Health Sciences, 80–0312, 80–1579
National Institute on Drug Abuse, 80–1523, 80–1782, 80–1841
Natvig, H., 80–1006
Navratil, J., 80–1768
Neas, I., 80–0207
Nebert, D. W., 80–0727
Neill, D. W., 80–1106
Neittaanmaki, L., 80–0367
Nel, J. G., 80–1591
Nellen, W. J., 80–0497
Neri, M., 80–1602
Nettesheim, P., 80–0065, 80–0281, 80–0719, 80–0729, 80–1121, 80–1660, 80–1661
Neufeld, H. N., 80–1624
Neuman, J., 80–0974
Neumann, G., 80–1580
Neumann, H.-J., 80–0465
Neurath, G. B., 80–0032
Nevasaari, K., 80–0067
Neville, A. M., 80–0760
Nevo, Z., 80–0904
New Zealand Medical Journal, 80–1581
Newhouse, M., 80–1696
Newhouse, M. L., 80–0292
Newman, A., 80–0156
Newman, R. H., 80–0028
Newmark, H. L., 80–0557
Newton, R. P., 80–0550, 80–0551, 80–0553, 80–0559, 80–0560, 80–0561
Newton, R. P., Jr., 80–0605
Nezelof, C., 80–1630
Nguyen, F., 80–0258
Nias, B. C., 80–0361
Nichols, B. C., 80–1585
Nicolaus, G., 80–1076
Nicolle, P., 80–0466
Nicolov, I. G., 80–1644
Nijhuis, H., 80–1594
Nikiforov, N., 80–0799
Nikolov, I., 80–0313
Nikulin, A., 80–0340
Nilsson, B., 80–1484
Nishida, K., 80–1601
Nishida, T., 80–0211, 80–0212, 80–0213, 80–0249, 80–0688, 80–0689, 80–1318, 80–1333
Nishimura, T., 80–0914
Nishio, H., 80–0539
Nishiyama, R., 80–0479
Nissen, C., 80–1196
Nissinen, A., 80–0367
Noble, M. J., 80–1758
Noel, J., 80–1617
Nogawa, K., 80–0250
Noguchi, K., 80–0523, 80–0641, 80–1264
Noguchi, M., 80–0240, 80–0694, 80–0695
Nolan, D. C., 80–1718
Noller, K. L., 80–0756
Noppa, H., 80–1485, 80–1722
Nora, A. H., 80–1454
Nora, J. J., 80–1454
Nordberg, G. F., 80–1362
Nordbo, H., 80–0392
Norden, G., 80–1629
Nordfors, K., 80–1333
Norman, V., 80–0232, 80–0521, 80–0522, 80–0545, 80–0562, 80–0563
Normand, C., 80–0730
North, W.R.S., 80–0961
Novak, R. M., 80–0591
Nuessel, E., 80–0362
Nyman, K., 80–0416
Nyqvist, B., 80–1187

Obel, E. B., 80–1736
O'Brien, I. M., 80–0096
O'Brien, P. J., 80–1377
Obroniecka, L., 80–1811
Ockene, J. K., 80–0422
O'Connell, E. R., 80–0127
O'Connor, A., 80–0987
O'Connor, S. A., 80–0337
Oddoy, A., 80–1524
Oddsson, E., 80–0806
O'Donnell, T. V., 80–1170
Oei, T.P.S., 80–0903
Oesch, F., 80–0912, 80–1125
O'Fallon, W. M., 80–0756

Offensend, F. L., 80–0451
Office of Population Censuses and Surveys, 80–0423
Offord, K. P., 80–1433
Ogasa, K., 80–0523
Ognianov, I., 80–0225
Ogonowski, J. W., 80–0683
O'Herlihy, C., 80–1737
Ohka, T., 80–0280
Ohloff, G., 80–0609, 80–1256
Ohmori, T., 80–0722
Ohnhaus, E., 80–1125
Ohnishi, A., 80–0659
Ohsumi, T., 80–1330
Ohtake, H., 80–1216
Okano, P., 80–1371
Oldendorf, W., 80–1645
Oleynikov, S. P., 80–1723
†Olin Corp., 80–0524, 80–0525, 80–0556, 80–0642
Oliver, J. S., 80–0061
Oliver, M. F., 80–0359, 80–1724
Olivi, H., 80–0945
Ollagnier, M., 80–1099
Olofsson, S.-O., 80–1440
Olsen, J., 80–1669
Olsen, L. K., 80–1786
Olsson, A. G., 80–0359, 80–1455
Omae, T., 80–1456
O'Malley, B., 80–1842
O'Malley, K., 80–1617
O'Malley, P. M., 80–0414
Onat, A., 80–0365
O'Neil, J. A., 80–1162
Ong, G. B., 80–1467
Ono, J., 80–0523
Opherk, D., 80–0362
O'Reilly, H. P., 80–0086
Orel, J. V., 80–0680
Ori, F., 80–0246
Oriente, P., 80–0963
Orjasaeter, H., 80–1335
Orlander, J., 80–0277
Orleans, C. S., 80–0819
Orlien, N., 80–1007
Ornaf, R. M., 80–0024
O'Rourke, T. W., 80–1492
Osborn, S. G., 80–0417
Osman, M. M., 80–0047
Otto, C., 80–1192
Otto, D. A., 80–0253
Owen, P., 80–0766
Owens, W. F., Jr., 80–0033, 80–0524, 80–0525, 80–0556, 80–0642
†P. J. Lorillard, 80–0519
Paarlberg, D., 80–1061
Paddle, G. M., 80–1487
Padgett, S., 80–1444
Paffenbarger, R. S., Jr., 80–0115
Paganelli, R., 80–0800
Paganuzzi, M., 80–0283
Pahren, H. R., 80–1634
Pallonen, U., 80–1255
Palmblad, J., 80–1505
Palmborg, M., 80–0984
Palmer, A., 80–0775
Palmer, C. A., 80–1293
Palmer, R. F., 80–0284
Pandey, A. K., 80–0686
Pandey, R. G., 80–0685
Panek, E., 80–0793
Pangos, E. A., 80–0206
Panico, S., 80–0963
Pannier, R., 80–0338
Papadimitrou, J. M., 80–0892
Papanek, M., 80–0543
Papeschi, G., 80–1372
Parker, C. M., 80–0909
Parker, F. J., 80–0526
Parker, J. A., 80–1016
Parker, J. B., 80–0643
Parkinson, S., 80–1338
Parliament of the Commonwealth of Australia, 80–0467
Pascale, J. V., 80–0608
Pasche, R. C., 80–0527
Pasquis, P., 80–1619
Patel, C., 80–0758
Patel, D. J., 80–1316
Patel, G. J., 80–0006, 80–1316
Patel, U. R., 80–1073
Patel, Y. C., 80–1457
Patra, P. B., 80–1336
Pattabiraman, T. N., 80–0726
Patterson, C. D., 80–1417
Pattillo, R. A., 80–1389
Patton, S., 80–0608
Paul, O., 80–0113, 80–1142, 80–1703, 80–1730
Paulev, P.-E., 80–1104
Paun, D., 80–1279
Pavia, D., 80–0339
Pavlovic, M., 80–0734
Paxton, R., 80–1646
Pearce, S. J., 80–0835
Pearson, K. H., 80–0278
Peat, J., 80–1431
Peat, J. K., 80–1691, 80–1701
Pechacek, T. F., 80–0838
Pederson, L. L., 80–1799
Pedoe, H. T., 80–1726
Peedin, G. F., 80–0008, 80–1585
Peeva, I., 80–0799
Pein, F. G., 80–0032
Pelkonen, O., 80–0067, 80–0727, 80–1128, 80–1225
Pellegrin, F., 80–0133, 80–1739
Pemberton, J., 80–1201
Penaloza, R., 80–0279
Pendlebury, D. J., 80–0383
Pene, C., 80–1253
Penven-Guilleau, R., 80–0247
Pepperell, R., 80–0259
Pequignot, G., 80–1229
Perdrizet, S., 80–0405, 80–1184, 80–1412, 80–1420
Pereira, R. P., 80–0342
Pereira, W., 80–1116
Perinelli, M. A., 80–0034
Perkins, P. R., 80–0528
Perkinson, L. B., 80–1550
Perks, W. H., 80–0096
Permutt, S., 80–1169
Perrault, J. L., 80–0946
Perrier, D. G., 80–1367
Persaud, T.V.N., 80–0733
Pershagen, G., 80–0261
Pertschuk, M. J., 80–1783
Petering, H. G., 80–0877
Peterson, W. A., 80–0880
Pethybridge, R., 80–1419
Pethybridge, R. J., 80–0107
Petit, M., 80–0424
Petkova-Bocharova, T., 80–0313
Peto, R., 80–0949
Pettersson, B., 80–0735
Petty, T. L., 80–1421
Peychev, P., 80–0799
Pfaffinger, I., 80–1647
Pflugmacher, J., 80–1327
Pharmaceutical Journal, 80–1582
Phelan, J. P., 80–0986
Phelpstead, J.W.P., 80–0495
†Philip Morris Inc., 80–0509, 80–0510, 80–0529, 80–0552, 80–0555, 80–0564, 80–0584, 80–0585, 80–0586, 80–0610, 80–0613, 80–0614, 80–0637, 80–0646, 80–0669, 80–1817
Phillips, D. F., 80–0118
Phillips, R., 80–1718
Pickett, A. B., 80–0308
Piechocki, D., 80–1442
Pierce, G. E., 80–0821
Pikula, B., 80–0340
Pilotti, A.-M., 80–0211

†Patent assignee.

Pimmel, R. L., 80–0331
Pindborg, J. J., 80–0989, 80–1486
Piolatto, G., 80–0292, 80–1696
Piper, D. W., 80–0139, 80–1489
Piscator, M., 80–0250
Pissiotas, G., 80–0472
Pitkar, V. K., 80–0989
Pitner, T. P., 80–0233
Pittet, A. O., 80–0606, 80–0607, 80–0608
Pizzo, S. V., 80–1466
Plamenac, P., 80–0340
Pleban, P., 80–1373
Pleban, P. A., 80–0278
Pleho, A., 80–1374
Pleuvry, B. J., 80–0947
Plummer, J. L., 80–0881, 80–0908
Pochay, V. E., 80–1116, 80–1626
Poddar, M. K., 80–1127
Podolskaya, O. V., 80–1723
Poehl, H.-G., 80–0446
Poellmann, L., 80–0397
Pokorny, C., 80–0780
Polak, E., 80–1129
Pollack, R. L., 80–0696
Pollard, J., 80–0188
Polsky, R. H., 80–0425
Pomerleau, O. F., 80–1783
Ponteva, M., 80–0366
Pool, J. L., 80–0468
Poole, A. D., 80–1800
Pop, R., 80–1375
Porsius, A. J., 80–1376
Porter, G., 80–0636
Post, C., 80–0736
Poston, R. N., 80–0790
Poulson, R., 80–0644, 80–0645
Poulton, P., 80–1784
Poupy, J.-L., 80–1254, 80–1798
Powell, D. R., 80–0837
Powell, G. E., 80–1525
Powell, L. W., 80–1615
Prabhu, S. R., 80–1314
Prah, J. D., 80–0253
Prasad Rao, J.A.V., 80–0003, 80–1085
Prasad, S. S., 80–0003
Prat, J. C., 80–1361
Pratt, P. C., 80–0948, 80–1692
Prentice, D. E., 80–0732, 80–1638
Preston, S. H., 80–0754
Pretorius, C.J.H., 80–1589, 80–1590, 80–1591
Price, L., 80–1343
Pride, N. B., 80–0342, 80–0949
Prineas, R. J., 80–1212
Prior, I.A.M., 80–0345
Pritchard, J. A., 80–0558
Procak, M., 80–1811
Prohoroff, J. P., 80–0508
Proni, T. J., 80–0821
Prough, R. A., 80–1130
Prouse, R. E., 80–0644, 80–0645
Prskalo, M., 80–1374
Pruett, J. J., 80–0904
Prugnaud, R. L., 80–0667
Pryjma, K., 80–1702
Przybylkiewicz, Z., 80–1702
Public Health Reports, 80–1161
Pugin, V. S., 80–0671
Pullan, P. T., 80–0140, 80–0259
Pullella, J. L., 80–1770
Punsar, S., 80–0975
Purcell, I., 80–1526
Purchase, I.F.H., 80–1487
Puska, P., 80–0367, 80–1220, 80–1255
Putman, C. E., 80–1185
Putz, V. R., 80–1131
Pyorala, K., 80–0975, 80–1213

Quanjer, P. H., 80–1189
Querleux, E., 80–1181
Queval, P., 80–1132
Quinn, A. D., 80–0627
Qureshi, S., 80–0968

†R. J. Reynolds Tobacco Co., 80–0500, 80–0579, 80–0602, 80–0611, 80–0639, 80–0649, 80–0650, 80–0651, 80–0652, 80–0653, 80–0654, 80–0655, 80–0656, 80–0657
Rabinowitz, B. O., 80–1210
Rademaker, A. W., 80–0971
Radford, E. P., 80–0737
Radhakrishnamurthy, B., 80–0870
Radow, B., 80–1369
Radwan, L., 80–1422
Radziszewski, E., 80–0409, 80–1625, 80–1765
Rafael'skaia, G. I., 80–0905
Rahimtula, A. D., 80–1377
Rainer, N. B., 80–0510, 80–0529, 80–0564, 80–0646, 80–1817
Rall, D. P., 80–0312
Ramachander, P. R., 80–0030
Ramadas, D., 80–0800
Ramcharan, S., 80–0133, 80–1739
Ramsay, L. E., 80–0368
Rana, S.V.S., 80–0062
Rand, M. J., 80–0739
Rankin, D. W., 80–1502
Rantakallio, P., 80–0134, 80–1738
Rattner, B. A., 80–0879
Ravnik, V., 80–0692
Rawbone, R. G., 80–1294, 80–1527, 80–1693, 80–1785
Rawlins, M. D., 80–1610
Raymond, L., 80–1399, 80–1409
†Raymond Lee Organization, Inc., 80–0661
Razeghi, M., 80–0011
Rebel-De Vries, H. A., 80–0369
Reddy, M.C.M., 80–0681
Reddy, M. S., 80–1133
Reddy, N. S., 80–0009
Redican, K. J., 80–1247, 80–1786
Reed, D., 80–0310
Reed, G., 80–1740
Rees, P. J., 80–0738
Rees, R. O., 80–0778
Reeves, R. G., 80–1328
Regan, T. J., 80–0125
Regli, J., 80–1423
Rehnlund, S. O., 80–0314
Reibel, J., 80–1486
Reid, W. W., 80–1333
Reif, A. E., 80–0928
Reilly, J. J., 80–0007
Reisch, W., 80–1086
Renaud, S., 80–0370
Renold, W., 80–0609
Renzetti, A. D., Jr., 80–0775
Renzi, G., 80–0946
Repace, J. L., 80–1378
Repine, J. E., 80–1115
Reunanen, A., 80–1213
Reuther, E., 80–0357
Reuther, R., 80–0357
Reynolds, A.-M., 80–1715
Reynolds, H. Y., 80–1643
Reynolds, J. H., 80–0234
Reynolds, M. L., 80–1017
Reznick-Schueller, H. M., 80–1134
Reznik, G., 80–1648
†Rhodia AG, 80–1807
†Rhodia, Inc., 80–0473
Richard, J., 80–0962
Richard, J.-L., 80–1229
Richards, J. G., 80–1365
Richardson, C., 80–1725
Richardson, C. R., 80–1487
Richardson, M. F., 80–0315
Richmond, J. B., 80–1295
Richter, M., 80–0235, 80–0236
Rickert, W. S., 80–1603

---

†Patent assignee.

Riedesser, W., 80–1818
Riemersma, R. A., 80–0359
Rifkind, B. M., 80–0379
Rigney, K. B., 80–0826
Riis, P., 80–0806
Rimm, A. A., 80–1205
Rimm, D. C., 80–1755
Rimpela, M., 80–0426, 80–1062, 80–1245
Ringsdorf, W. M., Jr., 80–1339
Rios, J. C., 80–1718
Riotton, G., 80–0761
Rissanen, A. M., 80–0126
Rist, M., 80–1400
Ritchie, J.W.K., 80–0135
Ritzel, G., 80–1193
Rivenson, A., 80–0891
Rivero, O., 80–1694
Rix, C. E., 80–0602
Roberts, D. L., 80–0579
Robertson, F. W., 80–0371
Robertson, G. B., 80–1379
Robertson, G. L., 80–1653
Robertson, J.I.S., 80–0360
Robillard, D., 80–1296
Robinson, J. C., 80–1603
Robinson, M., 80–1693
Robinson, R. C., 80–1371
Robitaille, N.-M., 80–1214
Robles, R., 80–0427
Rochon, J., 80–1214
Rodarte, J. R., 80–0773
Rodgers, M. P., 80–0439
Rodgers, R. J., 80–1135
Rodgers, R. L., 80–1136
Rodriquez, L., 80–1383
Rodway, J. S., 80–1252
Roed-Petersen, B., 80–1486
Roeder, W., 80–1166
Roels, H. A., 80–0334, 80–0386
Rogers, A. W., 80–1379
Rogers, J. D., 80–0035
Rogers, R. W., 80–0171
Rogers, W. E., 80–0010
Rogers, W. R., 80–1649
Rogot, E., 80–1395
Rogowsky, M., 80–1650
Rohl, A. N., 80–0326
†Rohm and Haas Co., 80–0481
Roland, J., 80–0288
Roman, T., 80–1544
Romanski, B., 80–0976
Romo, M., 80–0791
Ronen, I., 80–1749
Roos, B. A., 80–1162
Rosa, N., 80–1070, 80–1329, 80–1592
Rosazza, J. P., 80–0282
Rose, G., 80–1177, 80–1200, 80–1726
Rose, G. A., 80–0977
Rosecrans, J. A., 80–0040
Roselius, W., 80–0565
Roseman, J. M., 80–1191
†Rosen Enterprise, Inc., 80–0531
Rosen, W. E., 80–0530, 80–0531
Rosenbaum, M., 80–1013
Rosenberg, D. M., 80–1424
Rosenberg, L., 80–1215, 80–1230, 80–1727
Rosenfield, A., 80–0372
Rosenkrantz, H., 80–1651
Rosenman, K. D., 80–0978
Rosin, I., 80–1718
Rosner, B., 80–0777, 80–1215, 80–1712
Ross-Townsend, A., 80–0142
Ross, W. S., 80–1843
Rossi, S., 80–1602
Rossiter, C. E., 80–0776, 80–1695
Rossman, M. D., 80–0324
Rossner, S., 80–0359, 80–1455
Rostenberg, I., 80–0279
Rotenberg, K. S., 80–1652
Roth, D. S., 80–0647
Rothman, K. J., 80–1215, 80–1403
†Rothmans of Pall Mall (Australia) Ltd., 80–0496
†Rothmans of Pall Mall Canada Ltd., 80–0493
Roussel, P., 80–1655
Rowe, J. W., 80–1653
Rowland, J., 80–1659
Rowlands, S., 80–0971
Rowley, M. P., 80–0802
Rowsell, D. G., 80–0623, 80–0624
Roy, P. E., 80–0355
Rozelle, R. M., 80–0408
Ruasse, J.-P., 80–1250
Rubino, G. F., 80–0292, 80–1696
Rudd, R. W., 80–1063
Rude, T. H., 80–1122
Rudner, B., 80–0566
Rudra, L., 80–0683
Rumeau-Rouquette, C., 80–0382
Rundberg, E.G.S., Jr., 80–0610
Ruosteenoja, R., 80–0791
Rupp, W. M., 80–0940
Ruschhaupt, W. F., III, 80–0118
Rush, D., 80–0387, 80–1478
Rush, W. A., 80–0830
Rushton, J.-M., 80–0333
Russell, M.A.H., 80–1137, 80–1246, 80–1772, 80–1790
Russfield, A. B., 80–0883
Rutenfranz, J., 80–1227
Rutishauser, G., 80–1400
Ruzzon, T., 80–0283
Ryan, L. J., 80–1654
Ryan, W. P., 80–0496
Rybin, I. S., 80–1226
Rylander, R., 80–1380

Sabir, S. S., 80–0036
Sablonniere, B., 80–1655
Sabroe, S., 80–1669
Saccomanno, G., 80–0079, 80–0281
Sachs, D.P.L., 80–0786, 80–0838
Sackner, M. A., 80–0950
Sacks, S. T., 80–1452
Saffiotti, U., 80–0312
Sahley, T. L., 80–0906
Said, M., 80–0190
Sakamura, S., 80–0695
Sakashita, N., 80–0476, 80–0479
Sakuma, H., 80–1087, 80–1330, 80–1331
Sakuma, K., 80–1216
Sakurai, T., 80–1265
Saleh, M. M., 80–0684
Salonen, J., 80–0367, 80–1220
Salonen, J. T., 80–1728
Saloojee, Y., 80–1246, 80–1790
Salsbury, A. J., 80–1176, 80–1415
Salvaggio, J. E., 80–0899, 80–1636
Samet, J. M., 80–0777
Sampson, V., 80–0345
Samson, H., 80–0355
Samuel, J. L., 80–1451
Sande, H., 80–0385
Sanders, B., 80–1654
Sanders, C. L., 80–1141
Sanders, E. B., 80–0693
Sanders, J. M., 80–0589, 80–0596
Sanford, R. A., 80–0532
Sanmarco, M. E., 80–1706
Sanmarco, M. I., 80–0970
Sant, D., 80–1511
Santora, P. B., 80–0828
Sanyal, S., 80–1336
Sao, V. K., 80–0124
Sapolsky, H. M., 80–0189
Sarantos-Laska, C., 80–0739
Saric, M., 80–0774
Sasagawa, S., 80–0072
Sasse, E. A., 80–1389
Sastry, A. S., 80–0030, 80–0205

†Patent assignee.

Satiani, B., 80–1217
Sato, S., 80–0280, 80–0641
Sato, Y., 80–0641
Satz, N., 80–1463
Saudou, G.-F., 80–1000
Sauer, R. A., 80–0821
Saunders, J. H., 80–0712
Saunders, J. L., 80–0192
Saunders, J. W., 80–1381
Savolainen, E., 80–0975
Sawada, H., 80–0533, 80–0538
Saxton, D. W., 80–0136
Scalese, S., 80–0283
Scansetti, G., 80–0292, 80–1696
Schaanning, J., 80–0951
Schachter, S., 80–1528
Schaefer, K. E., 80–1663
Schaer, M., 80–1787
Schafer, P. W., 80–0050, 80–0265
Schanberg, S. M., 80–1705
Scharf, M. B. 80–0807
Scharfman, A., 80–1111, 80–1655
Schechtman, L. M., 80–1122
Schentag, J. J., 80–0268
Schepartz, A. I., 80–1819
Scheuenstuhl, H., 80–0904
Schievelbein, H., 80–0469, 80–1458, 80–1656
Schiffman, P., 80–0328
Schiltz, P., 80–0237, 80–0238
Schinke, S. P., 80–1788
Schlegel, J., 80–1656
Schlotzhauer, W. S., 80–0037, 80–1819
Schmassmann, H., 80–1125
Schmeltz, I., 80–0022, 80–0708, 80–1332
Schmid, H., 80–0724
Schmidt, F., 80–1030, 80–1297, 80–1404
Schmidt, J. A., 80–0208
Schmidt, O. K., 80–0534, 80–1545
Schmidt, U., 80–0724
Schmitt, F. L., 80–0590
Schneider, R. E., 80–1382
Schneiderman, M. A., 80–0312, 80–1283
Schoberberger, R., 80–1570
Schoenberg, J. B., 80–1425
Schoenberger, J. A., 80–1731
Schoenfelder, P. G., 80–0940
Schoenfisch, W. H., 80–1657
Schottenfeld, D., 80–0132
Schouten, M., 80–1826
Schreiber, H., 80–0281
Schreiber, W. L., 80–0596
Schrimpf, W. J., 80–1344
Schroll, M., 80–1729
Schuettmann, W., 80–0316
Schulte-Elte, K.-H., 80–0609
Schulzer, M., 80–1167
Schumacher, J. N., 80–0611
Schumaker, J., 80–0464
Schwartz, D., 80–0388
Schwartz, J. L., 80–1014
Schwarz, W., 80–1279, 80–1529
Schwebel, A. I., 80–0157
Schweizer, W., 80–1196
Schwetz, B. A., 80–0907
Sciandra, R., 80–0301
Scotch, N., 80–0117
Scott, M. P., 80–0716
Scrimgeour, W. G., 80–1799
Sculley, M. J., 80–0707
Scursatone, E., 80–1621, 80–1622, 80–1623
Seeger, W., 80–0668
Seehofer, F., 80–1820
Seethalakshmi, L., 80–1340
Segi, M., 80–0927
Seibel, B. L., 80–1801
Seid, D. A., 80–0715
Seideman, N., 80–1298
Seidenschnur, G., 80–0381
Seidman, H., 80–1394, 80–1397
Seitzman, L. H., 80–0943
Sekin, S., 80–1604
Sele, J., 80–1107
Selikoff, I. J., 80–0326, 80–0954, 80–1394, 80–1405
Selke, W. A., 80–0019, 80–0175
Selkirk, J. K., 80–1096
Sellars, S. L., 80–0317
Sellers, M. L., 80–1005, 80–1519
Sellman, E. J., 80–0496
Seltzer, C. C., 80–0293, 80–1064
Selvester, R. H., 80–0970, 80–1706
Selvin, S., 80–0172
Selzer, R. H., 80–1706
Semple, P. d'A., 80–0765
Sen, D., 80–0526
Serebryakova, I. K., 80–0979
Sershen, H., 80–0063
Sertain, G., 80–0852
†Service d'Exploitation Industrielle des Tabacs et des Allumettes, 80–1259
Sesti, A. G., 80–1372
Setzer, J. V., 80–1131
Severson, H. H., 80–0435
Severson, R. F., 80–0038, 80–0709, 80–0740, 80–0874
Seward, J. B., 80–1433
Sewchand, L. S., 80–0971
Sexstone, J. H., 80–0535
Sexton, G. J., 80–0094, 80–0095
Shader, R. I., 80–1108
Shafer, D. A., 80–0271
Shaffer, J. W., 80–0828
Shah, H. M., 80–1316
Shambhu, M. B., 80–0841
Shand, D. G., 80–0913
Shanmugaratnam, K., 80–1677
Shannon, I. L., 80–0239
Shapiro, S., 80–1230, 80–1727
Shapovalov, E. N., 80–1546
Sharpless, S. K., 80–1654
Shatalova, A. P., 80–1240
Shaw, H. A., 80–1481
Shawe, F. J., 80–1590, 80–1591
Shea, J., 80–0893
Shea, J. W., 80–1116, 80–1626
Sheahan, N. F., 80–0339
Sheen, S. J., 80–1071, 80–1320
Sheers, G., 80–0107, 80–1419
Sheets, T. J., 80–0203
Shekelle, R. B., 80–1142, 80–1718, 80–1731
Shekelle, S., 80–1731
Shelley, E., 80–0109
Shephard, R.J., 80–0071, 80–0162, 80–0968, 80–1218, 80–1426, 80–1518, 80–1697
Shiffman, S. M., 80–1802
Shimojima, N., 80–1087
Shinozuka, H., 80–1157
Shiono, P. H., 80–0133, 80–1739
Shiota, K., 80–1735
Shipley, M. J., 80–1177, 80–1200
Shishova, A. M., 80–1723
Shoop, G. J., 80–0010
Shorey, J. M., 80–1343
Shors, E. C., 80–1658
Shugars, O. K., 80–1031
Shurtleff, D., 80–0792
Shuster, E. J., 80–0587, 80–0588, 80–0591, 80–0592, 80–0596, 80–0597, 80–0598, 80–0599, 80–0600, 80–0601, 80–0603, 80–0606, 80–0607, 80–0627, 80–1808
Siabatto-Pinto, T., 80–0199
Sichinava, R. B., 80–1324
Siebenschein, R., 80–1463
Sieczkowski, J., 80–0582
Siegelaub, A. B., 80–1441
Siegenthaler, W., 80–1463
Siest, G., 80–0793

---

†Patent assignee.

Sievert, R. C., 80–0209, 80–0210
Sikka, K. K., 80–0121
Sillett, P. W., 80–1302
Siltanen, P., 80–0975
Silva, C., 80–1623
Silverman, F., 80–1426, 80–1697
Silvers, G. W., 80–1421
Simon, L., 80–0731
Simon, M. R., 80–0890
Simon, N., 80–1715
Simon, S. J., 80–0816
Simonsson, B. G., 80–0303
Simpson, M. I., 80–0593, 80–0594
Sinclair, D.J.M., 80–1427
Sindelar, R. D., 80–0282
Singer, G., 80–0903
Singh, B. L., 80–0685
Singh, V., 80–0785
Sinha, R., 80–0686
Sipal, Z., 80–1383
Siroka, A., 80–1768
Sirotenko, A. A., 80–1324
Sirtori, C., 80–0283
Sisken, J. E., 80–1387
Sixt, R., 80–1179
Sjostrand, M., 80–1380
Skarin, A. T., 80–1146
Skorianetz, W., 80–0609, 80–0612
Slanski, J. M., 80–0498
Slater, P., 80–1844
Slone, D., 80–1230, 80–1727
Smidt, U., 80–0341
Smiley, J. H., 80–0012
Smith, A. B., 80–0536
Smith, B. R., 80–0881, 80–0908
Smith, D. A., 80–0859
Smith, D. B., 80–0091
Smith, D. J., 80–1198
Smith, E. M., 80–1678
Smith, F. A., 80–0907
Smith, G. G., 80–0909
Smith, G.J.W., 80–1185
Smith, H., 80–0061
Snodgrass, D. R., 80–0900
Snook, M. E., 80–0037, 80–0038, 80–0710, 80–0740, 80–1088, 80–1319
Soboleva, L. G., 80–1182
Soghikian, K., 80–0167
Sokol, R. J., 80–1740
Solberg, L. A., 80–1408
Soldatos, C. R., 80–0807
Sonnenberg, A., 80–1482
Sontag, S. J., 80–1347
Soothill, J. F., 80–0800
Sorbello, A., 80–1188
Sornberger, G. C., 80–1116, 80–1618, 80–1626
Sosnowski, R. G., 80–1122
Soule, D. M., 80–1032
Southwick, E. W., 80–0594
Spangler, R. D., 80–1454
Specht, W., 80–1089
Spector, K. M., 80–1718
Speizer, F. E., 80–0098, 80–0767, 80–0952, 80–1215
Spence, J. W., 80–0702
Spencer, B. M., 80–0599, 80–0600, 80–0601
Spencer, R. M., 80–0040
Spengler, J., 80–0952
Spengler, J. D., 80–0098
Spiegel, H. E., 80–0934
Spillman-Thulin, I., 80–1845
Spivey, C. G., 80–0943
Spohr, U., 80–1138, 80–1206
Sporri, H., 80–0633
Sprecker, M. A., 80–0590, 80–0591, 80–0596
Sprince, H., 80–0909
Sprince, N. L., 80–1698
Spring, D. J., 80–0624
Stadel, B. V., 80–1718
Staehelin, H. B., 80–1193
Stahnke, G., 80–0015
Stamler, J., 80–0113, 80–0373, 80–0980, 80–1142, 80–1194, 80–1459, 80–1460, 80–1730, 80–1731
Stamler, J., 80–1461
Stamler, R., 80–1460, 80–1461, 80–1730, 80–1731
Stanescu, D., 80–0334
Stanford, R. E., 80–1421
Stanhope, J. M., 80–0778
Stanislawczyk, K., 80–0082
Stanulovic, D., 80–1299
Staples, R. E., 80–0907
Starke, G. R., 80–0475
Staszewski, J., 80–1406
Staubesand, J., 80–1647
†Stauffer Chemical Co., 80–1541
Staugard, F., 80–0981
Stead, N. W., 80–1466
Steck, W., 80–1206
Steele, V. E., 80–0741
Stefanov, D., 80–1068
Stein, H. P., 80–1281
Stein, Z., 80–1478
Steinau, P., 80–1821
Steinmetz, J., 80–0793
Steinmetz, J. R., 80–0982
Stekhun, F. I., 80–1488
Stellman, S. D., 80–1164, 80–1407
Stenback, F., 80–1659
Stenhouse, N. S., 80–1219
Stephan, E., 80–0374
Stephens, R. M., 80–0064
Stephenson, M. G., 80–1315
Stepney, R., 80–0860, 80–1751
Sterboul, J., 80–1233
Sterk, P. J., 80–1189
Sterling, T. D., 80–1147
Stevens, C. A., 80–1112, 80–1174
Stevens, D. J., 80–1033
Stevens, E., 80–1730
Stevens, M. H., 80–0910
Stevens, R. G., 80–0762
Stevens, U., 80–0760
Stevenson, A., 80–0183
Stewart, B. W., 80–1123
Stewart, R. A., 80–1525
Stewart, R. D., 80–0014
Stiksa, G., 80–0303
Stimmel, B., 80–1462
Stober, J. A., 80–1490
Stoeber, W., 80–1323
Stoeppler, M., 80–0362
Stoertzer, T., 80–0864
Stoianov, I., 80–0313
Stoichev, I., 80–0313
Stokely, J. R., 80–0023
Stokholm, J., 80–1416
Stoll, M., 80–1256
Stolley, P. D., 80–1718, 80–1727
Stone, D. B., 80–1786
Stone, J. D., 80–1789
Stoner, G. D., 80–0265
Stovall, J. C., 80–1034
Stovall, J. G., 80–1024
Strackee, J., 80–0972
Strasser, P. H., 80–1398
Stratton, T. T., 80–0435
Strauer, B. E., 80–1436
Streiff, R. R., 80–0973
Streissguth, A. P., 80–1473
Streja, D., 80–0375
Strober, W., 80–1643
Strong, J. P., 80–1457
Strubel, D. G., 80–0648
Struelens, B. S., 80–1657
Stuart, B. O., 80–0284, 80–1141
Stuart, R. B., 80–0991
Studer, A., 80–1463
†Studiengesellschaft Kohle mbH, 80–0565
Stumbles, C., 80–1800
Subramanyam, M., 80–0030

†Patent assignee.

Sudan, B.J.L., 80–1233
Sugawara, S., 80–0026, 80–0027, 80–0876, 80–1087, 80–1322, 80–1330, 80–1331
Sugimura, T., 80–0714
Sula, J., 80–0285
†Sumitomo Chemical Co., Ltd., 80–0479, 80–0520
Summerfield, A. B., 80–0820
Sundt, E., 80–0581
Suplisson, A., 80–0370
Suryanarayana, T. K., 80–1314
Susser, M., 80–1478
Sutherland, E. A., 80–0832
Sutorius, D. J., 80–0794
Sutton, S. R., 80–0145, 80–0146, 80–0147, 80–0440, 80–1246, 80–1761, 80–1790
Suzuki, K., 80–1384
Suzuki, S., 80–1428
Svendsen, T. L., 80–1465
†Svenska Tobaks AB, 80–0483
Swann, P. G., 80–0077
Swanson, G. T., 80–1822
Swanson, H. R., 80–0045
Swanson, M., 80–0089
Sweeney, R. R., 80–0161, 80–0955
Sweet, J. B., 80–0808, 80–0992
Swiatkowski, M., 80–0976
Swynghedauw, B., 80–1451
Sylwester, D. L., 80–0936
Syme, S. L., 80–1452
Szafranski, W., 80–1683

Tabak-Journal International, 80–1554
Tafro, S., 80–1374
Tagliacozzo, R., 80–1791, 80–1792
Tai, H., 80–0193
Tajik, A. J., 80–1433
Takagi, Y., 80–0240, 80–1605
Takahashi, R., 80–0476, 80–0479
Takalo, T., 80–0367
†Takasago Perfumery Co., Ltd., 80–0604
†Takeda Chemical Industries, Ltd., 80–0641
Takemoto, N., 80–1265
Takiff, H. E., 80–1115
Talmassons, G., 80–0763
†Tamag Basel AG, 80–0544, 80–0632
Tamir, I., 80–0379
Tamura, M., 80–1699
Tancogne, J., 80–0241, 80–0242, 80–0243, 80–0244
Tang, C.-K., 80–0758
Taniguchi, T., 80–0911
Tashkin, D. P., 80–1186
Tate, M. E., 80–1785
Tattersall, S. F., 80–0342, 80–0949
Taus, V., 80–1753
Taylor, C., 80–0428
Taylor, D. H., 80–1530
Taylor, R. P., 80–0534
Taylor, W. H., 80–0993, 80–1746
Tecklenburg, U., 80–1244
Teel, R. W., 80–0742
†Teledyne Industries, Inc., 80–1822
Telford, R., 80–1610
Tellier, L., 80–1747
Teng, L. C., 80–0613
†Tenneco Chemicals, Inc., 80–0566
Terreni, A., 80–1372
Terrill, T. R., 80–0007
Terris, M., 80–1718
Terzaghi, M., 80–0065
Testa, A., 80–1628
Theroux, P., 80–0348
Theuws, J.L.G., 80–1140
Thevenin, M., 80–1364, 80–1614
Thevenon, C., 80–0370
Thiel, P. G., 80–0746
Thilander, B., 80–0396
Thilly, C., 80–0969
Thilly, W. G., 80–0054
Thomas, C. B., 80–0828
Thomas, C. E., 80–1606
Thomas, D. B., 80–0086
Thomas, J., 80–1489
Thomas, P. C., 80–0127
Thompson, D. J., 80–1231
Thompson, J. F., 80–1022
Thompson, J. W., 80–1610, 80–1751
Thompson, L. W., 80–1705
Thompson, M. E., 80–0829
Thompson, P. D., 80–0066
Thomson, M., 80–0359
Thorgeirsson, T., 80–0806
Thornton, J., 80–0954
Thornton, R. E., 80–1531
Threatt, H. C., Jr., 80–0207
Thuillier, A., 80–1747
Tibblin, G., 80–0457
Tien, Y. K., 80–0335
Tiggelbeck, D. D., 80–0537
Tillkes, M., 80–1089
Tirmarche, M., 80–1679
Tobacco International, 80–0861, 80–0865, 80–1035, 80–1036, 80–1065, 80–1266, 80–1272, 80–1273, 80–1300, 80–1555, 80–1823, 80–1827, 80–1828, 80–1829
Tobacco Reporter, 80–0447, 80–0862, 80–1037, 80–1274, 80–1301, 80–1556
Tobacco Tax Council, 80–1275
Tockman, M. S., 80–0767, 80–0953, 80–1169
Toda, N., 80–1110
Tokarz, S., 80–1072
Tokida, A., 80–1264
Tokunaga, Y., 80–0275
Tola, S., 80–0318
Tomari, H., 80–1265
Tomaszewski, J. E., 80–1134
Tomita, H., 80–0230
Tompa, A., 80–0743
Tompkins, E. A., 80–0753
Tong, J. E., 80–0429
Tongas, P. N., 80–1803
Tonnel, A. B., 80–1655
Toole, J. F., 80–0795
Topping, D. C., 80–0729, 80–1121, 80–1660, 80–1661
Torjussen, W., 80–0110, 80–1408
Tormo Alfonso, V., 80–0376
Townsend, E. J., 80–0142
Townsend, J. L., 80–1464
Toyama, T., 80–1429
Toyoshima, I., 80–0538
Tramel, J. L., Jr., 80–0201
Transbol, I., 80–1232, 80–1745
Trap-Jensen, J., 80–1465
Travis, C. C., 80–1385
Trell, E., 80–1629
Tremoleda Dilmer, M., 80–0662
Trenkle, R. W., 80–0603
Triest, F. J., 80–1257
Tripathi, B. P., 80–0124
Tripathi, S. N., 80–0685
Trodahl, J. N., 80–0239
Troll, C., 80–1656
Trong-Lang, N. T., 80–0245
Trow, J. M., 80–1595
Troyer, R. J., 80–1839
Trudel, G., 80–0168
Trump, B. F., 80–0039, 80–0050, 80–0265
Tseng, C. Y., 80–0592, 80–0596
Tsibris, J.C.M., 80–1139

---

†Patent assignee.

Tso, T. C., 80–0024, 80–0443, 80–0708, 80–1074, 80–1332, 80–1565, 80–1846
Tsong, Y., 80–1194
Tsoukatos, M. P., 80–1018
Tsuji, K., 80–0280
Tsujimoto, A., 80–1102
Tu, S. M., 80–1674
Tuimala, R., 80–1225
Tuncalp, S., 80–1492
Tuomilehto, J., 80–0367, 80–1220
Turberville, C., 80–0760
Turino, G. M., 80–0047
Turndorf, H., 80–0758
Turner, A. S., 80–0958
Turner, D. M., 80–0093
Turner, J.A.M., 80–1302
Turner, J. D., 80–1709
Turner, P., 80–0835
Turner-Warwick, M., 80–1176, 80–1415
Turpin, G., 80–1199
Turton, C.W.G., 80–1176, 80–1415
Tye, C. Y., 80–1677
Tyrer, J. H., 80–1342
Tyroler, H. A., 80–1710

U.S. Consumer Product Safety Commission, 80–0470
U.S. Department of Agriculture, 80–0179
U.S. Department of Health, Education, and Welfare, 80–0471, 80–1303, 80–1304, 80–1305
U.S. Environmental Protection Agency, 80–0878
U.S. House of Representatives, 95th Congress, 2nd Session, 80–1040
U.S. Senate, 95th Congress, 2nd Session, 80–1558
U.S. Senate, 95th Congress, 1st Session, 80–1559
U.S. Supreme Court, 80–1041
Ubell, E., 80–0113
Uchida, E., 80–0744
Uchimiya, H., 80–1607
Ueda, A., 80–1216
Ueda, K., 80–0795
Uehara, M., 80–0539
Uhl, R. G., 80–0552, 80–0555
Ulus, I. H., 80–1386
Umamaheswara Rao, M., 80–1073
Umney, N. D., 80–0711
Undeutsch, K., 80–1227
†Union Carbide Corp., 80–0475
United Nations International Labour Office, 80–0929
United States Tobacco Journal, 80–1306, 80–1847
†University of Delaware, 80–0631
Upton, A. C., 80–0312
Uragoda, C. G., 80–1700
Uren, N. C., 80–0046
Uroshevich, M., 80–0540
Ushimaru, Y., 80–0902

Vahakangas, K., 80–0067
Vaidya, R. C., 80–0124
Vaisrub, S., 80–0128
Vajdics, Z., 80–0246
Vakselj, A., 80–0692
Valadaud-Barrieu, D., 80–1628
Valeriote, C., 80–0429
Valero, E., 80–0974
Van Auken, T. V., 80–0585, 80–0586, 80–0614
van Breda Kolff, K., 80–0886
Van Cantfort, J., 80–0912, 80–1107
Vance, J. W., 80–0268
van de Woestijne, K. P., 80–0097
van den Heuvel, P. D., 80–1345
van der Lende, R., 80–1189
van der Maas, L.L.J., 80–1189
van der Pligt, J., 80–1504
van der Putten, M., 80–1650
Van Doorn, R., 80–1140
Vangen, O., 80–0801
Van Hall, F., 80–0541, 80–0542
Vanini, G. C., 80–1622
Van Rensburg, S. J., 80–0746
Van Zwieten, P. A., 80–1376
Vartiainen, E., 80–1255
Vartsky, D., 80–1105
Vatrenjak, V., 80–1374
Vaughn, S., 80–1792
Veale, A., 80–0720
Veall, N., 80–0770
Veber, O. A., 80–1163
Ved Brat, S. S., 80–1387
Velasco Rami, J. A., 80–0376
Venables, P. H., 80–1633
Vercellotti, E., 80–1622
Vessey, M. P., 80–0377
Vestal, R. E., 80–0913
Vetter, W., 80–1463
Vezzoli, F., 80–0779
Viadana, E., 80–0082
Viart, P., 80–0247
Victorian Cancer News, 80–1090
Vidal, S., 80–0442
Vierke, S., 80–1778
Vinals, J. F., 80–0587, 80–0588, 80–0591, 80–0592, 80–0596, 80–0597, 80–0598, 80–0599, 80–0600, 80–0601, 80–0606, 80–0607, 80–0615, 80–0616, 80–0617, 80–0618, 80–0619, 80–0620, 80–0621, 80–0626, 80–0627, 80–1808, 80–1809, 80–1810
Viola, F. V., 80–1208
Virtamo, J., 80–0367, 80–1220
Vitzthum, O., 80–0565
Vock, M. H., 80–0587, 80–0588, 80–0590, 80–0591, 80–0592, 80–0596, 80–0597, 80–0598, 80–0599, 80–0600, 80–0603, 80–0606, 80–0607, 80–0616, 80–0617, 80–0618, 80–0619, 80–0621, 80–0626, 80–1808, 80–1809, 80–1810
Vogt, T. M., 80–0172
Vohra, M. M., 80–0267
Voirol, M., 80–1399, 80–1409
Voland, M. E., 80–1038
Vollmer, R. T., 80–0948, 80–1692
Vraney, G. A., 80–0780
Vujanic, G., 80–0340
Vuori, H., 80–1245
Vutuc, C., 80–0090, 80–0307, 80–0926, 80–0930, 80–1570
†Vyzkumny Ustav Chemickych Zarizeni, 80–0543

Wagenaas-Zegers, M.A.P., 80–1140
Wagner, I. U., 80–0398
Wagner, M.M.F., 80–0716
Wagoner, J. K., 80–0931
Wahlberg, I., 80–0211, 80–0212, 80–0213, 80–0248, 80–0249, 80–0688, 80–0689, 80–0871, 80–1318, 80–1333
Waite, I. M., 80–0988
Wake, F. R., 80–1583
Wakeham, H. H., 80–0441
Wakeham, H.R.R., 80–0378
Wald, N. J., 80–0755, 80–0983
Walker, E. K., 80–1608
Walker, J. A., 80–1205
Walker, R. J., 80–0728
Walker, V., 80–0993, 80–1746
Wallace, A. G., 80–1466
Wallace, R. B., 80–0379
Walldius, G., 80–0359, 80–1455
Waller, J. A., 80–0161, 80–0955
Waller, R. E., 80–0309
Wallin, I., 80–1333
Walshe, J., 80–0111

---

†Patent assignee.

Walter, E., 80–1138
Walter, S., 80–0781
Walters, D. B., 80–0037
Walters, W.A.W., 80–0987
Wandscheer, D., 80–1349
Wannamaker, J., 80–1731
Warburton, D. M., 80–1532, 80–1793
Ward, D. E., 80–1438
Wardrop, C.A.J., 80–0720
Warfield, A. H., 80–0622
Warr, G. A., 80–0745, 80–1430
Warshaw, R., 80–0954
Washizaki, M., 80–1699
Watanabe, M., 80–0659
Waters, D. D., 80–0348
Watson, D. S., 80–0380
Watson, F. M., III, 80–0669
Watson, H. R., 80–0623, 80–0624
Wattenberg, L. W., 80–0068
Wax, M. L., 80–1540
Wax, R. H., 80–1540
Weaver, A., 80–0091
Webber, L. S., 80–0870
Weber, A., 80–0809, 80–1388, 80–1748
Weber, A. L., 80–1698
Weber, E., 80–1138, 80–1206
Weber, K. B., 80–1482
Weber, K. H., 80–0001
Webster, I., 80–1175
Webster, J. S., 80–1762
Webster, P., 80–0335
Webster, R. M., 80–1584
Webster, W. S., 80–0069
Wechsler, H., 80–0158, 80–1008
Weeks, D. J., 80–1009
Weeks, W. W., 80–0701
Wehler, R., 80–0830
Wehner, A. P., 80–1141
Wehner, F. C., 80–0746
Wei, E. T., 80–0715
Weill, H., 80–0771
Weinberg, G. B., 80–1209
Weiner, A., 80–0832
Weisburger, J. H., 80–0319
Weiser, P., 80–0188
Weiser, P. C., 80–0070
Weiss, J. F., 80–0934
Weiss, N. S., 80–1231
Weiss, W., 80–0320, 80–0321, 80–1244
Weisz, D., 80–1443
Weker, R. R., 80–1626
Welborn, T. A., 80–1219
Welsch, M., 80–1412
Welsh, P. W., 80–0262
Wenger, A., 80–0708, 80–1332
Wennen, E., 80–1307
Wennmalm, A., 80–0796
Werner, C., 80–1662
Wesch, H., 80–0362
Wesnes, K., 80–1532, 80–1793
Wessel-Aas, T., 80–0951
West, A. A., 80–0644, 80–0645
West, C., 80–1218
West, D. J., 80–0417
West, K. M., 80–1490
Westerholm, P., 80–0131
Wetherall, C. F., 80–1804
Wetter, C., 80–0286
Wetterqvist, H., 80–0772
Wetzler, H. P., 80–0119
Wheeler, J. P., 80–0234
Whewell, R. T., 80–1758
Whichelow, M. J., 80–0159, 80–1479
Whidby, J. F., 80–0020, 80–0233, 80–0693
Whieldon, D., 80–0173
Whitaker, C. J., 80–0932, 80–0933
White, J. L., 80–0649, 80–0650, 80–0651, 80–0652, 80–0653, 80–0654, 80–0655, 80–0656, 80–0657
White, J. R., 80–1190
White, R., 80–0053
Whitehead, R., 80–1356
Whitman, P. K., 80–0287
WHO Chronicle, 80–1308
Wibowo, A.A.E., 80–0749
Widmer, L. K., 80–1196
Wied, G. L., 80–0281
Wiersma, G. B., 80–0193
Wierzba, M., 80–1544
Wiklund, O., 80–1434, 80–1440
Wilbur, R. L., 80–1649
Wilcox, M., 80–0480
Wilcox, R. G., 80–0288
Wildfeuer, W., 80–1091
Wildman, S. G., 80–1607
Wiley, J. A., 80–1309
Wilhelmsson, C., 80–1440
Wilkinson, E. J., 80–1389
†Wilkinson Sword Ltd., 80–0623, 80–0624
Wilkinson, W. E., 80–1710
Williams, C. L., 80–0430, 80–0811, 80–1533
Williams, D. G., 80–1534
Williams, D. J., 80–0061
Williams, D. L., 80–0514
Williams, D.R.R., 80–0294
Williams, J. F., 80–0691
Williams, K. A., 80–0073
Williams, M. C., 80–0101
Williams, R., 80–1310
Williams, R. B., Jr., 80–1705
Williams, R. R., 80–0119, 80–1221
Williams, R. S., 80–1466
Williams, T. B., 80–0545
Williamson, R. E., 80–0709
Willis, B. J., 80–0625
Willis, K. A., 80–0830
Wills, D., 80–0160
Wilson, A. F., 80–1173
Wilson, A. J., 80–1663
Wilson, C. G., 80–1124
Wilson, G. T., 80–1794
Wilson, J. R., 80–1334
Wilson, M. R., 80–0899, 80–1636
Wilson, P. A., 80–0510, 80–0529
Wilson, R. A., 80–1809
Wilson, R. W., 80–1247, 80–1786
Windsor, R. A., 80–1191
Wing, L.M.H., 80–0263
Wing, R. R., 80–0066
Wingard, D., 80–1838
Wingender, M. K., 80–1247
Winsor, G. M., 80–1152
Wise, M. E., 80–1189
Wisoff, B. G., 80–1443
Withycombe, D. A., 80–0620, 80–0626, 80–1810
Wober, M., 80–0146, 80–0147, 80–1761
Woeber, K., 80–1010
Wogan, G. N., 80–0039
Wojtyniak, B., 80–0994
Wolf, G. T., 80–0934
Wolfers, J., 80–0363
Wolfgang, J., 80–0998
Wolfson, M., 80–0098
Wolt, J., 80–0627
Woltz, W. G., 80–0005
Wong, J., 80–1467
Wood, A.J.J., 80–0913
Wood, C., 80–0987
Wood, J. D., 80–1100
Wood, P. D., 80–0066, 80–0120
Wood, W.M.L., 80–0658
Woodham, D. W., 80–1328
Woolcock, A., 80–1431
Woolcock, A. J., 80–1691, 80–1701
Worden, J. K., 80–0161, 80–0955
World Health Organization, 80–0747

†Patent assignee.

World Health Organization, 31st World Health Assembly., 80–0863
World Medicine, 80–1311
World Smoking and Health, 80–1805, 80–1848, 80–1849, 80–1850
World Tobacco, 80–1066, 80–1276
Wray, N. P., 80–0900
Wrba, H., 80–1150
Wright, G., 80–0071
Wright, G. R., 80–0162
Wright, J. P., 80–1744
Wright, S., 80–0163
Wulf, H. C., 80–1119
Wulff, H. R., 80–0806
Wulff, M., 80–0806
Wurtman, R. J., 80–1386
Wynder, E. L., 80–0024, 80–0187, 80–0430, 80–0891, 80–0935, 80–1164, 80–1407
Wysocki, J., 80–1664
Yagi, H., 80–0068
Yamaguchi, M., 80–0050
Yamashiro, S., 80–0901
Yamatake, Y., 80–0072
Yanaura, S., 80–0072, 80–0914
Yang, C. S., 80–1674
Yang, J. A., 80–0289
Yasumatsu, N., 80–1601
Yeager, H., Jr., 80–0101
Yeargin, B., 80–1312
Yergin, B. M., 80–0950
Yernault, J.-C., 80–1432
Yi, J. M., 80–1613
Yildirim, S., 80–0956
Yokomichi, I., 80–0476
Yokota, H., 80–0538, 80–0659
Yoshida, D., 80–0687
Yoshida, K., 80–0748, 80–1665
Yost, F. N., 80–0826
Young, J. C., 80–1603
Young, J. R., 80–0118
Younoszai, M. K., 80–0137
Yu, P. K., 80–0481

Zaborski, W., 80–1702
Zachariah, P. K., 80–1377
Zahiu, L., 80–1753
Zamel, N., 80–0328, 80–1432
Zanzi, I., 80–1105
Zbikowska, M., 80–0976
Zborowski, J., 80–1811
Zehnder, R., 80–1494
Ziegler, H. W., 80–1196
Zielhuis, R. L., 80–0749
Ziemecka, E., 80–1683
Zilkey, B., 80–0251
Zilkey, B. F., 80–0866, 80–1608
Zimmermann, H., 80–0957
Zizka, J., 80–0543
Zollinger, H. U., 80–1400
Zuspan, F. P., 80–0750

CUMULATIVE SUBJECT INDEX

ABNORMALITIES
effect of maternal smoking and occupational exposure on incidence, 80–0132
effect of maternal smoking on cleft lip and cleft palate incidence in infants, 80–0131
maternal smoking and, 80–0984

ABORTION
effect of maternal smoking and occupational exposure on incidence, 80–0132
effect of maternal smoking on incidence, 80–0133, 80–0799
induced, smoking habit and, 80–1736

ABSENTEEISM
in smoking vs. nonsmoking males vs. females, 80–1391

ACADEMIC ACHIEVEMENT
in smoking vs. nonsmoking adolescents in East Germany, 80–1515

ACCIDENTS
alcohol consumption and smoking-related fire mortality, 80–1838
1978 California Fire Incident Reporting System Annual Report, 80–0849
cigarettes as cause of fires, 80–1842
flammability standard for upholstered furniture, 80–0858
probabilistic model for smoking as cause of fires in upholstered furniture, 80–0451
smoking as cause of fires in upholstered furniture, 80–0858
smoking materials as cause of fires, 80–0470
smoking-related fires in California, 80–0453

ACETALDEHYDE
(*See also* **ALDEHYDES**)
in cigarette smoke condensate, interaction with acetone during chemical analysis, 80–0020
effect of adrenergic blocking agents, ascorbic acid, and cysteine on toxicity in rats, 80–0909
effect of derivatives on tobacco aroma and flavor, 80–0607
effect on respiratory system in hamsters, 80–0721
metabolism in lung cells, 80–0904

ACETALDEHYDE CONTENT
in cigarette smoke, effect of Cytrel substitute, 80–1544
in cigarette smoke, effect of paper perforation, 80–1264
in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
in tobacco, effect of 4-(methylthio)butane, 80–0608

ACROLEIN
(*See also* **ALDEHYDES**)
effect of adrenergic blocking agents, ascorbic acid, and cysteine on toxicity in rats, 80–0909
effect of smoke inhalation on lungs in rats, 80–0730

ACROLEIN CONTENT
in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
in cigarette smoke of Canadian brands, 80–1603

Adenoma *See* **NEOPLASMS**

ADOLESCENTS
attitudes in Great Britain, 80–1504
blood lipids and lipoproteins in smokers vs. nonsmokers, 80–0364
cotinine levels in blood as verification of self-reported smoking, 80–0430
effect of antismoking campaigns on smoking habit in France, 80–0405
effect of familial smoking habit on asthma incidence in New Zealand, 80–0778
effect of health education on smoking habit in Switzerland, 80–1787
effect of life skills training in prevention of smoking, 80–0811, 80–1533
effect of parental and peer smoking on smoking habit, 80–1518
effect of parental smoking and school regulations on smoking habit, 80–1412
effect of parental smoking on smoking habit in France, 80–0405
effect of parental smoking on smoking habit in Great Britain, 80–1527
effect of parental smoking on smoking habit in South Africa, 80–0141
effect of personality scores on subsequent smoking habit, 80–1498
effect of School Health Curriculum Project on smoking habit and attitudes, 80–1510
effect of significant others on smoking habit, 80–1243
effect of smoking and respiratory tract diseases on lung function, 80–1701

effect of smoking control programs on smoking habit in the United States, 80–1234
effect of smoking on lung function and respiratory symptoms, 80–0141
personality and sex behavior in smokers vs. nonsmokers, 80–1518
personality in smokers vs. nonsmokers, 80–1774
psychosocial variables in prediction of smoking habit, 80–1492
risk factors for cardiovascular diseases in Switzerland, 80–1193
smoking control programs in schools in East Germany, 80–1507
smoking habit, alcohol consumption, and drug abuse in Puerto Rico, 80–0427
smoking habit and alcohol consumption in Switzerland, 80–1244
smoking habit and drug abuse in the United States, 80–0414
smoking habit and personality in males vs. females, 80–1514
smoking habit in East Germany, 80–1515
smoking habit in Ethiopia, 80–0995
smoking habit in Finland, 80–0426
smoking habit in France, 80–0424, 80–1412
smoking habit in Great Britain, 80–1693
smoking habit in schools in Ohio, 80–1770
smoking habit in the United States, 80–0448, 80–0468

ADRENAL GLANDS
effect of nicotine on tyrosine hydroxylase activity in rats, 80–1386
effect of opiate agonists on nicotinic receptors in bovine chromaffin cells, 80–0896

Adrenaline *See* **CATECHOLAMINES**

ADVERTISING
antismoking campaigns and, 80–1562
antismoking campaigns in Europe and, 80–0857
antismoking campaigns in Great Britain and, 80–1572
in developing countries, 80–1307
effect of health warning on desire to smoke in smokers vs. nonsmokers, 80–0822
effect of restrictions on marketing techniques in the tobacco industry, 80–1066
effect on cigarette consumption in Great Britain, 80–1300
effect on smoking habit, 80–0147
effect on smoking habit in Canada, 80–1826
effect on tobacco substitute sales in Great Britain, 80–0177
recommendations of Department of Health and Social Security in Great Britain, 80–1301
recommendations of Federal Trade Commission for rotating health warnings, 80–1306
recommended ban for tobacco products in Europe, 80–1030
recommended ban in Australia, 80–1849
role of physicians in opposition to snuff dipping and tobacco chewing, 80–1045
smoking and, 80–1058, 80–1560
smoking habit in print media readers in Australia and, 80–1831
tobacco industry sponsorship of sports events on television in Great Britain, 80–1311
U.S. Federal Trade Commission report, 80–0448

AGING
effect of cigarette consumption levels on mortality rates in men, 80–0754
effect of maternal nicotine administration on body weight in offspring in rats, 80–1369
effect of maternal smoking on abruptio placentae and placenta praevia incidence, 80–1474
effect of maternal smoking on fetal and neonatal mortality rates, 80–1474
effect of smoking and workload on heart rate, 80–1716
effect of smoking on ischemic heart disease mortality risk, 80–1464
effect on blood and plasma viscosity in smokers vs. nonsmokers, 80–0788
effect on bronchitis incidence in smokers vs. nonsmokers, 80–1684
effect on cadmium and zinc levels in urine, 80–0261
effect on cardiovascular disease risk in oral contraceptive users, 80–1719
effect on diazepam metabolism, 80–1108
effect on echocardiographic examination, 80–1433
effect on isoproterenol and propranolol responsiveness in smokers vs. nonsmokers, 80–0913
effect on lung function in smoking vs. nonsmoking fertilizer plant workers, 80–0332
effect on smoking characteristics, 80–1520
effect on theophylline metabolism in smokers vs. nonsmokers, 80–1617
effect on vision in smokers vs. nonsmokers, 80–1483
mineral content in bone in smokers vs. nonsmokers and, 80–0805
role in effects of amphetamine, cocaine, nicotine, and strychnine on seizure susceptibility in mice, 80–0048

AGRICULTURAL PRACTICES
effect of acephate powder vs. emulsion on residue levels in tobacco, 80–0203
effect of aging and topping on sterol content of tobacco leaf, 80–0243
effect of benzophenones and benzhydrols on sucker growth, 80–0478
effect of benzylthiolcarbamate derivatives on sucker growth, 80–0476

effect of calcium fertilizer on tobacco leaf components, 80–0008
effect of calcium fertilizer on tobacco yield, 80–0008
effect of carbetamide on sucker growth, 80–0473
effect of chlorine content of irrigation water on physical and chemical characteristics of tobacco, 80–0195
effect of crop rotation on cigar wrapper tobacco quality and yield, 80–0685
effect of cultural practices on polyphenol content in chlorophyll-deficient varieties, 80–1071
effect of 2, 6-dinitroaniline derivatives on sucker growth, 80–0475
effect of diphenyl sulfides and diphenyl ethers on sucker growth, 80–0481
effect of environmental, physiological, and biochemical factors on nicotine content in tobacco, 80–0192
effect of ethephon, nitrogen rate, transplanting date, and variety on alkaloid and sugar content in tobacco, 80–1586
effect of ethephon, nitrogen rate, transplanting date, and variety on tobacco quality and yield, 80–1586
effect of ethephon on tobacco quality and ripening, 80–1585
effect of fatty acid alcohols and fatty esters on sucker growth, 80–1070
effect of fatty alcohols and 1, 2-dihydro-pyridazine-3, 6-dione salts on sucker growth, 80–0477
effect of fertilization and fumigation on bidi tobacco yield and quality, 80–0006
effect of fertilization and leaf population on tobacco quality and yield, 80–0201
effect of fertilization and moisture on tobacco smoke constituents, 80–0208
effect of fertilization and plant density on chemical and physical characteristics of flue-cured tobacco, 80–0681
effect of fertilization and plant density on nicotine content of tobacco leaf and smoke, 80–0198
effect of fertilization and seasonal variation on nutrient uptake, 80–0003
effect of fertilization and seasonal variation on tobacco and paddy yield, 80–0003
effect of fertilization and seasonal variation on tobacco leaf components, 80–0003
effect of fertilization, leaf maturity, and topping on tar content in tobacco smoke, 80–0687
effect of fertilization, light, and moisture on tobacco growth and disease resistance, 80–0208
effect of fertilization on agronomic and chemical characteristics of tobacco varieties, 80–1313
effect of fertilization on boron content and yield in *N. tabacum* L., 80–0199
effect of fertilization on chemical composition and quality of flue-cured tobacco, 80–0205
effect of fertilization on flecking, growth, and chemical composition of flue-cured tobacco., 80–0007
effect of fertilization on nutrient levels and phenol content in tobacco, 80–0197
effect of fertilization on polyphenol content in flue-cured tobacco, 80–0840
effect of fertilization on sterol content in tobacco leaf, 80–0242
effect of fertilization on tobacco leaf components, 80–0190, 80–0202
effect of fertilization on tobacco quality and yield, 80–1073
effect of fertilization on weather fleck in tobacco, 80–1069
effect of fertilization with chlorine on growth and quality of tobacco, 80–0004
effect of fertilization with magnesium on nitrogen and protein content in tobacco, 80–1072
effect of fertilization with magnesium on quality and yield of tobacco, 80–1072
effect of fertilization with nitrogen on quality and yield of chewing tobacco, 80–0686
effect of foliar nutrition on quality and yield of tobacco, 80–0200
effect of foliar nutrition on tobacco leaf components, 80–0200
effect of growth medium and seedling pullings on tobacco constituents and yield, 80–0866
effect of growth regulator on agronomic and physicochemical characteristics of tobacco, 80–1314
effect of herbicide on tobacco quality and yield, 80–0010
effect of insecticides on enzyme activity and pigment levels in tobacco, 80–1068
effect of irrigation on alkaloid and rutin content in tobacco leaf, 80–0684
effect of irrigation on growth and weight of tobacco, 80–0684
effect of low-profile vs. normal harvest on constituent levels in cigarette smoke, 80–0207
effect of maleic hydrazide on sucker growth, 80–0864, 80–0865
effect of maleic hydrazide on tobacco sales in West Germany, 80–1827
effect of maturation stage on tobacco leaf components, 80–0697
effect of *N*-alkyl-*N*-phenylsulfenyl-*N′*-phenyl-formamidine derivatives on sucker growth, 80–0472
effect of *N*-benzyl-2, 6-dinitro-3-amino-4-trifluoromethylanilines on sucker growth, 80–0480
effect of nematicide application on tobacco seed germination, 80–1316
effect of nutrient deficiency on chemical constituents in tobacco, 80–1315
effect of pesticide dosage on residue levels, 80–0191

effect of phosphorothionoamidate derivatives on sucker growth, 80–0479
effect of plant density on tobacco leaf components and smoke constituents, 80–0874
effect of planting time on tobacco quality and yield, 80–1067
effect of potassium content on tobacco quality, 80–0226
effect of priming and topping on alkaloid, nitrogen, and sugar levels, 80–1590
effect of priming and topping on tobacco quality and yield, 80–1590
effect of spacing and topping on constituent levels in tobacco, 80–1588
effect of spacing and topping on tobacco leaf components, 80–1591
effect of sucker control on agronomic and chemical characteristics of Burley tobacco, 80–0206
effect of tobacco ripeness on quality, 80–0009
effect of topping and spacing on growth and yield of flue-cured tobacco, 80–1589
effect of topping and stalk-cutting on nicotine content of tobacco leaf and smoke, 80–0198
effect of topping on nicotine content in Italian variety, 80–1260
effect of wilt-accelerating agents on tobacco curing and drying, 80–0474
effect on butralin residue levels in tobacco, 80–0244
effect on ketone and phenol content in tobacco, 80–1074
effect on nitrate and nitrogen content of tobacco soil, 80–0002
effect on tar content of tobacco smoke, 80–0198
effect on tobacco quality, 80–0035
methomyl as pesticide in tobacco plants, 80–0046
in production of less hazardous cigarettes, 80–1846

AIR POLLUTION
carbon monoxide and health risks in cyclists and, 80–1310
effect of cigarette pyrolysis on chlorofluoromethane decomposition, 80–0702
effect of cigarette smoke on annoyance response in nonsmokers, 80–1789
effect of cigarette smoke on particulate levels in indoor environments, 80–1378
effect of ozone on nitrogen content in tobacco, 80–1069
effect of parental smoking on respiratory tract disease incidence in children, 80–0952
effect on bronchitis incidence in smoking vs. nonsmoking industrial workers, 80–1418
effect on carboxyhemoglobin and thiocyanate levels in smokers vs. nonsmokers in rural vs. urban areas, 80–1623
effect on carboxyhemoglobin levels in smokers vs. nonsmokers, 80–1621
effect on carboxyhemoglobin levels in smoking vs. nonsmoking traffic policemen, 80–1614
effect on incidence of sputum abnormalities in smokers vs. nonsmokers in Yugoslavia, 80–0340
effect on lung function in smokers vs. nonsmokers, 80–0092, 80–0098
effect on lung function in smoking vs. nonsmoking industrial workers, 80–0763
effect on lung neoplasm mortality rates in Pennsylvania, 80–0320
effect on mortality rates in the United States, 80–0291
effect on respiratory symptom incidence in children in France, 80–1420
effect on respiratory symptoms in smokers vs. nonsmokers, 80–0098
smoking and lung neoplasms and, 80–0309
and smoking in respiratory tract disease etiology, 80–0188

ALCOHOL CONSUMPTION
adolescents in Switzerland, 80–1244
disability retirees in Australia, 80–1667
drug abuse in university students in Nigeria and, 80–0153
effect of maternal smoking on fetus, 80–1740
effect of nicotine on mother and offspring in rats, 80–1609
effect of smoking on bronchitis risk, 80–1413
effect on blood lipids and lipoproteins in adolescents, 80–0364
effect on carcinoembryonic antigen levels in smoking vs. nonsmoking liver disease patients, 80–1335
effect on electroencephalogram in smokers vs. nonsmokers vs. deprived smokers, 80–1633
effect on fetal mortality, 80–0133
effect on 17-ketosteroid content in urine, 80–1488
effect on mean red cell volume in smokers vs. nonsmokers, 80–0961
effect on multiple neoplasm risk in mouth neoplasm patients, 80–0315
effect on neoplasm mortality ratio in Japan, 80–0302
effect on nicotine pharmacokinetics in rats, 80–1091
effect on phenytoin metabolism in epileptics, 80–1342
effect on phosphatase activity in blood, 80–1488
effect on prostate and testes function, 80–1488
effect on psychomotor performance, 80–0429
effect on smoking habit, 80–1519
effect on spermatozoa count and mobility, 80–1488
interactive effect with benzo(*a*)pyrene on laryngeal neoplasm induction in hamsters, 80–0910
interactive effect with smoking on laryngeal neoplasm risk, 80–0086
interactive effect with smoking on neoplasm risk, 80–1403
interactive effect with smoking on relative risk for head and neck neoplasms, 80–0923
in laryngeal neoplasm etiology, 80–0091

liver cirrhosis vs. liver neoplasm patients, 80–1399
maternal smoking and fetal alcohol syndrome and, 80–0797
oral and pharyngeal neoplasms and, 80–1678
in oral neoplasm etiology, 80–0091, 80–0317
personality in smoking vs. nonsmoking alcoholics, 80–0830
in pharyngeal neoplasm etiology, 80–0091
as risk factor for tongue neoplasms, 80–0922
smoking and drug metabolism and, 80–1581
smoking and esophageal neoplasms and, 80–1153
smoking and mortality and, 80–1142
smoking and neoplasms and, 80–0924
smoking habit and 80–0155, 80–0827
smoking habit and cardiovascular diseases in physicians and, 80–0828
smoking habit and diet in stomach neoplasm patients and, 80–1163
smoking habit and drug abuse in military recruits in Switzerland and, 80–1494
smoking habit in college students and, 80–1008
smoking habit in male vs. female users of health services, 80–1238
smoking habit in oral and pharyngeal neoplasm patients and, 80–0933
smoking, housing, and respiratory symptoms in Denmark and, 80–1178
smoking, laryngeal neoplasms, and occupational exposure and, 80–0919
smoking-related fire mortality and, 80–1838
in smoking vs. nonsmoking university students in Australia, 80–0401

ALCOHOL CONTENT
determination in Bulgarian oriental tobacco, 80–0225

ALCOHOLS
effect of cinnamic derivatives on tobacco aroma and flavor, 80–0568
effect of 1, 2-dihydro-pyridazine-3, 6-dione salts and fatty alcohols on sucker growth, 80–0477
tricyclic derivatives for tobacco aroma and flavor enhancement, 80–1808

ALDEHYDE CONTENT
in cigarette smoke, effect of hydrogen peroxide-containing filters, 80–1817
in cigarette smoke, effect of polystyrene resins, 80–0841
in cigarette smoke, effect of ventilated filters, 80–1016
in cigarette smoke of Canadian brands, 80–1603

ALDEHYDES
(*See also* **ACETALDEHYDE; ACROLEIN; FORMALDEHYDE)**
effect of carboxaldehydes on tobacco aroma and flavor, 80–0591
effect of curing on content in tobacco leaf, 80–0012
effect of dialdehydes on tobacco stiffness and filling capacity, 80–0519
effect of ion-exchange resin in filters on delivery, 80–0513
effect of 2-phenyl-3-(fur-2-yl)-prop-2-3n-1-al on tobacco flavor, 80–0578
effect of polymer-coated carbon particle filters on levels in cigarette smoke, 80–0495

ALIPHATIC HYDROCARBONS
determination in *Natu* tobacco, 80–0031

ALKALOID CONTENT
(*See also* **NICOTINE CONTENT)**
in cigarette smoke, effect of Cytrel substitute, 80–1544
determination in tobacco by nonaqueous conductimetric titration, 80–0705
determination in tobacco by spectrophotometry, 80–0701
in flue-cured tobacco varieties in Pakistan, 80–0204
in Taiwan vs. United States varieties, 80–0025
in tobacco, effect of agricultural practices, 80–0866, 80–1586
in tobacco, effect of climatic conditions, 80–1592
in tobacco, effect of environmental and genetic factors, 80–0238
in tobacco, effect of fertilization, 80–0197
in tobacco, effect of nutrient deficiency, 80–1315
in tobacco, effect of spacing and topping, 80–1588
in tobacco, effect of sucker control agents, 80–1070
in tobacco, effect on ash formation, 80–0237, 80–0238
in tobacco leaf, effect of agricultural practices, 80–0201, 80–1591
in tobacco leaf, effect of curing, 80–0012
in tobacco leaf, effect of homogenized leaf curing, 80–1261
in tobacco leaf, effect of irrigation, 80–0684
in tobacco varieties, effect of enzyme activity, 80–1381
in tobacco varieties, effect of fertilization, 80–1313

ALKALOID METABOLISM
(*See also* **NICOTINE METABOLISM)**
biosynthesis in tobacco, 80–0867, 80–0871

ALKALOIDS, TOBACCO
(*See also* **ANABASINE; COTININE; NICOTINE; NORNICOTINE)**
biosynthesis of nicotine and nornicotine, 80–0231
determination by pyrolysis-gas chromatography, 80–1329
effect of calcium fertilizer on levels in leaf, 80–0008
effect of priming and topping on levels, 80–1590
identification of N′-formylanabasine and N′-formylanatabine in Burley tobacco, 80–1601
synthesis of related compounds, 80–0221

use of deuterium for analysis of formation and structure, 80–0245

ALLERGY
effect of tobacco extracts in smoking vs. nonsmoking coronary disease patients, 80–0976
smoking in thromboangiitis obliterans etiology, 80–0790
tobacco smoke in atopic dermatitis etiology, 80–1233

ALTITUDE
effect of carbon monoxide on work capacity of nonsmokers, 80–0070

Alveolar cell carcinoma *See* **CARCINOMA, BRONCHOGENIC**

Alveoli *See* **PULMONARY ALVEOLI**

AMIDES
determination in cigarette smoke condensate, 80–0018
effect on tobacco aroma and flavor, 80–0577

AMINE CONTENT
in tobacco smoke, effect of silicic acid—polystyrene filter, 80–1813

AMINES
effect of metal complexes on cyanide content in tobacco smoke, 80–0564
effect of nitric oxide content of cigarette smoke, 80–0032

AMINO ACIDS
effect of aging and curing on content in tobacco, 80–1258
effect of curing on levels in tobacco leaf, 80–1816
effect of ozone on levels in tobacco, 80–1069
effect of reaction product of valine, sugar, and carbonyl compounds on tobacco flavor, 80–0622
in tobacco leaf and smoke, 80–0867
in tobacco leaf, effect of curing, 80–0012

AMMONIA
inorganic compounds as tobacco substitutes, 80–0630

AMMONIA CONTENT
in cigarette smoke, effect of agricultural practices, 80–0207

ANABASINE
(*See also* **ALKALOIDS, TOBACCO**)
identification of *N′*-formylanabasine in Burley tobacco, 80–1601

ANABASINE CONTENT
in tobacco leaf and smoke, effect of tobacco varieties, 80–1086

ANGINA PECTORIS
(*See also* **CORONARY DISEASE**)
effect of carbon monoxide and non-nicotine cigarettes in patients, 80–0782
effect of passive smoking in patients, 80–0358
effect of smoking and other risk factors on recurrence in coronary bypass patients, 80–1443
isosorbide dinitrate in therapy of, 80–1192
smoking habit in young female patients, 80–1724

ANTIBODIES
(*See also* **IMMUNE SYSTEM; IMMUNITY**)
antinicotine antibody production in goats and rabbits, 80–1613
effect of tobacco extracts on IgE production in smokers vs. nonsmokers, 80–0899
effect of tobacco leaf and smoke condensate extracts on production in guinea pigs and rabbits, 80–0262
immunoglobulin levels in bronchoalveolar lavage fluid in smokers vs. nonsmokers, 80–1111
immunoglobulin levels in smoking vs. nonsmoking grain handlers vs. teachers, 80–1348
smoking and IgD and IgG levels, 80–0279

ANTIGEN—ANTIBODY REACTIONS
(*See also* **IMMUNE SYSTEM; IMMUNITY**)
antinicotine antibody production in goats and rabbits, 80–1613
effect of cigarette smoke on secondary response in mice, 80–0270
induced by tobacco extracts in smoking vs. nonsmoking coronary disease patients, 80–0976
tobacco extracts and tobacco smoke in rodents, 80–1636

ANTISMOKING CAMPAIGNS
(*See also* **HEALTH EDUCATION; PREVENTION OF SMOKING**)
Action on Smoking and Health in Great Britain, 80–1571
advertising and, 80–1562
advertising in Great Britian and, 80–1572
attitudes of nurses in New Zealand, 80–0160
attitudes in the United States, 80–1839
attitudes of tobacco industry, 80–1286
in Austria, 80–1836, 80–1837
in China, 80–1850
in coronary disease intervention program, 80–0422
in coronary disease intervention program in Belgium, 80–0969
Department of Health, Education, and Welfare initiatives, 80–0185
Department of Health, Education, and Welfare objectives for 1990, 80–0471
effect of antismoking messages in consonant vs. dissonant smokers, 80–0407

effect of health education in cancer screening program, 80–0301
effect on children in France, 80–1000
effect on legislation in the United States, 80–1312
effect on smoking habit in adolescents in France, 80–0405
effect on smoking habit in France, 80–1235
effect on smoking habit in the United States, 80–1511
effect on tobacco industry in Finland and Sweden, 80–1062
in Egypt, 80–1576
in Europe, 80–0857
evaluation of American Cancer Society Stop Smoking Program, 80–0436
in France, 80–0455, 80–0466, 80–1834
in Great Britain, 80–1050
health education as preventive medicine for neoplasms in adults, 80–1513
health education in East Germany, 80–1529
health education in France, 80–1769
health education in preventive medicine program, 80–0428
health education in schools in Japan, 80–1003
in health education programs for hypertensives, 80–0380
history in the United States, 80–0845
in hospitals, 80–1298
in hospitals in Great Britain, 80–0183
legal restrictions on smoking in public places in the United States, 80–0845
multimedia program for prevention of cardiovascular diseases, 80–0120
in myocardial infarct intervention program, 80–0356
objectives of the Ministry of Social Affairs in Quebec, 80–1296
opposition by the Tobacco Action Network, 80–1042
in Planned Parenthood clinics in the United States, 80–1764
policy in Canada, 80–1296
political obstacles in the United States, 80–0189
in prevention of bronchial and lung neoplasms in Austria, 80–0306
as preventive medicine for hypertension in college students, 80–1197
as preventive medicine for lung neoplasms in Great Britain, 80–0297
recommendations for legislation in Great Britain, 80–1287
recommendations for programs, 80–0187
recommendations in Australia, 80–1849
recommendations in developing countries, 80–1307
recommendations in Europe, 80–1030
recommendations of Federal Trade Commission, 80–1306
recommendations of the Federal Trade Commission, 80–1065
recommendations of the International Union Against Cancer workshop, 80–1580
in restaurants in Switzerland, 80–1845
role of gynecologists and obstetricians, 80–1569
role of health organizations in Finland, 80–1048
role of health professionals, 80–0184
role of pediatricians, 80–1055
role of physicians, 80–0173, 80–1562
in schools for health professionals in the United States, 80–1236
in schools in Sweden, 80–1055
in schools in the U.S.S.R., 80–1242
strategy in Australia, 80–1840
television program in Finland, 80–1255
tobacco industry counterorganization to prevent restriction of smoking legislation, 80–1557
in the U.S.S.R., 80–0959
in the workplace, 80–1833

ANTISMOKING MATERIALS
bibliography for health education in schools, 80–1303
Canadian Cancer Society tabloids, 80–1848
card file in East Germany, 80–1279
self-help book for cessation of smoking, 80–1804

ANTITRYPSIN DEFICIENCY
effect on lung function in smokers vs. nonsmokers, 80–0342
lung diseases and, 80–0944
lung function in smokers vs. nonsmokers and, 80–0095
lung function in smokers vs. nonsmokers vs. ex-smokers and, 80–1417
neutrophil elastase as risk factor for lung function abnormalities in patients, 80–0333
respiratory symptoms and obstructive airway disease in smokers vs. nonsmokers, 80–0765
smoking and lung function in patients, 80–0946
smoking and obstructive airway diseases and, 80–0100, 80–1172
in smoking vs. nonsmoking bronchitics, 80–0105
in smoking vs. nonsmoking emphysema patients, 80–0325

ANTITRYPSIN LEVELS
in blood, effect of smoke inhalation in dogs, 80–0274
in blood, effect of smoking, 80–0266
in lung, effect of smoke inhalation in rats, 80–0266
in pulmonary alveolar macrophages in smokers vs. nonsmokers, 80–0329
in smokers vs. nonsmokers, 80–0327, 80–0934, 80–1364
in smoking vs. nonsmoking vs. ex-smoking dust-exposed workers, 80–1410

ANXIETY
effect of coronary disease risk factor intervention program, 80–1703

effect of passive smoking in nonsmokers, 80–0823

AORTA
(*See also* **ARTERIES; BLOOD VESSELS**)
effect of carbon monoxide, hydrogen cyanide, and nitric oxide in rabbits, 80–1117, 80–1353
effect of smoke inhalation on enzyme activity in rats, 80–1350

ARECA
effect on DNA synthesis in lymphocytes and tumor cells, 80–0289
effect on gastrointestinal system in rats, 80–0902

ARECA CHEWING
(*See also* **TOBACCO CHEWING**)
effect on saliva constituents and flow rate, 80–1133
in oral neoplasm etiology, 80–0317
and tobacco chewing, in leukoplakia etiology, 80–0394
and tobacco chewing, in mouth neoplasm etiology in Pakistan, 80–0394

AROMATIC HYDROCARBON CONTENT
in cigarette smoke of green vs. pale-yellow tobacco varieties, 80–0709
in tobacco pyrolyzate, effect of ozone on lipid fraction, 80–1819
in tobacco smoke, effect of ozonization, 80–1823
in tobacco smoke, effect of palladium, 80–0545
in tobacco smoke, effect of palladium and ammonium salts, 80–0563
in tobacco smoke, effect of plant density, 80–0874
in tobacco smoke, effect of zinc oxide and metal nitrates, 80–0562

AROMATIC HYDROCARBONS
(*See also* **BENZ(*a*)ANTHRACENES; BENZO(*a*)PYRENE; BENZOPYRENES; METHYLCHOLANTHRENE**)
as carcinogens in food, 80–0463
determination by partition column chromatography, 80–0217
determination in cigarette smoke condensate, 80–0228
determination of indoles and carbazoles in cigarette smoke condensate, 80–0710
determination of multi-alkylated polynuclear derivatives in cigarette smoke condensate, 80–0038
effect of nicotine pyrolysis on formation, 80–0708
mutagenicity of soot for *Salmonella typhimurium*, 80–0054
silica particles as cocarcinogens, 80–0057
in skin neoplasm induction in mice, 80–0740

ARRHYTHMIA
(*See also* **HEART DISEASES; HEART FUNCTION**)
electrocardiogram in diagnosis of, 80–1438
smoking as risk factor, 80–0125
in smoking vs. nonsmoking healthy adults, 80–0958
ventricular premature beats and other coronary risk factors in smokers vs. nonsmokers, 80–1712

ARSENIC CONTENT
in tobacco varieties in Yugoslavia, 80–0692

ARTERIAL DISEASES
coronary artery stenosis in smokers vs. nonsmokers with peripheral vascular disease, 80–0118
renal artery stenosis in smoking vs. nonsmoking hypertensives, 80–0360
smoking habit and, 80–1196

ARTERIES
(*See also* **AORTA; BLOOD VESSELS**)
effect of carbon monoxide, hydrogen cyanide, and nitric oxide in rabbits, 80–1117
effect of carbon monoxide, hydrogen cyanide, and nitric oxide on coronary and pulmonary vessels in rabbits, 80–1353
effect of nicotine in brain in dogs, 80–1110
effect of nicotine on noradrenaline release and perfusion pressure in ear in rabbits, 80–0739
effect of smoke inhalation and carbon monoxide in pigs, 80–1647
effect of smoke inhalation, carbon monoxide, and nicotine in pigs, 80–0272
fatty streak incidence in smokers vs. nonsmokers, 80–1457

ARTERIOSCLEROSIS
(*See also* **ARTERIAL DISEASES; CORONARY DISEASE**)
carbon monoxide in etiology of, 80–1458
carbon monoxide in etiology of in pigs, 80–0272
effect of smoking on rate of change, 80–1706
model for smoking in etiology of, 80–1457
nicotine in etiology of, 80–1370, 80–1458
nicotine in etiology of in pigs, 80–0272
risk factors in Israel, 80–0116
smoke inhalation in etiology of, in baboons, 80–1649
smoke inhalation in etiology of, in pigs, 80–0272
smoking and personality and, 80–0970
smoking habit and Type A behavior in coronary disease patients and, 80–1705
smoking in etiology of, 80–1439, 80–1453, 80–1458
tobacco allergy and, 80–0790
trends in cigarette consumption and mortality rate, 80–1450

ARTHRITIS
smoking and lung function in patients, 80–0941

ARYL HYDROCARBON HYDROXYLASE
(*See also* **ENZYMES**)

- activity in liver in animals, 80–1363
- activity in liver in smokers vs. nonsmokers, 80–0712
- activity in smokers vs. nonsmokers vs. lung neoplasm patients, 80–1675
- effect of antioxidants and steroids on activity in rats, 80–1377
- effect of benzo(*a*)pyrene on activity in lungs in rats, 80–1627
- effect of cigarette smoke condensate on activity in liver in rats, 80–1107
- effect of cigarette smoke condensate on activity in lungs in rats, 80–1107
- effect of maternal smoking on activity in placenta, 80–1225
- effect of methylcholanthrene on activity in lungs in rats, 80–0912, 80–1097
- effect of smoke inhalation on activity in kidney and lungs in rats, 80–0912
- effect of smoke inhalation on activity in kidneys in rats, 80–1107
- effect of smoke inhalation on activity in liver in rats, 80–1097
- effect of smoke inhalation on activity in lungs in rats, 80–1097, 80–1122
- effect of tobacco extract on activity in rats, 80–1127
- induced by benzo(*a*)pyrene and methylcholanthrene in liver and lungs in rodents, 80–0052
- induced by benzo(*a*)pyrene, dimethylbenzanthracene, and 3-methylcholanthrene in lung cells in rats, 80–0742

ASBESTOS
- cessation of smoking as preventive medicine for lung neoplasms in workers, 80–1280
- effect of exposure on chest radiograph and lung function in dockyard workers, 80–1695
- effect of exposure on respiratory symptoms in smoking vs. nonsmoking drywall construction workers, 80–0326
- effect of exposure on risk for lung neoplasms in shipyard workers, 80–1208
- effect of smoking on chest radiograph in workers, 80–0937
- effect on laryngeal neoplasm mortality in smoking vs. nonsmoking workers, 80–1668
- effect on laryngeal neoplasm risk, 80–0086
- effect on lung function and respiratory symptom incidence in smoking vs. nonsmoking workers, 80–0776
- effect on lung neoplasm mortality in smoking vs. nonsmoking workers, 80–1668
- effect on mesothelioma and pulmonary fibrosis incidence in smoking vs. nonsmoking vs. ex-smoking dockyard workers, 80–1419
- interactive effect with smoking on lung neoplasm mortality rates, 80–1145, 80–1405
- in lung neoplasm induction in hamsters, 80–1141
- mortality rates in smoking vs. nonsmoking workers, 80–1394
- pleural plaques in smoking vs. nonsmoking exposed patients, 80–0103
- in regenerated tobacco composition, 80–0632
- role of physicians in prevention of lung neoplasms in shipyard workers, 80–0935
- smoking and ferruginous bodies in workers, 80–0943
- smoking and lung neoplasms and, 80–0929
- smoking habit in mesothelioma patients and, 80–1146
- and tobacco in neoplasm etiology, 80–0713

ASBESTOSIS
- (*See also* **OCCUPATIONAL DISEASES)**
- effect of smoking on lung function and respiratory symptoms in patients, 80–0776
- induction in hamsters, 80–1141
- mortality in smoking vs. nonsmoking workers, 80–1145
- mortality in workers in Italy, 80–0292
- mortality in smokers vs. nonsmokers, 80–1394
- smoking and occupational exposure in etiology of, 80–0777
- in smoking vs. nonsmoking dockyard workers in Great Britain, 80–0107
- in smoking vs. nonsmoking workers, 80–1696

ASCORBIC ACID
- (*See also* **VITAMIN C LEVELS)**
- effect of marihuana vs. tobacco smoke on mitotic abnormality incidence in lung cells, 80–1637
- effect on nicotine pharmacology in urogenital system in rats, 80–1340
- effect on nitrogen dioxide and nitrosamine content of tobacco, 80–0557

ASH, CIGARETTE
- effect of alkaloid content in tobacco, 80–0237, 80–0238
- effect of boron compounds and tobacco fines on blooming, 80–0647

ASTHMA
- (*See also* **RESPIRATORY TRACT DISEASES)**
- bronchial threshold to histamine, 80–0102
- effect of history on lung function in smoking vs. nonsmoking children, 80–1431
- effect of nontobacco cigarettes on lung function in patients, 80–1699
- effect of parental smoking on incidence in adolescents in New Zealand, 80–0778
- effect of passive smoking on lung function and respiratory symptoms in patients, 80–1697
- effect of smoke inhalation in patients, 80–0102
- in electronics workers, 80–0096
- histamine metabolism in smoking vs. nonsmoking patients, 80–0772
- lung function in patients, 80–1168

in smokers vs. nonsmokers with α_1-antitrypsin deficiency, 80–0095
smoking in electronics workers and, 80–0096

Atherosclerosis *See* **ARTERIOSCLEROSIS**

ATTITUDES
(*See also* **BEHAVIOR**)
control of smoking in public places in Hawaii, 80–0826
credibility ratings of cessation of smoking methods, 80–0435
effect of antismoking campaigns in France, 80–1235
effect of expectancy in self-control reduction of smoking, 80–0413
effect of fear arousal in smokers vs. nonsmokers, 80–0810
effect of health education and socioeconomic status in children, 80–1786
effect of health warning in cigarette advertisements on desire to smoke in smokers vs. nonsmokers, 80–0822
effect of parental approval on smoking habit in children, 80–1055
effect of parental smoking in children, 80–1752
effect of passive smoking on annoyance response in nonsmokers, 80–1789
effect of School Health Curriculum Project in adolescents in the United States, 80–1510
effect of smoking control programs in adolescents, 80–1234
effect on acupuncture treatment success, 80–1773
effect on recidivism, 80–0817
engineers and physicians in Finland, 80–1245
ex-smokers vs. recidivists, 80–0152
health effects of smoking in smokers vs. nonsmokers, 80–1761
health professionals in the United States, 80–0813
health risk of smoking in males vs. females, 80–1792
medical school faculty in England, 80–1237
medical students in the U.S.S.R., 80–1240
medical students in the United States, 80–0814
passive smoking in the United States, 80–0862
physicians in Australia, 80–1502
Roper Organization report of public attitude toward smoking, 80–0861
self-attribution and reduction of smoking, 80–1499
self-categorization of smoking habit and cognitive dissonance, 80–1791
in smokers accepting vs. rejecting smoking cessation treatment, 80–0145
smokers vs. nonsmokers, 80–0146, 80–0955
smoking and antismoking campaigns in nurses in New Zealand, 80–0160
smoking during social interaction in smokers vs. nonsmokers vs. ex-smokers, 80–0404
smoking and health in Great Britain, 80–1571
smoking and health in the United States, 80–0848
smoking and lung diseases in smokers vs. nonsmokers, 80–0161
smoking as deviant behavior, 80–1839
smoking as risk factor for lung neoplasms in smokers vs. nonsmokers, 80–0926
smoking habit and drug use in adolescents in Great Britain, 80–1504
smoking habit and health practices in the United States, 80–1052
smoking habit and marihuana use in the United States, 80–1523
smoking habit in college students in the United States, 80–1778
smoking habit in the United States, 80–1522
smoking restrictions in the United States, 80–1839
in smoking vs. nonsmoking psychosomatic disorder patients, 80–0406
smoking vs. nonsmoking railroad workers in East Germany, 80–1493
teachers toward cancer education in Great Britain, 80–1756
tobacco industry in the United States, 80–1286
tobacco industry toward smoking and health, 80–1053
university students in Romania, 80–1508

AUTONOMIC NERVOUS SYSTEM
effect of nicotine pharmacology on sympathetic nerve terminals in cats, 80–1361

AVERSIVE THERAPY
in cessation of smoking, 80–0431, 80–0834, 80–1011
credibility ratings of cessation of smoking methods, 80–0435
effect of rapid smoking on cardiovascular system, 80–0786
effect on reduction of smoking, 80–1779
electric shock delivery in cessation of smoking, 80–0666
focused smoking in cessation of smoking, 80–0836, 80–1536
intensive smoking in cessation of smoking, 80–0437
rapid smoking in cessation of smoking, 80–0166, 80–1800
risk—benefit evaluation of rapid smoking, 80–0838

BEHAVIOR
(*See also* **ATTITUDES; MOTIVATION; PERSONALITY**)
cessation of smoking and, 80–1540
cessation of smoking in recidivists vs. ex-smokers and, 80–0169
cognitive efficacy vs. symptom attribution in reduction of smoking, 80–1497
effect of abrupt withdrawal on cessation of smoking, 80–1013

effect of antismoking messages in consonant vs. dissonant smokers, 80–0407
effect of carbon monoxide, 80–0898
effect of carbon monoxide on vigilance performance, 80–0253
effect of cessation of smoking on bruxism, 80–0821
effect of cessation vs. reduction of smoking on withdrawal symptoms, 80–1802
effect of expectancy in self-control reduction of smoking, 80–0413
effect of experimental and social conditions on smoking characteristics, 80–1500
effect of fear appeal on cessation of smoking, 80–0171
effect of fear arousal in cessation of smoking, 80–0438
effect of locus of control on cessation of smoking, 80–1013
effect of maintenance strategies on reduction of smoking, 80–1499
effect of maternal smoking on sucking latency and pressure in neonates, 80–1473
effect of nicotine, 80–0860
effect of nicotine on cognitive decisionmaking, and psychomotor performance, 80–0255
effect of parental smoking in children, 80–1752
effect of passive smoking on aggression in angered vs. nonangered nonsmokers, 80–0415
effect of passive smoking on aggression in nonsmokers, 80–0823
effect of self-management and social support on cessation of smoking, 80–0439
effect of self-monitoring on cessation of smoking, 80–1794
effect of smoking, 80–0860
effect of smoking and alcohol consumption on psychomotor performance, 80–0429
effect of smoking on contingent negative variation, 80–1496
effect of smoking on social interaction in male psychiatric patients, 80–0425
effect of smoking on social interaction in smokers vs. nonsmokers vs. ex-smokers, 80–0404
effect of social interaction on smoking characteristics, 80–0143
effect on coronary disease incidence in smokers vs. nonsmokers, 80–0121
infant, effect of maternal smoking during pregnancy, 80–0136
locus of control scale in smokers vs. nonsmokers vs. ex-smokers, 80–0157
modification in cessation of smoking, 80–0170, 80–0433, 80–0812, 80–0832, 80–0834
modification in maintenance of nonsmoking, 80–0825
orality and smoking habit, 80–0820
personality in high- vs. low-arousal smokers, 80–1771
positive reinforcement in reduction of smoking, 80–0164
psychological effects of cessation of smoking, 80–1783
relaxation in cessation of smoking, 80–1797
role playing by children in antismoking education in Sweden, 80–0419
self-control in cessation of smoking, 80–0431, 80–1248
self-control in smokers vs. ex-smokers, 80–0156
self-regulation in ex-smokers vs. recidivists, 80–0152
self-report as verification of cessation of smoking, 80–1568
self-reported smoking by adolescents, 80–0430
smoking as an addiction, 80–0151, 80–0154, 80–1239, 80–1782
smoking as contagious behavior, 80–1503
smoking as deviance, 80–1839
smoking habit and, 80–0155
smoking habit as addiction vs. arousal modulation, 80–1777
smoking habit as rational choice, 80–0145
social psychology theories in smokers vs. ex-smokers, 80–0148
Type A in coronary disease patients, 80–1705

BEHAVIOR, ANIMAL
effect of body weight and self-regulation on nicotine uptake in rats, 80–0903
effect of carbon monoxide, 80–0898
effect of nicotine and heredity in mice, 80–1654
effect of nicotine on fighting behavior in rats, 80–1135
effect of smoke inhalation and genotype in mice, 80–1334
nicotine as discriminative stimulus in rats, 80–0040

BENZ(*a*)ANTHRACENES
(*See also* **AROMATIC HYDROCARBONS**)
dimethylbenz(*a*)anthracene in tracheal neoplasm induction in rats, 80–0729, 80–1660, 80–1661
effect of dimethylbenz(*a*)anthracene on histopathology in tracheal mucosa in rats, 80–1660, 80–1661
effect of dimethylbenz(*a*)anthracene on tracheal epithelium in rats, 80–1121
effect of dimethylbenzanthracene on aryl hydrocarbon hydroxylase activity in lung cells in rats, 80–0742
effect of silica particles on membrane uptake, 80–0057
in lung neoplasm induction in mice, 80–0886

BENZO(*a*)PYRENE
(*See also* **AROMATIC HYDROCARBONS**)
in aryl hydrocarbon hydroxylase induction in liver and lungs in rodents, 80–0052

in bronchial neoplasm induction in hamsters, 80-1658
carcinogenicity in tracheal epithelium in rats, 80-0741
DNA binding in esophageal cells, 80-0265
DNA binding in human bronchus and colon cultures, 80-0039
DNA binding in lungs in rats, 80-1130
effect of cigarette smoke on metabolism in lungs in rabbits, 80-1367
effect of nicotine on metabolism in lung in rats, 80-0067
effect of particle size on pulmonary clearance in hamsters, 80-1345
effect of particle size on respiratory tract neoplasm induction in hamsters, 80-1345
effect of pulmonary alveolar macrophages on mutagenicity, 80-0050
effect on aryl hydrocarbon and prolyl hydroxylase activity in lungs in rats, 80-1627
effect on aryl hydrocarbon hydroxylase activity in lung cells in rats, 80-0742
effect on metaplasia incidence in bronchi in dogs and hamsters, 80-1658
effect on tracheobronchial epithelium in hamsters, 80-0281
hydroxy derivative binding to macromolecules in embryo cells in hamsters, 80-1096
hydroxy derivative metabolism in embryo cells in hamsters, 80-1096
9-hydroxybenzo(*a*)pyrene binding to DNA in lungs in rats, 80-1130
interactive effect with alcohol on laryngeal neoplasm induction in hamsters, 80-0910
interactive effect with influenza virus in lung neoplasm etiology, 80-0285
in lung and tracheal neoplasm induction in rats, 80-1120
in lung neoplasm induction in hamsters, 80-1360
in lung neoplasm induction in mice, 80-0886
metabolism in esophageal cells, 80-0265
metabolism in lung cell cultures, 80-0060
metabolism in lung microsomal fractions, 80-1383
metabolism in lung tissue of lung neoplasm patients, 80-0884
metabolism in lungs in rabbits, 80-0051, 80-0881, 80-0908
metabolism in lungs in rats, 80-0743
metabolism in lymphocytes and monocytes, 80-1371
metabolism in lymphocytes and pulmonary alveolar macrophages, 80-1095
metabolism in myometrium, ovary, and placenta, 80-1139
metabolism in placenta in smokers vs. nonsmokers, 80-1128
metabolism in pulmonary alveolar macrophages, 80-0900
metabolite binding to DNA and RNA in liver and lungs in rodents, 80-0727
mutagenesis in lungs in rats, 80-1130
mutagenesis with soot in *Salmonella typhimurium*, 80-0054
mutagenicity in hamster cells, 80-0743
release rate from polymer-coated and uncoated beeswax in fetal calf serum, 80-0287
in respiratory tract neoplasm induction in hamsters, 80-0721, 80-1659

BENZO(*a*)PYRENE CONTENT
in cigarette smoke, effect of laminated tobacco foil, 80-0484
in cigarette smoke, effect of protein granule filters, 80-0523

Benzo(*a*)pyrene hydroxylase *See* **ARYL HYDROCARBON HYDROXYLASE**

BENZOPYRENES
(*See also* **AROMATIC HYDROCARBONS**)
effect of benzo(*a*)pyrene 4, 5-oxide on glutathione *S*-transferase activity in rats, 80-0912
effect of butylated hydroxyanisole on lung neoplasms induced in mice, 80-0068

Betel nut chewing *See* **ARECA CHEWING**

Bidi smoking *See* **SMOKING, BIDI**

BILLS
(*See also* **LEGISLATION**)
cigarette bootlegging in the United States, 80-1558, 80-1559
Clean Indoor Air Bill in Wisconsin, 80-0846
effect of antismoking campaigns in the United States, 80-1312

BIOASSAY
cigarette smoke condensate toxicity in brown fat cells in hamsters, 80-0735

BIRTH WEIGHT
(*See also* **FETAL GROWTH; NEONATE; PREMATURITY**)
effect of demographic, health, and socioeconomic factors, 80-1477
effect of maternal smoking, 80-0134, 80-0382, 80-0387, 80-0801, 80-1225, 80-1736
effect of maternal smoking and body weight, 80-0130
effect of maternal smoking and diet, 80-1478
effect of maternal smoking and other factors in blacks vs. whites, 80-0798
effect of maternal smoking and socioeconomic status, 80-1738

effect of nicotine in rats, 80–0733
effect of parental smoking, 80–1224
effect of paternal smoking, 80–1224
effect of smoke inhalation in rats, 80–0799

BLADDER NEOPLASMS
effect of artificial sweeteners on risk in smokers vs. nonsmokers, 80–1398
effect of cigar vs. cigarette vs. pipe smoking on risk, 80–1152
effect of smoking and artificial sweeteners on risk, 80–1158, 80–1161, 80–1164
effect of smoking characteristics on risk, 80–1152
effect of smoking habit on risk in males vs. females, 80–1152
effect of smoking on incidence in phenacetin abusers, 80–1400
effect of smoking on relative risk, 80–0762
effect of tobacco chewing on risk, 80–1152
effect of vitamin A on relative risk, 80–0088
smoking and diet and, 80–0088
smoking habit in patients, 80–0307

BLOOD
(*See also* **ERYTHROCYTES; HEMATOCRIT; LEUKOCYTES)**
effect of age on viscosity in smokers vs. nonsmokers, 80–0788
effect of cessation of smoking, 80–1441
effect of coronary disease intervention program on sedimentation rate, 80–1446
effect of maternal smoking on immune system in umbilical cord, 80–0800
effect of smoke inhalation, carbon monoxide, and nicotine on viscosity in pigs, 80–0272
effect of smoking on 2, 3-diphosphoglycerate levels, 80–0726
effect of smoking on parameters, 80–1656
viscosity in smokers vs. nonsmokers, 80–0971

BLOOD CHEMICAL ANALYSIS
cadmium levels in smokers vs. nonsmokers, 80–1105, 80–1373, 80–1634
determination of carbon monoxide content by gas chromatography vs. Oximeter, 80–0014
determination of cotinine and nicotine levels, 80–0219
determination of manganese levels in smokers vs. nonsmokers, 80–0278
determination of thiocyanate levels in smokers vs. nonsmokers, 80–0870
effect of smoking characteristics on thiocyanate levels, 80–1098
effect of smoking on cadmium, lead, and mercury levels in maternal and cord blood, 80–0386
nicotine content in post-mortem samples, 80–0061
thiocyanate levels in smoking vs. nonsmoking mothers, 80–0385

BLOOD CHOLESTEROL
(*See also* **BLOOD LIPIDS; HYPERCHOLESTEREMIA)**
apolipoprotein A-I levels in smoking vs. nonsmoking myocardial infarct patients, 80–1440
coronary disease risk factors and stress in smokers vs. nonsmokers, 80–1715
effect of coronary disease intervention program, 80–1446
effect of dietary fats on levels in smokers vs. nonsmokers, 80–0370
effect of exercise on high density lipoprotein levels in coronary disease patients, 80–0375
effect of smoke inhalation in baboons, 80–1649
effect of smoking on levels, 80–0793, 80–1218
effect of weight loss on high density lipoprotein levels, 80–0066
lipoprotein levels in smoking vs. nonsmoking myocardial infarct patients and relatives, 80–0371
smokers vs. nonsmokers, 80–1455
in smoking vs. nonsmoking adolescents, 80–0364
in smoking vs. nonsmoking Maoris in New Zealand, 80–0345

BLOOD CIRCULATION
(*See also* **HEART FUNCTION; HEMODYNAMICS)**
effect of high- vs. low-nicotine cigarettes, 80–1206
effect of nicotine, 80–0064
effect of nicotine in uterus in monkeys, 80–1384
effect of nicotine in uterus in pregnant rats, 80–1338
effect of smoking and beta adrenoceptor blocking agents in forearm, 80–1465
effect of smoking on reactive hyperemia in forearms, 80–0796

BLOOD COAGULATION
(*See also* **FIBRINOLYSIS)**
effect of smoking and pindolol, 80–1720

BLOOD LIPIDS
(*See also* **BLOOD CHOLESTEROL; HYPERLIPIDEMIA)**
effect of alcohol consumption on levels in Maoris in New Zealand, 80–0345
effect of exercise in coronary disease patients, 80–0375
effect of high- vs. low-nicotine cigarettes, 80–1505
as ischemic heart disease risk factor in males in Scotland and Sweden, 80–0359
lipoprotein levels in smoking vs. nonsmoking myocardial infarct patients and relatives, 80–0371
smokers vs. nonsmokers, 80–1455
smoking and oral contraceptives and, 80–0379
in smoking vs. nonsmoking adolescents, 80–0364

in smoking vs. nonsmoking Maoris in New Zealand, 80–0345

BLOOD PLASMA
effect of age on viscosity in smokers vs. nonsmokers, 80–0788
effect of high- vs. low-nicotine cigarettes on nicotine content, 80–1206
effect of high- vs. low-nicotine cigarettes on vasopressin levels, 80–1653

BLOOD PLATELETS
effect of antidepressants on serotonin uptake in smokers vs. nonsmokers, 80–1366
effect of dietary fats on function in smokers vs. nonsmokers, 80–0370
effect of smoke inhalation, carbon monoxide, and nicotine on aggregation in pigs, 80–0272
effect of smoke inhalation in baboons, 80–1649
effect of smoking and pindolol on adhesiveness, 80–1720
effect of smoking on aggregation, 80–0350

BLOOD PRESSURE
(*See also* **HYPERTENSION**)
blood glucose levels in smokers vs. nonsmokers and, 80–1445
coronary disease risk factors and stress in smokers vs. nonsmokers, 80–1715
effect of carbon monoxide and non-nicotine cigarettes in angina patients, 80–0782
effect of cessation of smoking and exercise, 80–1708
effect of coronary disease intervention program, 80–1446
effect of dietary fats in smokers vs. nonsmokers, 80–0370
effect of nicotine, 80–0064
effect of nicotine content in cigarettes, 80–1138, 80–1206
effect of nicotine in cats, 80–1376
effect of nicotine in dogs, 80–1351
effect of nicotine in pregnant rats, 80–1338
effect of nicotine in uterus in monkeys, 80–1384
effect of rapid smoking, 80–0786
effect of smoking, 80–1444
effect of smoking and beta adrenoceptor blocking agents, 80–1465
effect of smoking in heavy- vs. light- vs. ex-smokers, 80–0785
effect of smoking in myocardial infarct patients, 80–0965
effect of smoking in smokers vs. nonsmokers, 80–0784
smokers vs. nonsmokers, 80–0784
smoking habit and heart disease in hypertensives, 80–0374

BLOOD PROTEINS
effect of age on fibrinogen levels in smokers vs. nonsmokers, 80–0788
effect of smoking on levels, 80–1664
α_2-globulin levels in smokers vs. nonsmokers, 80–1364
glycoprotein levels in smokers vs. head and neck neoplasm patients, 80–0934
glycoprotein levels in smokers vs. nonsmokers, 80–0934
high density lipoprotein apoprotein levels in smokers vs. nonsmokers, 80–0254
immunoglobulin levels in smokers vs. nonsmokers, 80–1631

BLOOD SUGAR
(*See also* **DIABETES**)
arterial diseases and electrocardiogram abnormalities and, 80–1196
asymptomatic hyperglycemia and coronary heart disease, 80–1730
effect of nicotine and carbon monoxide in rats, 80–0879
effect of smoke inhalation in baboons, 80–1649
effect of smoking and pindolol, 80–1720
effect of smoking on coronary disease mortality rates, 80–1200
effect of smoking on levels, 80–0975, 80–1629
effect on electrocardiogram abnormalities in smokers vs. nonsmokers vs. ex-smokers, 80–1445
hyperglycemia and coronary disease in smokers vs. nonsmokers vs. ex-smokers in Japan, 80–1216
hyperglycemia as risk factor for coronary disease, 80–1461
L-fucose levels in mammary neoplasm diagnosis in smokers vs. nonsmokers, 80–1389
smoking and coronary disease and, 80–1204, 80–1713, 80–1729, 80–1731
smoking and coronary disease and hyperglycemia, 80–1219
smoking and hyperglycemia as risk factors for coronary disease, 80–1460
smoking, coronary disease, and post-load levels, 80–1213

BLOOD VESSELS
(*See also* **AORTA; ARTERIES**)
effect of maternal smoking in placenta and umbilical cord, 80–1468
effect of maternal smoking on umbilical artery and vein, 80–1732

BODY HEIGHT
effect of maternal smoking on neonate, 80–1225

BODY TEMPERATURE
effect of cigarette length in fingertips, 80–1751

BODY WEIGHT
(*See also* **OBESITY**)
- blood glucose levels in smokers vs. nonsmokers and, 80–1445
- effect of cessation of smoking, 80–1446
- effect of cessation of smoking in women, 80–1485, 80–1722
- effect of coronary disease intervention program, 80–1446
- effect of ischemic heart disease risk factor reduction program, 80–1229
- effect of maternal nicotine administration and aging in offspring in rats, 80–1369
- effect of maternal nicotine and alcohol consumption in offspring in rats, 80–1609
- effect of maternal smoking and weight gain on placenta disorder incidence, 80–1475
- effect of maternal smoking on prepregnancy weight, 80–1481
- effect of nicotine and alcohol consumption in pregnant rats, 80–1609
- effect of nicotine in rats, 80–0276, 80–1662
- effect of smoke inhalation in baboons, 80–1649
- effect of smoke inhalation in hamsters, 80–1141
- effect of smoking in women, 80–1485
- effect of weight loss on smoking habit, 80–0991
- effect on coronary disease incidence and risk factors in women, 80–1722
- effect on self-regulation of nicotine uptake in rats, 80–0903
- maternal, effect on birthweight and growth of infants, 80–0130
- maternal smoking and prematurity and, 80–0381
- smoking and physical fitness and, 80–1567

Bradycardia *See* **ARRYTHMIA**

BRAIN
- effect of cotinine and nornicotine on nicotine binding in synaptosomes in rats, 80–1665
- effect of nicotine and cotinine on 5-hydroxytryptamine metabolism in rats, 80–0887
- effect of nicotine and pilocarpine on catecholamines in brain in rats, 80–1102
- effect of nicotine content in cigarettes on cerebral cortex electrical activity, 80–0255
- effect of nicotine on arteries in dogs, 80–1110
- effect of nicotine on cyclic guanosine 3′, 5′-monophosphate levels in rats, 80–1101
- effect of nicotine on noradrenaline release from hypothalamic synaptosomes, 80–0748
- effect of nicotine on protein metabolism in adult vs. newborn rodents, 80–0063
- effect of nicotine on tuberoinfundibular dopamine neurons in rats, 80–1365
- effect of nicotine pharmacology on receptors in synaptosomes in rats, 80–1665
- effect of pH on nicotine uptake in rats, 80–1645
- effect of smoking and alcohol consumption on phenytoin metabolism in epileptics, 80–1342
- nicotine distribution in cats, 80–1376

Breast cancer *See* **MAMMARY NEOPLASMS**

BREAST FEEDING
(*See also* **LACTATION**)
- effect of maternal smoking, 80–1479
- in smoking vs. nonsmoking mothers, 80–0159

BRONCHI
(*See also* **RESPIRATORY SYSTEM**)
- DNA binding of benzo(*a*)pyrene, 80–0039
- dysplasia, hyperplasia, and metaplasia in smokers vs. nonsmokers, 80–1159
- effect of benzo(*a*)pyrene on metaplasia incidence in dogs and hamsters, 80–1658
- effect of nicotine on contractility, 80–0072
- enzyme and protein content in bronchoalveolar lavage fluid in smokers vs. nonsmokers, 80–1111
- mucus gland hyperplasia in smoking vs. nonsmoking cotton textile workers, 80–1692

BRONCHIAL EPITHELIUM
(*See also* **TRACHEOBRONCHIAL EPITHELIUM**)
- effect of smoking and plutonium irradiation, 80–0311
- lead-210 and polonium-210 levels in smokers vs. nonsmokers, 80–0737

BRONCHIAL NEOPLASMS
(*See also* **CARCINOMA, BRONCHOGENIC; LUNG NEOPLASMS**)
- antismoking campaigns as preventive medicine in Austria, 80–0306
- histological type in smokers vs. nonsmokers in Hong Kong, 80–1670
- induced by benzo(*a*)pyrene in hamsters, 80–1658
- smoking habit in patients, 80–0296, 80–1676

BRONCHIOLES
- effect of nicotine on contractility, 80–0072

BRONCHITIS
(*See also* **CHRONIC BRONCHOPULMONARY DISEASE; OBSTRUCTIVE AIRWAY DISEASES**)
- alpha$_1$-antitrypsin deficiency in smoking vs. nonsmoking patients, 80–0105
- bacterial adherence to pharyngeal cells in smokers vs. nonsmokers vs. bronchitics, 80–1685
- effect of adrenergic agonists on tracheal mucosa in patients, 80–0950
- effect of aging on incidence in smokers vs. nonsmokers, 80–1684
- effect of air pollution on incidence in smoking vs. nonsmoking industrial workers, 80–1418

effect of cessation of smoking and exercise on physical fitness in patients, 80–1427
effect of history on lung function in smoking vs. nonsmoking children, 80–1431
effect of smoking and alcohol consumption on risk, 80–1413
effect of smoking and dust exposure on incidence in industrial workers, 80–0341
effect of smoking and social factors on incidence in children, 80–1691
effect of smoking and sulfur dioxide on incidence in copper smelter workers, 80–1686
effect of smoking on incidence in welders, 80–1681
lung function as predictor of susceptibility in smokers, 80–0099
lung function in grain workers and, 80–1682
lung function in patients, 80–0335
lung function in smoking vs. nonsmoking vs. ex-smoking bronchitics, 80–1422
relative risk in smokers vs. nonsmokers, 80–1184
in smokers vs. nonsmokers in Czechoslovakia, 80–0106
in smokers vs. nonsmokers vs. ex-smokers in a fertilizer plant, 80–0332
in smokers vs. nonsmokers with α_1-antitrypsin deficiency, 80–0095
smoking and, 80–1156
smoking habit and lung function and, 80–0338
smoking habit in patients, 80–1155
in smoking vs. nonsmoking asphalt workers, 80–1187
in smoking vs. nonsmoking drywall construction workers, 80–0326
in smoking vs. nonsmoking dust-exposed workers, 80–0108
in smoking vs. nonsmoking tea blenders, 80–1700

Buccal cancer *See* **MOUTH NEOPLASMS**

Buergers disease *See* **THROMBOANGIITIS OBLITERANS**

Bysinosis *See* **OCCUPATIONAL DISEASES; PNEUMOCONIOSIS**

CADMIUM
(*See also* **METALS**)
effect on fetal growth in mice, 80–0069
effect on kidney function in cadmium workers, 80–0334
effect on lung function in smoking vs. nonsmoking cadmium workers, 80–0334
kinetic model of metabolism, 80–1362

CADMIUM LEVELS
in blood and feces of smoking vs. nonsmoking battery factory workers, 80–0250
in blood and placenta in smokers vs. nonsmokers, 80–0386
in blood and urine in smoking vs. nonsmoking jewelry factory workers, 80–0880
in blood, effect of smoking, 80–0749
in blood, feces, tissues, and urine in smokers vs. nonsmokers, 80–0269
in blood in smokers vs. nonsmokers, 80–1362
in blood in smoking vs. nonsmoking coronary disease patients, 80–0362
in blood in smoking vs. nonsmoking myocardial infarct patients, 80–0366
in blood, tissue, and urine in smokers vs. nonsmokers, 80–1634
in cigarettes and diet, 80–1083
in cigarettes in New Zealand brands, 80–1595
determination in particulate matter and gas phase cigarette smoke, 80–0034
in feces in smokers vs. nonsmokers, 80–0895
in German cigarettes, 80–0229
in smokers vs. nonsmokers, 80–1105, 80–1373
in tobacco varieties in Yugoslavia, 80–0692
in urine, effect of smoking and age, 80–0261

Cancer *See* **NEOPLASMS**

CANNABIS
(*See also* **DRUG ABUSE**)
effect of exercise on lung function in users, 80–1641
effect of smoke and vitamin C on mitotic abnormality incidence in lung cells, 80–1637
effect of smoke inhalation in lungs in rats, 80–1651
effect of smoke inhalation on pulmonary defense system in rats, 80–1626
effect of smoking on learning, 80–1768
effect on bacteria in lungs in rats, 80–1116
effect on pulmonary alveolar macrophages in rats, 80–1343
effect on smoking habit, 80–1519
effect on theophylline metabolism, 80–0268
effect on theophylline metabolism in smokers vs. nonsmokers, 80–0055
health effects, 80–0456
history of origin and usage, 80–1835
mutagenicity of smoke condensate in *Salmonella typhimurium*, 80–0715, 80–0746
NIDA report on health effects and research, 80–1841
recommendations for legislation in Australia, 80–0467
smoking habit in the United States, 80–1523
therapeutic potential, 80–0449
use in adolescents and adults in the United States, 80–0399
use in Australia, 80–0467
use in high school students in the United States, 80–0414

CARBOHYDRATES
(*See also* **SUGAR CONTENT**)
characterization of glycophosphoceramide in tobacco leaf, 80–0224
determination in Bulgarian oriental tobacco, 80–0225
determination in Polish bright and dark tobacco, 80–0218
effect of alpha-amylase and amyloglucosidase on sugar content in tobacco, 80–0558
effect of maturation stage on starch levels in tobacco, 80–0697
effect of nutrient deficiency on levels in tobacco, 80–1315
effect of planting time on levels in tobacco, 80–1067
effect of polysaccharide wrapper on burning characteristics and smoke quality of cigarettes, 80–0509
effect of reaction product of sugar, carbonyl compounds, and valine on tobacco flavor, 80–0622
esters with aliphatic acid as tobacco substitutes, 80–0659
thermally degraded carbohydrates as tobacco substitutes, 80–0628, 80–0643
in tobacco leaf, effect of homogenized leaf curing, 80–1261
as tobacco substitutes, 80–0635, 80–0659

CARBON DIOXIDE
effect of cigarette paper and filters on diffusion, 80–0019
effect of combustion and thermal decomposition on formation in cigarettes, 80–0868
as solvent for nicotine extraction in tobacco, 80–0565

CARBON DIOXIDE CONTENT
in cigarette smoke, effect of 2-pyrrolidone polymer filters, 80–0515

CARBON MONOXIDE
air pollution and health risks in cyclists and, 80–1310
in arteriosclerosis and cardiovascular disease etiology, 80–1458
in arteriosclerosis etiology in pigs, 80–0272
effect of cigarette paper and filters on diffusion, 80–0019
effect of cigarette paper on delivery, 80–0512
effect of combustion and thermal decomposition on formation in cigarettes, 80–0868
effect of inhaling in angina patients, 80–0782
effect of passive smoking on eyes, heart rate, and respiration, 80–1388
effect of smoking characteristics on delivery, 80–1501
effect on aorta and coronary arteries in rabbits, 80–1117
effect on aorta, arteries, and lungs in rabbits, 80–1353
effect on arteries in pigs, 80–1647
effect on auditory discrimination, 80–0162
effect on behavior in animals and man, 80–0898
effect on blood parameters and tissues in rats, 80–0879
effect on carboxyhemoglobin levels, 80–1092
effect on carboxyhemoglobin levels in nonsmokers, 80–1358, 80–1625
effect on carboxyhemoglobin levels in nonsmokers in enclosed spaces, 80–0378
effect on carboxyhemoglobin levels in smokers vs. nonsmokers, 80–1198
effect on carboxyhemoglobin levels in smoking vs. nonsmoking mechanics, 80–1622
effect on electrocardiogram in smokers vs. nonsmokers, 80–1198
effect on fetal development in mice and rabbits, 80–0907
effect on heart function in nonsmokers, 80–1210
effect on heart rate of nonsmokers during exercise at high altitude, 80–0070
effect on learning, 80–1092
effect on lipid content in lungs and trachea in squirrels, 80–0062
effect on protein synthesis in lungs in rats, 80–1346
effect on psychomotor performance, 80–1131
effect on red blood cell parameters in smoking vs. nonsmoking submarine patrolmen, 80–1663
effect on retina in cats, 80–0725, 80–0894
effect on vigilance performance, 80–0253
effect on work capacity of nonsmokers at high altitude, 80–0070
uptake in conducting airways in lungs, 80–1341

CARBON MONOXIDE CONTENT
(*See also* **CARBOXYHEMOGLOBIN LEVELS**)
in alveolar air, effect of cigarette draw resistance, 80–1759
in alveolar air, effect of nicotine content in cigarettes, 80–1760
in alveolar air, effect of smoking characteristics, 80–1760, 80–1785
in blood, effect of oronasal cigarette smoke exposure in monkeys, 80–1657
in blood, mathematical model of smokers, 80–1126
in Canadian cigarette brands, 80–0869
in cigarette smoke, effect of burn control additives and paper permeability, 80–0175
in cigarette smoke, effect of cigarette paper, 80–0033
in cigarette smoke, effect of filter additives, 80–1017
in cigarette smoke, effect of filters, 80–0029
in cigarette smoke, effect of humidity and puff parameters, 80–1599
in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
in cigarette smoke, effect of 2-pyrrolidone polymer filters, 80–0515
in cigarette smoke, effect of tobacco-free center in cigarette, 80–0668
in cigarette smoke, effect of ventilated filters, 80–1016

in cigarette smoke of Canadian brands, 80–1603
in cigarettes, effect on carboxyhemoglobin levels, 80–1352
in cigarettes, effect on smoking characteristics, 80–1757
in cigarettes, recommendations for publication, 80–1288
determination in blood by gas chromatography vs. Oximeter, 80–0014
determination in cigarette smoke, 80–0698
effect of cigarette paper pyrolysis in enclosed spaces, 80–1129
effect of smoke inhalation in baboons, 80–1649
in expired air and blood in smokers vs. nonsmokers, 80–0738
in expired air, as outcome predictor in cessation of smoking, 80–0172
in expired air, effect of modification of smoking characteristics, 80–0816
in expired air, effect of ventilated cigarette holders, 80–1767
in expired air in nonsmokers, 80–1358
in herbal cigarettes in Australia, 80–1090
in less hazardous cigarettes, 80–1321
recommendations for cigarette package labeling in Great Britain, 80–0013
in tobacco, effect of boiling, drying, and rehydration, 80–0547, 80–0548
in tobacco smoke, effect of ruthenium catalyst, 80–1820
in tobacco smoke, effect of tobacco varieties, 80–1086
in tobacco smoke, effect of transition metals, 80–0566

Carbonyl compounds *See* **ALDEHYDES; CARBOXYLIC ACIDS; KETONES**

CARBOXYHEMOGLOBIN LEVELS
(*See also* **CARBON MONOXIDE CONTENT**)
effect of active charcoal filter, 80–1765
effect of air pollution in smokers vs. nonsmokers, 80–1621
effect of air pollution in smoking vs. nonsmoking traffic policemen, 80–1614
effect of carbon monoxide, 80–1092
effect of carbon monoxide and non-nicotine cigarettes in angina patients, 80–0782
effect of carbon monoxide content in cigarettes, 80–1352
effect of carbon monoxide exposure in smoking vs. nonsmoking mechanics, 80–1622
effect of carbon monoxide in nonsmokers, 80–1358, 80–1625
effect of carbon monoxide in smokers vs. nonsmokers, 80–1198
effect of cigar smoke in lung disease patients, 80–0353
effect of cigarette length, 80–1246
effect of cigarette paper pyrolysis in nonsmokers, 80–1129
effect of 48-hour abstinence from smoking in pregnant women, 80–0720
effect of inhaling in cigar and pipe smokers, 80–1302
effect of maternal smoking during pregnancy, 80–1619
effect of nicotine content in cigarettes, 80–1138
effect of passive smoking in nonsmokers in enclosed spaces, 80–0378
effect of smoking, 80–0973
effect of smoking characteristics, 80–1790
effect on auditory discrimination, 80–0162
effect on hematocrit, 80–0973
effect on hemoglobin affinity for oxygen in smokers vs. nonsmokers, 80–0731
effect on lung function in nonsmokers, 80–1186
in evaluation of less hazardous cigarettes, 80–0409
in heavy vs. light smokers vs. nonsmokers, 80–1359
in mother and infant, effect of maternal smoking, 80–0137
secondary Poisson process models of smoking, 80–1642
in smokers vs. nonsmokers, 80–0738, 80–0885
in smokers vs. nonsmokers in rural vs. urban areas, 80–1623
in smoking vs. nonsmoking police officers, 80–0071
in smoking vs. nonsmoking welders, 80–1650

CARBOXYLIC ACIDS
citric and malic acid in tobacco wastes, 80–1326
effect of dicarboxylic acids and derivatives on tobacco expansion, 80–0500
effect of isopropenyl ester polymers on tobacco flavor, 80–0584
effect of isovaleric and methyl valeric acids on flavor of tobacco substitutes, 80–0628
effect of 2-methyl-4-pentenoic acid ethyl ester derivatives on tobacco aroma and flavor, 80–0592

CARCINOEMBRYONIC ANTIGEN LEVELS
in mammary neoplasm diagnosis in smokers vs. nonsmokers, 80–1389
smoking and respiratory tract infections and, 80–0760
in smoking vs. nonsmoking alcoholic liver disease patients, 80–1335

CARCINOGENESIS
(*See also* **CARCINOGENS; NEOPLASMS**)
benzo(*a*)pyrene and diol-epoxide I in tracheal epithelium in rats, 80–0741
benzo(*a*)pyrene and silicon dioxide in hamsters, 80–1659
bibliography of lead-210 and polonium-210 in tobacco smoke, 80–1385

cigarettes in bacterial vs. mouse skin response tests, 80–0251
dimethylbenz(*a*)anthracene in trachea in rats, 80–1660
environment and heredity and, 80–0921
N'-nitrosonornicotine and 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone in rats, 80–0722

CARCINOGENICITY REDUCTION
(*See also* **CARCINOGENESIS**)
effect of tobacco processing, 80–0460

CARCINOGENS
(*See also* **CARCINOGENESIS; COCARCINOGENS**)
cigarette smoke condensate fractions, 80–0740
dimethylbenz(*a*)anthracene, 80–1660
in food, 80–0463
4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone, 80–0722
N'-nitrosonornicotine, 80–0722
smoking and occupational exposure in lung neoplasm etiology, 80–0081

CARCINOMA, BRONCHOGENIC
(*See also* **BRONCHIAL NEOPLASMS; LUNG NEOPLASMS**)
effect of smoking and heredity on incidence, 80–0921
induced by benzo(*a*)pyrene in hamsters, 80–1658
smoking and, 80–0857, 80–1156
smoking and incidence in France, 80–1673
smoking habit in patients, 80–1155

CARCINOMA, EPIDERMOID
(*See also* **BRONCHIAL NEOPLASMS; LUNG NEOPLASMS; SKIN NEOPLASMS**)
in smoking vs. nonsmoking nickel workers, 80–0305

Cardiac output *See* **HEART FUNCTION**

CARDIOVASCULAR DISEASES
(*See also* **CEREBROVASCULAR DISORDERS; CORONARY DISEASE; HEART DISEASES; ISCHEMIA; MYOCARDIAL INFARCT; THROMBOSIS; VASCULAR DISEASES**)

carbon monoxide in etiology of, 80–1458
effect of blood glucose levels on mortality in smokers vs. nonsmokers, 80–1445
effect of risk factor reduction program on smoking habit, 80–1749
effect of smoking and aging on risk in oral contraceptive users, 80–1719
effect of smoking and oral contraceptives on incidence, 80–0354
effect of smoking and oral contraceptives on mortality, 80–0377
effect of smoking and oral contraceptives on risk, 80–0352
effect of smoking on incidence and mortality, 80–1729
effect of smoking on mortality rates in Japan, 80–1456
effect of smoking on mortality ratios, 80–0916
effect of smoking on mortality risk in hypertensives, 80–1437
effect of smoking on risk, 80–1462
environmental pollution and, 80–0978
mortality in Mormons vs. non-Mormons, 80–0119
mortality in Switzerland, 80–0076
mortality rates of smokers vs. nonsmokers, 80–0290
mortality ratios in smokers vs. nonsmokers vs. ex-smokers in the United States, 80–1395
multimedia antismoking campaign in prevention program, 80–0120
nicotine in etiology of, 80–1458
risk factor clustering, 80–1709
risk factors, 80–0112
risk factors in adolescents in Switzerland, 80–1193
risk factors in Sweden, 80–0981
smoking and, 80–0112, 80–0792, 80–0857, 80–0959
smoking and hyperlipidemia in oral contraceptive users and, 80–1199
smoking and mortality rates in the United States, 80–0752
smoking as risk factor for mortality in black vs. white males vs. females, 80–1710
smoking habit and alcohol consumption in physicians and, 80–0828
smoking in etiology of, 80–0077, 80–1458
sociocultural mobility and, 80–0115
worldwide funding for health education as preventive medicine, 80–1044

CARDIOVASCULAR SYSTEM
(*See also* **BLOOD VESSELS; HEART**)
effect of maternal smoking in fetus, 80–1732
effect of passive smoking in nonsmokers, 80–0964
effect of rapid smoking, 80–0786

CATECHOLAMINE LEVELS
effect of smoking and beta adrenoceptor blocking agents on adrenaline and noradrenaline levels in blood, 80–1465
in urine, effect of high- vs. low-nicotine cigarettes, 80–1505
in urine, effect of smoking in oral contraceptive users, 80–0750

Catechol *See* **PHENOLS**

CATECHOLAMINES
effect of abstinence from smoking on adrenaline and noradrenaline levels, 80–0043

effect of nicotine and pilocarpine in brain in rats, 80–1102
effect of nicotine on dopamine metabolism in rats, 80–1349
effect of nicotine on noradrenaline release from hypothalamic synaptosomes, 80–0748
effect of nicotine on noradrenaline release in artery in rabbits, 80–0739
effect of nicotine on norepinephrine release in animals, 80–0267

CELLS
(*See also* **CELLS, EPITHELIAL; ERYTHROCYTES; GOBLET CELLS; LEUKOCYTES)**
acinar cell dysplasia in pancreas in smokers vs. nonsmokers, 80–1157
bacterial adherence to pharyngeal cells in smokers vs. nonsmokers vs. bronchitics, 80–1685
effect of betel nut and tobacco on DNA synthesis in lymphocytes and tumor cells, 80–0289
effect of high- vs. low-tar cigarette smoke condensates on mitochondria in brown fat cells in hamsters, 80–0735
effect of nicotine on HeLa cell division, 80–1387
lymphocytes and T cells in smoking vs. nonsmoking sarcoidosis patients, 80–0324

CELLS, EPITHELIAL
(*See also* **GOBLET CELLS)**
carcinogenicity of benzo(*a*)pyrene and diol-epoxide I in trachea in rats, 80–0741

CENTRAL NERVOUS SYSTEM
effect of nicotine on arousal and emotion, 80–1002
effect of nicotine on seizure susceptibility in mice, 80–0048
effect of nicotine on tyrosine hydroxylase activity in rats, 80–1386

CEREBROVASCULAR DISORDERS
(*See also* **CARDIOVASCULAR DISEASES)**
effect of smoking and oral contraceptives on hemorrhagic stroke risk, 80–0803
effect of smoking on risk for subarachnoid hemorrhage in cerebral aneurysm patients, 80–0128
risk factors for cerebral infarction and transient ischemic attacks, 80–0795
risk factors for stroke in the young and middle-aged, 80–0361
smoking habit in stroke patients, 80–0357, 80–0361, 80–1442

Cervix neoplasms *See* **UTERINE NEOPLASMS**

CESSATION OF SMOKING
(*See also* **REDUCTION OF SMOKING; SMOKING CONTROL PROGRAMS; WITHDRAWAL CLINICS)**
American Cancer Society's three point program for physician use, 80–1047
attitudes of ex-smokers vs. recidivists, 80–0152
attitudes toward method counseling, 80–0161
behavior in ex-smokers vs. recidivists and, 80–0169
carbon monoxide in expired air and blood thiocyanate levels as outcome predictors, 80–0172
in coronary disease intervention program, 80–1446
credibility ratings of methods, 80–0435
effect of abstinence on adrenaline and noradrenaline levels, 80–0043
effect of abstinence on cognition, 80–0043
effect of abstinence on electroencephalogram, 80–1632
effect of American Cancer Society Stop Smoking Program, 80–0436
effect of attitudes on acupuncture treatment success, 80–1773
effect of coronary risk factor intervention program, 80–1726
effect of education and health awareness in obstructive lung disease patients, 80–0940
effect of exercise on physical fitness in bronchitics, 80–1427
effect of 24-hour abstinence and exercise on cardiorespiratory functions, 80–1708
effect of 48-hour abstinence on oxygen availability in pregnant women, 80–0720
effect of locus of control, 80–1013
effect of motivation and spouse's smoking habit on success, 80–1801
effect of nicotine chewing gum on withdrawal symptom incidence, 80–1535
effect of personality, 80–1498
effect of rapid smoking on cardiovascular system, 80–0786
effect of self-control on success, 80–0156
effect of smoking habit and attitudes on recidivism, 80–0817
effect of social support and paraprofessional training on recidivism, 80–0434
effect on abruptio placentae and placenta praevia incidence, 80–1474
effect on alcohol consumption and body weight, 80–0837
effect on body weight, 80–1446
effect on body weight in women, 80–1485, 80–1722
effect on bruxism, 80–0821
effect on cardiorespiratory symptoms and coronary disease risk factors, 80–1441
effect on cardiovascular disease risk, 80–1462
effect on coronary heart disease mortality rates, 80–1221
effect on fetal and neonatal mortality rates, 80–1474
effect on hematocrit, 80–0973
effect on lung function, 80–0094

effect on lung neoplasm mortality rates in asbestos workers, 80–1405
effect on lung neoplasm risk, 80–0925
effect on mortality rate in myocardial infarct patients, 80–0356, 80–1728
effect on mortality ratios in the United States, 80–1395
effect on myocardial reinfarction incidence, 80–0789
effect on prognosis in myocardial infarct patients, 80–0376
effect on pulmonary surfactant phospholipid levels, 80–0323
effect on respiratory symptom incidence, 80–1577
effect on sleep, 80–0807
evaluation of Atlanta Lung Association Withdrawal Clinics, 80–0432
in heavy vs. light smokers, 80–1498
mail program, 80–0431
manual for education of chronic obstructive pulmonary disease patients, 80–1165
methods, 80–0833
methods, abrupt vs. gradual, 80–0166
methods, abrupt withdrawal, 80–1013
methods, acupuncture, 80–1254, 80–1537, 80–1773, 80–1798
methods, aerobic exercise programs, 80–0187
methods, antismoking pill, 80–1250
methods, auricular acupuncture, 80–1795
methods, auricular therapy, 80–1253
methods, aversive therapy, 80–0166, 80–0431
methods, aversive therapy with maintenance, 80–0437
methods, behavioral, 80–0431, 80–0433, 80–0812, 80–0825, 80–0832, 80–0834, 80–1538, 80–1803
methods, bibliotherapy, 80–1801
methods, cigarette holder for electric shock delivery, 80–0666
methods, counseling and focused smoking, 80–1536
methods, counseling and hypnosis, 80–1799
methods, detoxifying therapeutic composition, 80–0667
methods, drug therapy vs. psychotherapy, 80–1011
methods, fear, 80–0171, 80–0438
methods, focused smoking, 80–0836
methods, four-level intervention program, 80–0819
methods, group counseling, 80–0167
methods, group therapy, 80–0831
methods, healing, 80–1796
methods, hypnosis, 80–0817
methods, minimal intervention, 80–0831
methods, multicomponent, 80–0837
methods, muscle relaxation, 80–0834
methods, nicotine fading vs. self-monitoring vs. American Cancer Society program, 80–1012
methods, positive reinforcement, 80–0164
methods, propranolol, 80–0835
methods, rapid smoking, 80–1800
methods, relaxation, 80–1797
methods, review, 80–1014
methods, self-control, 80–0431, 80–1248
methods, self-help, 80–0163, 80–0170, 80–1804
methods, self-hypnosis, 80–1251
methods, self-management and social support, 80–0439
methods, self-monitoring, 80–1794
methods, television program in Finland, 80–1255
methods, ventilated cigarette holders, 80–1790
motivation and, 80–0999, 80–1170, 80–1540
motivation in medical students in the U.S.S.R. and, 80–1240
mouthpiece for progressive nicotine reduction in cigarette smoke, 80–0662
nicotine and cotinine levels in urine as verification of self-report, 80–0288
obstacles to quitting, 80–0165
oral behavior and, 80–0820
personality and choice of method, 80–1010
as preventive medicine, 80–1304, 80–1305
as preventive medicine for coronary disease, 80–1725
as preventive medicine for lung neoplasms in asbestos-exposed workers, 80–1280
as preventive medicine in obstructive airway disease patients, 80–1423
programs, SOS Stop Smoking Clinic, 80–1252
programs, Five-Day Plan in Great Britain, 80–1805
programs in hospitals, 80–1284
programs in the workplace, 80–1249, 80–1252, 80–1291
psychological effects, 80–1783
research methodology, 80–1539
risk—benefit evaluation of rapid smoking, 80–0838
role of physicians, 80–0173, 80–0850, 80–1047, 80–1191, 80–1563, 80–1770
self-regulatory behavior and, 80–0152
self-report as verification, 80–1568
success rates in men and women, 80–0996
survey of use of aids in the United States, 80–0815
theory for predicting recidivism, 80–0440
valerian and vitamins in treatment of withdrawal symptoms, 80–0664
7-week behavior modification program, 80–0832
withdrawal symptoms and, 80–1802

Chewing tobacco *See* **TOBACCO CHEWING**

CHILDREN
effect of air pollution and passive smoking on respiratory symptom incidence in France, 80–1420
effect of antismoking campaigns in France, 80–1000
effect of asthma and bronchitis in infancy on lung function in smokers vs. nonsmokers, 80–1431
effect of health education and socioeconomic status on attitudes toward smoking, 80–1786

effect of health education on onset of smoking, 80–0408
effect of maternal smoking and socioeconomic status on morbidity and mortality, 80–1738
effect of maternal smoking during pregnancy on morbidity, 80–0134
effect of maternal smoking on development, 80–0985
effect of parental attitudes and behavior, 80–1752
effect of parental attitudes on smoking habit, 80–1055
effect of parental smoking and air pollution on respiratory tract disease incidence, 80–0952
effect of parental smoking on respiratory symptom incidence, 80–0802, 80–1416
effect of parental smoking on smoking habit, 80–1000, 80–1055
effect of parental smoking on smoking habit in Great Britain, 80–1527
effect of passive smoking on lung function, 80–1425
effect of smoking and respiratory tract diseases on lung function, 80–1701
effect of smoking and social factors on bronchitis incidence, 80–1691
effect of teachers' smoking on smoking habit in Great Britain, 80–0402
nicotinism in infants, 80–0398
respiratory symptoms in smokers vs. nonsmokers, 80–1754
smoking habit and personality, 80–1525
smoking habit in Sweden, 80–0419

CHROMIUM
(*See also* **METALS**)
in German cigarettes, 80–0229

Chronic bronchitis *See* **BRONCHITIS**

CHRONIC BRONCHOPULMONARY DISEASE
(*See also* **BRONCHITIS; EMPHYSEMA; LUNG DISEASES; OBSTRUCTIVE AIRWAY DISEASES; RESPIRATORY TRACT DISEASES**)
role of physicians in cessation of smoking counseling in patients, 80–1191
smoking in etiology of, 80–0852

Chronic obstructive lung disease *See* **CHRONIC BRONCHOPULMONARY DISEASE; OBSTRUCTIVE AIRWAY DISEASES**

CIGAR WRAPPER
effect of crop rotation on tobacco quality and yield, 80–0685
effect of polysaccharides on burning characteristics and smoke quality, 80–0509
effect of reconstituted tobacco foam vs. natural leaf on tar delivery, 80–0497

CIGARETTE BUTTS
effect of cigarette length reduction on nicotine content, 80–1751
effect of ventilated cigarette holders on weight, 80–1767

Cigarette holders *See* **SMOKING ARTICLES**

CIGARETTE PAPER
cadmium and lead content in New Zealand brands, 80–1595
determination of humic acid and dyes, 80–0706
determination of trace elements, 80–0011
effect of burn control additives on carbon monoxide content of smoke, 80–0175
effect of gamma alumina filler on tobacco smoke constituents, 80–0556
effect of perforation on smoke constituent levels, 80–1264
effect of permeability on carbon monoxide content of smoke, 80–0175
effect of porosity and length on gaseous components of smoke, 80–0230
effect of porosity on chemical composition of smoke, 80–0033
effect of porosity on nicotine content of mainstream smoke, 80–0246
effect of pyrolysis on carbon monoxide levels in enclosed spaces, 80–1129
effect of pyrolysis on carboxyhemoglobin levels in nonsmokers, 80–1129
effect on carbon monoxide and carbon dioxide diffusion, 80–0019
effect on carbon monoxide, nicotine, and tar delivery, 80–0512
silicon dioxide coating for self-extinguishing cigarettes, 80–0494

Cigarette smoke *See* **SMOKE, CIGARETTE**

Cigarette smoke, gas phase *See* **GAS PHASE, CIGARETTE SMOKE**

Cigarette smoking *See* **SMOKING**

Cigarette tars *See* **TARS, CIGARETTE**

CIGARETTES
American Automatic Merchandiser survey of vending machine sales, 80–1267
cadmium and lead content in New Zealand brands, 80–1595
carbon monoxide and tar content in herbal brands in Australia, 80–1090
carbon monoxide, nicotine, and tar content of Canadian brands, 80–0869
carcinogenicity in bacterial vs. mouse skin response tests, 80–0251

consumption and imports in Egypt, 80–1553
consumption in Nigeria, 80–1491
consumption in the United States, 80–1028
consumption in West Germany, 80–0446
determination of alkaloid content by nonaqueous conductimetric titration, 80–0705
determination of carbon monoxide content, 80–0698
determination of nicotine and tar contents in brands in Great Britain, 80–0700
determination of smoke constituents in Canadian brands, 80–1603
determination of uranium content in Indian brands, 80–1596
dispenser for reduction of smoking, 80–0660
effect of advertising on consumption in Great Britain, 80–1300
effect of air dilution on smoke components, 80–0234
effect of alumina and silica filter on smoke constituents, 80–0505
effect of combustion and thermal decomposition on carbon oxide formation, 80–0868
effect of draw resistance on smoking characteristics, 80–1759
effect of high- vs. low-delivery brands on smoking characteristics, 80–1757
effect of high- vs. low-nicotine content on vasopressin levels in blood, 80–1653
effect of high- vs. low-tar smoke condensates on mitochondria in brown fat cells in hamsters, 80–0735
effect of inhaling filtered vs. nonfiltered smoke on nitrosamine retention in respiratory system, 80–0718
effect of length on blood nicotine, carboxyhemoglobin, and nicotine delivery, 80–1246
effect of length on smoking characteristics and smoking habit, 80–1246
effect of length reduction on smoking characteristics, 80–1751
effect of masculine and feminine brand names on flavor evaluation in males vs. females, 80–1763
effect of nicotine and tar content on respiratory system, 80–1687
effect of nicotine content on circulatory and metabolic parameters, 80–1138
effect of nicotine content on memory and psychomotor performance in high- vs. low-arousal smokers, 80–1534
effect of nicotine content on smoking habit, 80–0829, 80–1777
effect of periphery and rod on smokestream formation, 80–0028
effect of physical parameters and stalk position on smoke constituent levels, 80–1608
effect of polysaccharide wrapper on burning characteristics and smoke quality, 80–0509
effect of pyrolysis on chlorofluoromethane decomposition, 80–0702
effect of pyrolysis on nicotine degradation, 80–0708
effect of smoking filtertipped vs. non-filtertipped types on lung function, 80–0781
effect of tar content on pulmonary alveolar macrophages in rats, 80–1638
effect of tar content on respiratory symptom incidence and sputum production, 80–1177
effect of tar content on smoking characteristics, 80–1785
effect of tar content on smoking characteristics and habit, 80–1750
effect of tar content on smoking habit, 80–1294
effect of taxation on consumption in Kentucky, 80–1032
effect of tobacco-free center on carbon monoxide content of smoke, 80–0668
effect of tube-like smoke vector on smoke constituents, 80–0516
exports in the United States, 80–1276
fluoride and sugar content, 80–0696
heavy metal content of German brands, 80–0229
identification of tobacco mosaic virus and para-tobacco mosaic virus in German brands, 80–0286
as ignition sources for upholstered furniture, 80–0451
industry's viewpoint of research needs, 80–1289
legislation for flammability, 80–1842
legislation on bootlegging in the United States, 80–1558, 80–1559
linear multiple regression model for consumption in Switzerland, 80–1825
low-tar brand sales in the United States, 80–1555
mathematical model for predicting composition of smoke, 80–0017
Maxwell international estimates for sales, 80–1552
menthol in dermatitis etiology, 80–0138
methoprene content in butt and smokestreams, 80–1266
nickel content of Norwegian vs. U.S. brands, 80–0110
nicotine and tar content in U.S. brands, 80–0220
production in Pakistan, 80–1824
recommendations for carbon monoxide content in Great Britain, 80–0013
recommendations for development of less hazardous brands, 80–0859
recommendations for publication of carbon monoxide content, 80–1288
recommendations of Federal Trade Commission for advertising and labeling, 80–1306
sales of low-tar brands in Canada, 80–1036
sales trends in Great Britain, 80–0176
silicon dioxide paper coating for self-extinguishing feature, 80–0494
smoking characteristics in evaluation of health effects of less hazardous variety, 80–0409

- synthetic mouthpiece paper, 80–1818
- tar reduction in Canadian brands, 80–1036
- taxation in the United States, 1863-1979, 80–1275
- use of negatively charged, hydrophobic fibers in filters and tobacco, 80–0507
- ventilated filter wrap for delivery of volatile flavoring compounds, 80–0525
- worldwide consumption, 80–1026
- worldwide trends in brand sales, 80–1271

CIGARETTES, HIGH-NICOTINE
- effect of smoke inhalation on elastase and antitrypsin levels in dogs, 80–0274
- effect on arousal and emotion in introverts vs. extraverts, 80–1001
- effect on blood circulation, blood pressure, and heart rate, 80–1206
- effect on muscle tonus, 80–0044
- effect on neurophysin levels in blood, 80–0259
- effect on nicotine levels in blood plasma, 80–1206
- effect on vasopressin levels in blood, 80–1653
- physiological and psychological effects, 80–1505

CIGARETTES, LESS HAZARDOUS
- agricultural and processing techniques in the United States, 80–1846
- determination of gas phase smoke constituents in Canadian brands, 80–1603
- determination of tar content, 80–1606
- effect of health education on consumption in the United States, 80–1511
- effect on health risk, 80–1292
- effect on lung neoplasm mortality rate, 80–1679
- effect on smoking habit, 80–0150
- health effects, 80–0181, 80–0464
- lung neoplasm mortality in Austria and, 80–1150
- recommendations for development, 80–0859
- reduction of carbon monoxide, 80–1321
- sales of low-tar brands in the United States, 80–1028
- smoking characteristics in evaluation of health effects, 80–0409
- statistical methodology for risk evaluation, 80–1283
- trends in manufacturing, 80–1565

CIGARETTES, LOW-NICOTINE
- effect of smoke inhalation on elastase and antitrypsin levels in dogs, 80–0274
- effect on arousal and self-reported emotion in introverts vs. extraverts, 80–1001
- effect on blood circulation, blood pressure, and heart rate, 80–1206
- effect on muscle tonus, 80–0044
- effect on nicotine levels in blood plasma, 80–1206
- effect on smoking habit, 80–0459
- effect on vasopressin levels in blood, 80–1653
- physiological and psychological effects, 80–1505
- smoking habit and, 80–1528

CIGARETTES, NONTOBACCO
- effect of herbal cigarettes on lung function in asthmatics, 80–1699
- effect of smoking in angina patients, 80–0782

CIGARS
- consumption, production, and exports in the Netherlands, 80–1269
- effect of reconstituted tobacco foam on aroma and flavor, 80–0497
- exports in the United States, 80–1276
- 1979 Maxwell Report on sales, 80–0843
- production and sales in Holland, 80–1268
- sales trends in Great Britain, 80–0176
- zinc as tracer for evaluation of hydrophobic coatings in reconstituted tobacco, 80–0498

CIGARS, LITTLE
- consumption and production in the Netherlands, 80–1269

CILIARY ACTIVITY
(*See also* **PULMONARY CLEARANCE**)
- effect of ipratropium bromide in obstructive airway disease patients, 80–0339

Ciliastatic agents *See* **ACETALDEHYDE; ACROLEIN; FORMALDEHYDE**

COCARCINOGENS
(*See also* **CARCINOGENS**)
- benz(*a*)anthracene and silica particles, 80–0057
- benzo(*a*)pyrene and silicon dioxide in hamsters, 80–1659
- benzo(*a*)pyrene and soot in *Salmonella typhimurium*, 80–0054
- cigarette smoke and asbestos, 80–0713, 80–1394
- cigarette smoke and coffee, 80–0085
- cigarette smoke and influenza virus in lung neoplasm etiology, 80–0285
- cigarette smoke and occupational exposure, 80–0081
- cigarette smoke and radiation, 80–0316

COGNITION
(*See also* **LEARNING; PERCEPTION**)
- effect of abstinence from smoking, 80–0043
- effect of nicotine content in cigarettes, 80–0255
- efficacy vs. symptom attribution in reduction of smoking, 80–1497
- self-categorization of smoking habit and cognitive dissonance, 80–1791

COLON
(*See also* **INTESTINES**)
- DNA binding of benzo(*a*)pyrene, 80–0039

COMBUSTION TEMPERATURE
effect of filters on chemical composition of cigarette smoke, 80–0029

CONTRACEPTIVES, ORAL
effect of smoking and aging on cardiovascular disease risk in users, 80–1719
effect of smoking on myocardial infarct risk in users, 80–1718
effect on blood lipids and lipoproteins in adolescents, 80–0364
effect on epinephrine and norepinephrine levels in urine in smokers vs. nonsmokers, 80–0750
effect on phenytoin metabolism in epileptics, 80–1342
effect on theophylline metabolism, 80–0268
interactive effect with smoking and hypertension on myocardial infarct risk, 80–1215
interactive effect with smoking on cardiovascular disease incidence, 80–0354, 80–0372
interactive effect with smoking on cardiovascular disease mortality, 80–0377
interactive effect with smoking on cardiovascular disease risk, 80–0352
interactive effect with smoking on hemorrhagic stroke risk, 80–0803
interactive effect with smoking on subarachnoid hemorrhage risk, 80–0128
smoking and hyperlipidemia in users, 80–1199
smoking, blood lipids, and blood pressure in users vs. nonusers, 80–0379
smoking habit during pregnancy in users, 80–1739
smoking habit in users, 80–1463

COPPER CONTENT
effect of nicotine in organs in rats, 80–1374
in German cigarettes, 80–0229
in Iraqi vs. U.S. tobacco varieties, 80–0036
in tobacco varieties in Yugoslavia, 80–0692

Coronary atherosclerosis *See* **ARTERIOSCLEROSIS; CORONARY DISEASE**

CORONARY DISEASE
(*See also* **ANGINA PECTORIS; ARTERIOSCLEROSIS; HEART DISEASES; MYOCARDIAL INFARCT; THROMBOSIS**)
allergic response to tobacco extract in smoking vs. nonsmoking patients, 80–0976
antismoking campaigns in risk factor intervention program in Belgium, 80–0969
asymptomatic hyperglycemia and, 80–1730
blood sugar and, 80–0975
cadmium levels in blood in smoking vs. nonsmoking patients, 80–0362
cessation of smoking and diet in intervention program, 80–1446
cessation of smoking as preventive medicine, 80–1725
coronary artery stenosis in smokers vs. nonsmokers, 80–0118
effect of body weight on incidence and risk factors in women, 80–1722
effect of cessation of smoking on mortality rates, 80–1221
effect of cessation of smoking on risk factors, 80–1441
effect of exercise on high density lipoprotein, cholesterol, and insulin levels in patients, 80–0375
effect of exercise on incidence in smokers vs. nonsmokers, 80–0972
effect of ischemic heart disease risk factor reduction program on smoking habit and body weight, 80–1229
effect of passive smoking in patients, 80–0358
effect of risk factor intervention program on anxiety and depression, 80–1703
effect of risk factor intervention program on heart function, 80–1703
effect of risk factor reduction on mortality rates, 80–1194
effect of risk factors on arteriosclerotic heart disease mortality rates, 80–1209
effect of smoking and aging on ischemic heart disease mortality risk, 80–1464
effect of smoking and blood sugar on mortality rates, 80–1200
effect of smoking and cultural upbringing on prevalence in Japanese in the United States, 80–1452
effect of smoking and other risk factors, 80–1201
effect of smoking and other risk factors on erythrocytes, 80–1449
effect of smoking and other risk factors on incidence, 80–1714
effect of smoking and socioeconomic status on risk, 80–0966
effect of smoking, ethnic group, and socioeconomic status on risk, 80–1448
effect of smoking on heart function in patients, 80–1436
effect of smoking on incidence in women in Great Britain, 80–0983
effect of smoking on mortality rate, 80–0293
effect of smoking on prognosis in bypass patients, 80–1205
effect of smoking on risk, 80–1054, 80–1454
health education in intervention program in Belgium, 80–1447
hyperglycemia as risk factor, 80–1461
hyperglycemia in smokers vs. nonsmokers vs. ex-smokers and, 80–1216
intervention program for high-risk individuals, 80–0422
morbidity and mortality in smokers vs. nonsmokers vs. ex-smokers, 80–1717

mortality in marathoners, 80–0122
mortality in the United States, 80–1211, 80–1459
psychological risk factors in smokers vs. nonsmokers, 80–0121
psychosocial factors in the Framingham study, 80–1203
psychosocial risk factors, 80–0117
questionnaire for assessment of risk factors, 80–0982
relative risk in first degree relatives of myocardial infarct patients, 80–0126
relative risk in smokers vs. nonsmokers, 80–0115
risk factor intervention program, 80–1726
risk factor intervention program in Finland, 80–0367
risk factors, 80–0373, 80–0974, 80–0980, 80–1713, 80–1729, 80–1730, 80–1731
risk factors and stress in smokers vs. nonsmokers, 80–1715
risk factors for sudden death, 80–0791
risk factors in Aborigines in Australia, 80–0344
risk factors in Finland, 80–1220
risk factors in ischemic heart disease patients, 80–0979
risk factors in males in Scotland and Sweden, 80–0359
risk factors in Maoris in New Zealand, 80–0345
risk factors in nonagenarians, 80–0349
risk factors in prediction and prevention, 80–0114
risk factors in Quebec, 80–1214
risk factors in safety personnel in California, 80–0127
risk factors in women, 80–0369, 80–0787
role of health insurance incentives in prevention, 80–1043
self-scoring risk test, 80–0113
smokers vs. nonsmokers vs. ex-smokers, 80–1723
smoking and, 80–0127, 80–0755, 80–1711
smoking and blood sugar and, 80–1204, 80–1713, 80–1729, 80–1731
smoking and coronary artery stenosis, 80–0348
smoking and hyperglycemia and, 80–1219
smoking and hyperglycemia as risk factors, 80–1460
smoking and incidence in psychiatric clinic patients, 80–1049
smoking and mortality trends in the United States, 80–1721
smoking and other risk factors in multicomponent ischemic heart disease prevention program, 80–1723
smoking and personality and, 80–0970
smoking and post-load blood sugar levels and, 80–1213
smoking and vasoconstrictor prostaglandins in coronary artery spasm etiology, 80–0967
smoking as risk factor in women, 80–0977
smoking habit and alcohol consumption in physicians and, 80–0828
smoking habit and electrocardiogram abnormalities, 80–1196
smoking habit and Type A behavior in patients, 80–1705
smoking habit in patients, 80–1155
smoking habit in patients in Canada, 80–0355
smoking in etiology of, 80–0347
ventricular premature beats and other risk factors in smokers vs. nonsmokers, 80–1712

Coronary flow *See* **BLOOD CIRCULATION**

Coronary vessels *See* **BLOOD VESSELS**

COTININE
(*See also* **ALKALOIDS, TOBACCO; NICOTINE METABOLITES)**
determination in blood and urine in dogs, 80–0897
effect of iminium oxidase on nicotine metabolism, 80–1612
effect on 5-hydroxytryptamine metabolism in brain in rats, 80–0887
effect on nicotine binding in brain in rats, 80–1665
effect on reflexes in rats, 80–0887
pharmacokinetics in rats, 80–1652
synthesis of related compounds, 80–0221
use of deuterium for analysis of formation and structure, 80–0245

COTININE LEVELS
in blood and urine, effect of nicotine content in cigarettes, 80–1352
in blood, as verification of self-reported smoking, 80–0430
in blood, effect of ethanol in rats, 80–1091
determination in blood and urine, 80–0219
effect of smoking in males vs. females, 80–1656
in urine, as verification of self-report in cessation of smoking, 80–0288
in urine, effect of smoke inhalation in baboons, 80–1649

COUGH
(*See also* **REFLEXES; RESPIRATORY SYMPTOMS)**
effect of nicotine on reflex in dogs, 80–0914
in smoking vs. nonsmoking adolescents in France, 80–1412
in smoking vs. nonsmoking children, 80–1754

COUNSELING
in cessation of smoking, 80–0167, 80–1536, 80–1799
effect of antismoking campaign in coronary disease intervention program in Belgium, 80–0969

CYANIDE CONTENT
(*See also* **THIOCYANATE LEVELS)**
in cigarette smoke, effect of polystyrene resins, 80–0841
in cigarette smoke of Canadian brands, 80–1603

determination in cigarette smoke, 80–1075
in tobacco smoke, effect of metal salt—amine complexes, 80–0564
in tobacco smoke, effect of transition metals, 80–0566
in tobacco smoke, interactive effect of metal—amine complex with polyalkylene oxide in filters, 80–0514

CYANIDES
in cigarette smoke, effect of filters, 80–0029
effect of hydrogen cyanide on aorta and coronary arteries in rabbits, 80–1117
effect of hydrogen cyanide on aorta, arteries, and lungs in rabbits, 80–1353

DIABETES
(*See also* **BLOOD SUGAR**)
bone mineral content in smoking vs. nonsmoking patients, 80–1745
carbohydrate and lipid metabolism, and insulin requirements in smoking vs. nonsmoking patients, 80–1232
effect of smoking on plasma vasopressin and human neurophysin levels, 80–0140
glossal central papillary atrophy in smokers vs. nonsmokers, 80–1742
nephropathy incidence in smoking vs. nonsmoking patients, 80–0804
proteinuria and retinopathy in smokers vs. nonsmokers, 80-1490
smoking and mortality in patients, 80–1741
smoking habit in patients, 80–0804

Diacetyl *See* **CARBOXYLIC ACIDS; KETONES**

DIBENZ(*a, h*)ANTHRACENE
(*See also* **AROMATIC HYDROCARBONS**)
induction of tracheal neoplasms in rats, 80–0065

DIBENZ(*a, j*)ACRIDINE
in respiratory tract neoplasm induction in hamsters, 80–1659

DIET
bladder neoplasms and, 80–0088
colonic neoplasm mortality in Great Britain and, 80–0294
in coronary disease intervention program, 80–1446
effect of artificial sweeteners on bladder neoplasm risk in smokers vs. nonsmokers, 80–1398
effect of dietary fats on blood platelet function in smokers vs. nonsmokers, 80–0370
effect of maternal smoking and nutritional supplementation on birth weight, 80–1478
effect of nicotine on food intake in rats, 80–1662
effect of smoke inhalation and vitamin A deficiency on larynx in hamsters, 80–0901
effect of smoking and artificial sweeteners on bladder neoplasm risk, 80–1158, 80–1161, 80–1164
effect of smoking and coffee consumption on myocardial infarct risk in women, 80–1727
gastroesophageal reflux and, 80–0389
intestinal neoplasms and, 80–0927
in neoplasm etiology, 80–0319
smoking habit and alcohol consumption in stomach neoplasm patients and, 80–1163

DNA
(*See also* **RNA**)
benzo(*a*)pyrene and nitrosamine binding in esophageal cells, 80–0265
benzo(*a*)pyrene binding in human bronchus and colon cultures, 80–0039
benzo(*a*)pyrene binding in lungs in rats, 80–1130
benzo(*a*)pyrene metabolite binding in embryo cells in hamsters, 80–1096
benzo(*a*)pyrene metabolite binding in liver and lungs in rodents, 80–0727
cigarette smoke condensate-induced chromosome aberrations in lung neoplasm etiology, 80–0723
effect of betel nut and tobacco on synthesis in lymphocytes and tumor cells, 80–0289
effect of marihuana smoke and vitamin C on mitotic abnormality incidence in lung cells, 80–1637
effect of nicotine on HeLa cell division, 80–1387
effect of smoking and coffee consumption on synthesis in leukocytes, 80–0271
effect of tobacco smoke and vitamin C on mitotic abnormality incidence in lung cells, 80–1637
effect of tobacco smoke condensate on repair in yeast, 80–1109
9-hydroxybenzo(*a*)pyrene binding in lungs in rats, 80–1130
incorporation of acetaldehyde in lung cells, 80–0904
incorporation of formaldehyde in lung cells, 80–0904

DRUG ABUSE
(*See also* **CANNABIS**)
attitudes in adolescents in Great Britain, 80–1504
in Australia, 80–0467
effect of alcohol consumption and marihuana smoking on smoking habit, 80–1519
effect of heroin addiction on DNA synthesis in leukocytes, 80–0271
effect of heroin administration on smoking characteristics, 80–1005
effect of smoking and phenacetin abuse on urinary tract neoplasm incidence, 80–1400
high school students in the United States, 80–0414
information sources guide for social scientists, 80–0458
smoking habit and alcohol consumption in adolescents in Puerto Rico and, 80–0427
smoking habit and alcohol consumption in military recruits in Switzerland and, 80–1494
smoking habit in the elderly and, 80–0142

- smoking habit in university students in Nigeria and, 80–0153
- teachers' handbook for health education, 80–1517

DRUG METABOLISM
- aminopyrine, effect of cigarette smoke in liver microsomes in rats, 80–1620
- aniline, effect of cigarette smoke in liver microsomes in rats, 80–1620
- antipyrine and paracetamol in smokers vs. nonsmokers, 80–1103
- antipyrine, effect of smoking and glutethimide, 80–1615
- caffeine pharmacokinetics in smokers vs. nonsmokers, 80–1099
- clomipramine in smoking vs. nonsmoking depressed patients, 80–1368
- diazepam bioavailability in smokers vs. nonsmokers, 80–1375
- diazepam, effect of smoking and aging on kinetics, 80–1108
- lidocaine in lungs in smokers vs. nonsmokers, 80–0736
- pharmaceutical label for effect of smoking and, 80–1582
- phenytoin, effect of smoking and alcohol consumption in epileptics, 80–1342
- smoking and alcohol consumption and, 80–1581
- theophylline, effect of allopurinol in smokers vs. nonsmokers, 80–0263
- theophylline, effect of smoking and aging, 80–1617
- theophylline, effect of smoking and other variables, 80–0268
- theophylline, in smokers vs. nonsmokers, 80–0055

Duodenal ulcer *See* **ULCER, PEPTIC**

DUST
- benzo(*a*)pyrene and silicon dioxide in respiratory tract neoplasm induction in hamsters, 80–1659
- effect of cigarette smoke inhalation on free lung cells in guinea pigs, 80–1380
- effect of occuptional exposure on lung function, 80–1682
- effect of smoke inhalation on lungs in rats, 80–0730
- effect of smoking in grain elevator and sawmill workers, 80–1167
- effect of smoking on bronchitis incidence in industrial workers, 80–0341
- effect of tobacco processing on aroma and flavor, 80–1546
- effect on antitrypsin levels in smoking vs. nonsmoking vs. ex-smoking workers, 80–1410
- effect on byssinosis incidence in textile workers, 80–0771
- effect on lung function and respiratory symptoms in sisal workers, 80–0336
- effect on lung neoplasm incidence in smoking vs. nonsmoking nickel workers, 80–0305
- hyperplasia and metaplasia in respiratory tract of smoking vs. nonsmoking cotton textile workers, 80–1692
- interactive effect of smoking with occupational exposure, 80–0108

EARS
- (*See also* **HEARING**)
- effect of auricular therapy on cessation of smoking in otorhinolaryngology patients, 80–1253

ECONOMICS
- American Automatic Merchandiser survey of cigarette vending machine sales, 80–1267
- cigar, cigarette, and tobacco exports in the United States, 80–1276
- cigar consumption, production, and exports in the Netherlands, 80–1269
- cigar production and sales in Holland, 80–1268
- cigarette sales in the United States, 80–1028
- cigarette sales in West Germany, 80–0446
- effect of advertising restrictions on marketing techniques, 80–1066
- effect of cigarette taxation on revenues in Kentucky, 80–1032
- effect of filter additives on manufacturing and production practices, 80–1017
- effect of government programs on tobacco industry benefit distribution, price stability, and production, 80–1021
- effect of mechanization on tobacco industry workers in the United States, 80–1550
- effect of price and quality on tobacco exports in the United States, 80–1270
- effect of price support termination on tobacco industry, 80–1022
- effect of tariff rates on tobacco exports in the United States, 80–1549
- effect of tobacco industry, 80–1027
- effect of tobacco industry diversification on nontobacco income, 80–0447
- effect of tobacco industry in Kentucky, 80–1019, 80–1024
- effect of tobacco industry in the United States, 80–1023, 80–1034, 80–1282
- effect of tobacco industry on population in the United States, 80–1020
- exports of Burley tobacco in the United States, 80–1033
- factors affecting marketing of tobacco, 80–1551
- health insurance incentives for reduction of coronary disease, 80–1043
- impact of tobacco price support program in the United States, 80–1040
- international tobacco industry oligopoly, 80–1548

linear multiple regression model for cigarette consumption in Switzerland, 80–1825
low-tar cigarette sales in the United States, 80–1555
Maxwell international estimates for cigarette brand sales, 80–1552
1979 Maxwell Report on cigar sales, 80–0843
production quotas for flue-cured tobacco in the United States, 80–1035
proposals for apportioning smoking-related health costs to smokers, 80–0452
recommendations for tobacco subsidies and taxation in Europe, 80–1030
role of Tobacco Board in India, 80–0842, 80–0844
tobacco health care costs vs. tobacco revenues in Europe, 80–0857, 80–1030
tobacco industry in Great Britain, 80–1301
tobacco industry in Italy, 80–1556
tobacco industry in Pakistan, 80–1824
tobacco industry in Thailand, 80–0444
tobacco industry in the Dominican Republic, 80–1037
tobacco industry in Turkey, 80–0178
tobacco industry policy for the 1970's in the United States, 80–1061, 80–1063
tobacco leaf exports in the United States, 80–1828
tobacco marketing in Third World countries, 80–1843
tobacco product sales in health-related businesses, 80–1547
tobacco production and sales in the United States, 80–1829
tobacco production units in the United States, 80–1025
trends in tobacco consumption and production in the United States, 80–1029
worldwide funding for health education as preventive medicine for cardiovascular disease, 80–1044
worldwide tobacco expenditures and taxation, 80–1026
worldwide trends in cigarette sales, 80–1271

ELECTROCARDIOGRAM
in arrhythmia diagnosis, 80–1438
effect of blood glucose levels on abnormalities in smokers vs. nonsmokers vs. ex-smokers, 80–1445
effect of blood sugar and other factors on abnormalities, 80–1713, 80–1729, 80–1730, 80–1731
effect of carbon monoxide in smokers vs. nonsmokers, 80–1198
effect of nicotine in rats, 80–0734
effect of smoking and blood sugar on abnormalities, 80–1200
effect of smoking and glucose tolerance, 80–0962
effect of smoking in heavy- vs. light- vs. ex-smokers, 80–0785
effect of smoking in smokers vs. nonsmokers, 80–0784
hyperglycemia in smokers vs. nonsmokers vs. ex-smokers and abnormalities, 80–1216
smokers vs. nonsmokers, 80–0784
smokers vs. nonsmokers vs. ex-smokers in electronics factory, 80–0963
smoking habit and abnormalities, 80–1196

ELECTROENCEPHALOGRAM
alpha activity in smokers vs. nonsmokers vs. abstainers, 80–1632
effect of carbon monoxide in nonsmokers, 80–1625
effect of nicotine and heredity in mice, 80–1654
effect of nicotine content in cigarettes, 80–0255
effect of nicotine on contingent negative variation, 80–1610
effect of recording on smoking characteristics, 80–1500
effect of smoking and alcohol consumption on alpha correlates, 80–1633
effect of smoking on contingent negative variation, 80–1610
effect of visual stimulation in smokers vs. nonsmokers vs. abstainers, 80–1632

EMPHYSEMA
(*See also* **CHRONIC BRONCHOPULMONARY DISEASE; OBSTRUCTIVE AIRWAY DISEASES)**
antitrypsin deficiency and, 80–0944
antitrypsin deficiency in smoking vs. nonsmoking patients, 80–0325
cigarette smoke in etiology of, 80–0053
effect of smoking and uranium ore dust inhalation on incidence in animals, 80–0284
lung morphology in smoking vs. nonsmoking cotton industry workers, 80–0948
prevalence in smoking vs. nonsmoking cotton textile workers, 80–1692
smoking and lung function and, 80–0097
smoking in etiology of, 80–0939

ENVIRONMENTAL POLLUTION
in cardiovascular disease etiology, 80–0978
health effects, 80–1579

ENZYME ACTIVITY
acetylaminofluorene hydroxylase, aryl hydrocarbon hydroxylase, and nicotine oxidase in liver in animals, 80–1363
angiotensin I convertase and lysozyme in pulmonary alveolar macrophages in smokers vs. nonsmokers, 80–1174
antielastase activity of antitrypsin in lungs in smokers vs. nonsmokers, 80–0327
aryl hydrocarbon hydroxylase in liver in smokers vs. nonsmokers, 80–0712
aryl hydrocarbon hydroxylase in smokers vs. nonsmokers vs. lung neoplasm patients, 80–1675

- effect of antioxidants and steroids on aryl hydrocarbon hydroxylase in rats, 80–1377
- effect of areca chewing on amylase in saliva, 80–1133
- effect of benzo(*a*)pyrene on aryl hydrocarbon and prolyl hydroxylase in lungs in rats, 80–1627
- effect of benzo(*a*)pyrene 4, 5-oxide on glutathione transferase in rats, 80–0912
- effect of cigarette smoke condensate in liver in rats, 80–1107
- effect of enflurane and halothane in smokers vs. nonsmokers, 80–1106
- effect of insecticides on catalase and peroxidase in tobacco, 80–1068
- effect of maternal smoking on aryl hydrocarbon hydroxylase in placenta, 80–1225
- effect of methylcholanthrene on aryl hydrocarbon hydroxylase in lungs in rats, 80–0912
- effect of methylcholanthrene on epoxide hydrase in lungs in rats, 80–1097
- effect of nicotine and carbon monoxide on alanine and aspartate aminotranferases in rats, 80–0879
- effect of nicotine in epididymis and testes in rats, 80–1340
- effect of nicotine on adenosine triphosphatase in kidneys in rabbits, 80–0724
- effect of nicotine on tyrosine hydroxylase in adrenals and ganglia in rats, 80–1386
- effect of smoke inhalation in aorta, heart, and lungs in rats, 80–1350
- effect of smoke inhalation in kidneys and lungs in rats, 80–1107
- effect of smoke inhalation in pulmonary alveolar macrophages in rats, 80–0041
- effect of smoke inhalation on α_1-antitrypsin in lung in rats, 80–0266
- effect of smoke inhalation on aryl hydrocarbon hydroxylase in kidney and lungs in rats, 80–0912
- effect of smoke inhalation on epoxide hydrase in liver and lungs in rats, 80–1097
- effect of smoking and alcohol consumption on phosphatase in blood, 80–1488
- effect of smoking on δ-aminolevulinic acid dehydratase, 80–0749
- effect of smoking on α_1-antitrypsin in blood, 80–0266
- effect of smoking on myeloperoxidase and alkaline phosphatase, 80–0258
- effect of tobacco-betel chewing on salivary amylase in saliva, 80–1133
- effect of tobacco extracts in rats, 80–1127
- effect on alkaloid content in tobacco varieties, 80–1381
- elastase in emphysema etiology, 80–0053
- elastase in pulmonary alveolar macrophages, 80–0047
- elastase in pulmonary alveolar macrophages in smokers vs. nonsmokers, 80–1174
- elastase, lactate dehydrogenase, and lysozyme in pulmonary alveolar macrophages in smokers vs. nonsmokers, 80–1112
- epoxide hydratase and glutathione *S*-transferase in lungs in lung cancer patients, 80–0888
- glycosidase and protease in pulmonary alveolar macrophages of smokers vs. nonsmokers 80–1655
- glycosidase in bronchoalveolar lavage fluid in smokers vs. nonsmokers, 80–1111
- iminium oxidase in nicotine metabolism, 80–1612
- in lungs in lung neoplasm patients, 80–1125
- pepsin I secretion in smoking vs. nonsmoking peptic ulcer patients, 80–0993

ENZYMES
- acetylaminofluorene hydroxylase, 80–1363
- acid phosphatase, 80–1340, 80–1350
- adenosine triphosphatase, 80–0724
- alanine and aspartate aminotransferases, 80–0879
- alkaline phosphatase, 80–0258, 80–1340
- δ-aminolevulinic acid dehydratase, 80–0749
- amylase, 80–0558, 80–1133
- amyloglucosidase, 80–0558
- angiotensin I convertase, 80–1174
- aniline hydroxylase, 80–1127
- aryl hydrocarbon hydroxylase, 80–0712, 80–0742, 80–1127, 80–1627
- catalase, 80–0041, 80–0530, 80–0531, 80–1068
- elastase, 80–0047, 80–0053, 80–0274, 80–0327, 80–1112, 80–1174
- epoxide hydratase, 80–0888, 80–1097, 80–1107, 80–1125
- AS-esterase, 80–1350
- β-glucuronidase, 80–1127
- glutamic oxalacetic transaminase, 80–1350
- glutathione peroxidase, 80–0041
- glutathione reductase, 80–0041
- glutathione *S*-transferase, 80–0888, 80–0912, 80–1107, 80–1125
- glycosidase, 80–1111, 80–1655
- iminium oxidase, 80–1612
- lactate dehydrogenase, 80–1112, 80–1350
- lysozyme, 80–1112, 80–1174
- monooxygenase, 80–1125
- muramidase, 80–0667
- myeloperoxidase, 80–0258
- neutrophil elastase, 80–0333
- nicotine oxidase, 80–1363
- pepsin, 80–0993
- peroxidase, 80–0041, 80–1068
- phosphatase, 80–1488
- prolyl hydroxylase, 80–1627
- protease, 80–1655
- α_1-proteinase, 80–0053
- succinic dehydrogenase, 80–1340, 80–1350
- superoxide dismutase, 80–0041
- tyrosine hydroxylase, 80–1386

Epinephrine *See* **CATECHOLAMINES**

ERYTHROCYTES
(*See also* **BLOOD; HEMATOCRIT; HEMOGLOBIN)**
cell count and volume in smokers vs. nonsmokers, 80–0971
effect of carbon monoxide on cell count in smoking vs. nonsmoking submarine patrolmen, 80–1663
effect of cessation of smoking and exercise on cell count, 80–1708
effect of smoke inhalation in baboons, 80–1649
effect of smoking and alcohol consumption on mean cell volume, 80–0961
effect of smoking and other coronary disease risk factors, 80–1449
effect of smoking on deformability, 80–1344
smoking, propanolol, and sedimentation rate, 80–1382

ESOPHAGEAL NEOPLASMS
benzo(*a*)pyrene and nitrosamine metabolism in esophageal cells of patients and controls, 80–0265
induced by nitrosonornicotine in rats, 80–0024
smoking and alcohol consumption and, 80–1153

ESOPHAGUS
(*See also* **GASTROINTESTINAL SYSTEM)**
DNA binding of benzo(*a*)pyrene and nitrosamines, 80–0265
effect of nicotine on peristalsis in opossums, 80–1100

EX-SMOKERS
age at onset of menopause, 80–1230
antitrypsin levels in dust-exposed workers, 80–1410
asbestos-related lesions in dockyard workers in Great Britain, 80–0107
attitudes and self-regulatory behavior, 80–0152
attitudes toward smoking during social interaction, 80–0404
attributions about smoking, 80–0147
bladder neoplasm risk, 80–1152
blood pressure in myocardial infarct patients, 80–0965
bronchitis in fertilizer plant workers, 80–0332
bronchitis incidence in copper smelter workers, 80–1686
bronchoalveolar cell count in pulmonary fibrosis patients, 80–1176
bronchoalveolar lavage cell count in cryptogenic fibrosing alveolitis patients, 80–1415
chest radiograph and lung function in dockyard workers, 80–1695
coronary disease, 80–1723
coronary disease and hyperglycemia in Japan, 80–1216
coronary disease morbidity and mortality, 80–1717
determination of alpha particle activity in lungs, 80–1379
distribution of polonium-210 from cigarette smoke in lungs, 80–0257
effect of blood glucose levels on electrocardiogram abnormalities, 80–1445
effect of mitogens on lymphocyte transformation in asbestos workers, 80–0716
effect of practolol on prognosis in myocardial infarct patients, 80–0965
effect of smoking on lung function, 80–1428
effect of sulfur dioxide on lung function in copper smelter workers, 80–1686
electrocardiogram in electronics workers, 80–0963
health practices and mortality rates, 80–1392
locus of control scale, 80–0157
lung function, 80–0335, 80–0949, 80–1170, 80–1177, 80–1181, 80–1183, 80–1428
lung function and antitrypsin deficiency, 80–1417
lung function and respiratory symptoms in welders, 80–1681
lung function in α_1-antitrypsin deficient patients, 80–0095
lung function in beryllium workers, 80–1698
lung function in bronchitics, 80–1422
lung function in fertilizer plant workers, 80–0332
lung function in obstructive airway disease patients, 80–0322
lung neoplasm risk, 80–0925
mesothelioma and pulmonary fibrosis in dockyard workers, 80–1419
mortality rates in asbestos workers, 80–1394
mortality ratios in the United States, 80–1395
motivation, 80–1170
relative risk for intermittent claudication in hypertensives, 80–0368
respiratory symptoms and sputum cytology in cosmetologists, 80–0775
self-control, 80–0156
simulated cigarette and holder for reduction of passive smoking, 80–1830
social psychology theories of behavior, 80–0148

EXERCISE
(*See also* **PHYSICAL ACTIVITY)**
coronary disease mortality in marathoners, 80–0122
effect of carbon monoxide on work capacity of nonsmokers at high altitude, 80–0070
effect of cessation of smoking in bronchitics, 80–1427
effect of cessation of smoking on cardiorespiratory functions, 80–1708
effect of passive smoking on lung function in nonsmokers, 80–1426
effect of smoking on prognosis in myocardial infarct patients, 80–0968
effect on coronary disease incidence in smokers vs. nonsmokers, 80–0972

effect on fibrinolysis, 80–1466
effect on heart rate in smokers vs. nonsmokers, 80–1195
effect on high density lipoprotein, cholesterol, and insulin levels in coronary disease patients, 80–0375
effect on lung function in cigarette vs. marihuana smokers, 80–1641

EYE DISEASES
diabetic retinopathy in smokers vs. nonsmokers, 80–1232, 80–1490

EYES
(*See also* **VISION**)
effect of carbon monoxide on retina in cats, 80–0894
effect of cigarette smoke on conjunctiva in hamsters, 80–0882
effect of passive smoking, 80–1748
effect of passive smoking on blink rate and irritation, 80–0809, 80–1388
effect of smoke inhalation, carbon monoxide, and nicotine on retina in cats, 80–0725
effect of smoking on movement, 80–1496

FATTY ACIDS
(*See also* **TRIGLYCERIDES**)
determination in tobacco by gas-liquid chromatography, 80–0016
determination in tobacco leaf, 80–1084
determination of valeric acids in cigarette smoke of tobacco varieties, 80–1598
distribution of palmitic acid in mainstream vs. sidestream smoke, 80–1332
effect of alcohol and ester derivatives on sucker growth, 80–1070
effect of ozonization on levels in tobacco, 80–1823
effect of smoking and pindolol on levels in blood, 80–1720
in green vs. pale yellow tobacco varieties, 80–0709
in smoke condensate in tobacco varieties, 80–0876

FEAR
in cessation of smoking, 80–0438
effect on attitudes and beliefs in smokers vs. nonsmokers, 80–0810

FERTILITY
effect of nicotine and alcohol consumption in rats, 80–1609
sperm density and motility in smoking vs. nonsmoking infertile patients, 80–1480

FETAL GROWTH
(*See also* **BIRTH WEIGHT**)
effect of cadmium in mice, 80–0069
effect of maternal nicotine and alcohol consumption, 80–1609
effect of maternal smoking and alcohol consumption, 80–1740
effect of maternal smoking and body weight, 80–0130
effect of maternal smoking on dystrophy incidence, 80–1470
effect of nicotine in rats, 80–0049

FETAL MORTALITY
(*See also* **INFANT MORTALITY**)
effect of maternal smoking, 80–0133, 80–1222, 80–1474
effect of maternal smoking and alcohol consumption, 80–0133
effect of maternal smoking and socioeconomic status, 80–1738
effect of nicotine and alcohol consumption in rats, 80–1609
effect of nicotine on stillbirth incidence in rats, 80–0890
maternal smoking and, 80–0984

FETUS
(*See also* **NEONATE; PREGNANCY**)
effect of carbon monoxide on development in mice and rabbits, 80–0907
effect of maternal smoking, 80–0384, 80–0799, 80–0985, 80–1222, 80–1734
effect of maternal smoking and alcohol consumption, 80–1740
effect of maternal smoking on blood vessels in umbilical cord, 80–1468
effect of maternal smoking on breathing movements, 80–0135, 80–1223
effect of maternal smoking on cardiovascular system, 80–1732
effect of maternal smoking on disorder incidence, 80–1476
effect of maternal smoking on movement, 80–0987
effect of maternal smoking on reactivity, 80–0986
effect of nicotine on acidosis and hypoxia incidence in monkeys, 80–1384
effect of parental smoking, 80–1471
effect of smoke inhalation in rats, 80–0799
effect of transplacental *N*-nitrosodiethylamine on metabolism in trachea in hamsters, 80–1134
estrogen:creatinine ratio in prediction of complications in smokers vs. nonsmokers, 80–1737
maternal smoking, alcohol consumption, and fetal alcohol syndrome, 80–0797

FIBRINOLYSIS
(*See also* **BLOOD COAGULATION**)
effect of coronary disease intervention program, 80–1446
effect of smoking and exercise, 80–1466
effect of smoking and pindolol, 80–1720

FILTERS
- activated carbon with sucrose and menthol delivery, 80–0537
- alumina and silica, 80–0505
- annular design, 80–0491, 80–0492
- annular design with mouthpiece, 80–1822
- baffle type cigarette mouthpiece, 80–0539
- 1, 4-butanediol diacetate as plasticizer for cellulose acetate fiber rods, 80–0533
- carboxymethyl cellulose, 80–0511
- cartridge for pipes, 80–0679
- cellulose acetate for reduced nicotine and tar delivery in cigarette smoke, 80–0538
- cellulose and coniferous bark particles, 80–0543
- chitin-modified tobacco in cigarette or pipe holders, 80–0631
- cigarette holder, 80–0673
- colloidal alumina and silica for particulate retention, 80–0503
- concentric fibrous cylinders, 80–0541, 80–0542
- cylindrical cup-like and H-shaped design, 80–0517
- design for unfiltered smoke dilution with air, 80–0522
- determination of humic acid and dyes in wrapper, 80–0706
- determination of porosity by permeability measurements, 80–0442
- effect of active charcoal on carboxyhemoglobin levels, 80–1765
- effect of active charcoal on smoking characteristics, 80–1765
- effect of additives on constituent levels, 80–1017
- effect of composition on cigarette smoke constituents, 80–0839
- effect of polystyrene resins on aldehyde and hydrogen cyanide levels in cigarette smoke, 80–0841
- effect of ventilation on cigarette smoke constituent levels, 80–1016
- effect on carbon monoxide and carbon dioxide diffusion from cigarettes, 80–0019
- effect on chemical composition of cigarette smoke, 80–0029
- effect on health, 80–1292
- effect on nitrosamine retention in respiratory system, 80–0718
- effect on phenol content in cigarette smoke, 80–1079
- effect on pyridine content of mainstream and sidestream cigarette smoke, 80–0015
- electric field design, 80–0536
- fiber sheet as cylindrical casing, 80–0501
- film-forming substances for porous wrappers, 80–0518
- grooved design, 80–0490
- hydrogen peroxide for aldehyde reduction in cigarette smoke, 80–1817
- ion-exchange resin, 80–0513
- layered design for selective filtration, 80–0487, 80–0488, 80–0489
- manufacture by split rod technique, 80–0535
- metal—amine complex with polyalkylene oxide, 80–0514
- metal oxides and oxyhydrates, 80–1807
- methylated silica—water, 80–0504, 80–0506
- microporous styrene polymers, 80–0529
- molded plastic channels for directional change of smoke path, 80–0485, 80–0486
- negatively charged hydrophobic fibers, 80–0507
- organic oils and powders, 80–0510
- permeable polymer membrane, 80–0483
- polymer-coated carbon particles, 80–0495
- polyolefin fibers, 80–1821
- protein granules, 80–0523
- 2-pyrrolidone polymer, 80–0515
- resins and organic and metal compounds, 80–0526
- silicic acid—polystyrene for amine and nitrosamine removal from tobacco smoke, 80–1813
- smoke flow path restriction and expansion in cigarette holders, 80–0508
- thermoplastic polymers, 80–0493
- transparent coating for visibility of efficiency, 80–0527
- two-stage disc design, 80–0540
- upstream and downstream chambers in cigarette holder, 80–0482
- valve for controlling cigarette smoke delivery, 80–0524
- ventilated wrap for delivery of volatile flavoring compounds, 80–0525
- wool fibers for cigars, cigarettes, or pipes, 80–0496

Forced expiratory volume tests *See* **RESPIRATORY FUNCTION TESTS**

Forced vital capacity tests *See* **RESPIRATORY FUNCTION TESTS**

FORMALDEHYDE
- effect of adrenergic blocking agents, ascorbic acid, and cysteine on toxicity in rats, 80–0909
- effect of transition metals on levels in tobacco smoke, 80–0566
- effect on tracheobronchial epithelium in hamsters, 80–0281
- metabolism in lung cells, 80–0904

2-FURALDEHYDE
- in lamina vs. midrib cigarette smoke condensates, 80–0027
- in smoke condensate in tobacco varieties, 80–0876

GALLBLADDER
- effect of smoking on prognosis for hypoxemia in cholecystectomy patients, 80–1179

GANGLIA
effect of nicotine on tyrosine hydroxylase activity in rats, 80–1386

GAS PHASE, CIGARETTE SMOKE
determination of constituents in Canadian brands, 80–1603
determination of hydrogen sulfide content by automated colorimetry, 80–0691
determination of trace metals, 80–0034
effect of air dilution on components, 80–0234
effect of cigarette periphery and rod on smokestream formation, 80–0028
effect of passive smoking on eyes, nose, and throat, 80–1748
effect on benzo(*a*)pyrene metabolism in lungs in rabbits, 80–1367
effect on eyes, nose, and throat in passive smokers, 80–0809
effect on heart muscle in rats, 80–1640
effect on mitotic recombination and respiratory mutation rate in yeast, 80–1628
effect on sister chromatid exchange rate in lymphocytes, 80–1628
nonvolatile and semivolatile components, 80–0232

Gastric acidity *See* **GASTRITIS**

GASTRIC SECRETION
effect of passive smoking, 80–1226
effect of smoking, 80–1226

Gastric ulcer *See* **ULCER, PEPTIC**

GASTRITIS
in smoking vs. nonsmoking peptic ulcer patients, 80–0806

GASTROINTESTINAL DISEASES
(*See also* **GASTRITIS; LIVER CIRRHOSIS; ULCER, PEPTIC)**
lung function in smoking vs. nonsmoking gastroesophageal reflux patients, 80–0780
smoking and biliary reflux, 80–1228
in smoking vs. nonsmoking chemical workers, 80–1227

GASTROINTESTINAL SYSTEM
(*See also* **INTESTINES)**
deposition and clearance of cigarette smoke in rats, 80–1648
effect of areca in rats, 80–0902
effect of nicotine on ingestion in rats, 80–1093

GENETICS
(*See also* **HEREDITY)**
in control of constituents in tobacco, 80–0196
in control of constituents in tobacco leaf and smoke, 80–0867
effect of breeding on nicotine content in tobacco, 80–0194

GLYCERIN
(*See also* **HUMECTANTS; TOBACCO ADDITIVES)**
in smoke condensate in tobacco varieties, 80–0876

GLYCOLS
(*See also* **HUMECTANTS; TOBACCO ADDITIVES)**
effect on moisture content of tobacco, 80–1811

GOBLET CELLS
(*See also* **CELLS, EPITHELIAL)**
metaplasia in bronchi in smoking vs. nonsmoking cotton textile workers, 80–1692

GROUP THERAPY
in cessation of smoking, 80–0831, 80–1011

GYNECOLOGIC DISORDERS
effect of maternal smoking on placenta, 80–1222

Gynecologic neoplasms *See* **UTERINE NEOPLASMS**

HEALTH EDUCATION
(*See also* **ANTISMOKING CAMPAIGNS; PREVENTION OF SMOKING; SMOKING CONTROL PROGRAMS)**
antismoking campaigns as preventive medicine for neoplasms in adults, 80–1513
in antismoking campaigns in adolescents, 80–0468
antismoking campaigns in cancer screening program, 80–0301
antismoking campaigns in cardiovascular diseases prevention program, 80–0120
antismoking campaigns in East Germany and, 80–1529
antismoking campaigns in Europe and, 80–0857
antismoking campaigns in France and, 80–1769
antismoking campaigns in hypertension control program for college students, 80–1197
antismoking campaigns in hypertensives, 80–0380
antismoking campaigns in schools in Japan, 80–1003
attitudes of teachers toward cancer in Great Britain, 80–1756
bibliography of antismoking materials for use in schools, 80–1303
cardiovascular disease risk factor reduction program in Israel, 80–1749
cessation of smoking counseling in chronic obstructive pulmonary disease patients, 80–1191
in coronary disease intervention program in Belgium, 80–0969, 80–1447

effect of antismoking campaigns on mortality in myocardial infarct patients, 80–0356
effect of coronary risk factor intervention program on smoking habit, 80–1726
effect of School Health Curriculum Project in adolescents in the United States, 80–1510
effect of socioeconomic status on attitudes toward smoking in children, 80–1786
effect on onset of smoking in children, 80–0408
effect on smoking habit in adolescents in Switzerland, 80–1787
effect on smoking habit in the United States, 80–1511
evaluation of heart education program, 80–0998
Health Hazard Appraisal inventory, 80–1247
life skills training as preventive medicine for neoplasms, 80–1533
manual for education of chronic obstructive pulmonary disease patients, 80–1165
mass media program for lung diseases, 80–0161
in prevention of smoking, 80–0853
role of dentists, 80–1293
role of dentists and hygienists, 80–1844
role of gynecologists and obstetricians in antismoking programs, 80–1569
smoking control programs in schools in Australia, 80–1526
sociocultural factors in tobacco and alcohol programs, 80–1521
teachers' handbook for drug abuse education, 80–1517
worldwide funding for prevention of cardiovascular disease, 80–1044

HEALTH PRACTICES
abstinence from smoking and altitude acclimatization, 80–1575
effect of annual health checkups on morbidity and mortality, 80–0074
effect of smoking on health status score, 80–1309
lifestyle intervention as preventive medicine for smoking-related disease, 80–1578
mortality rates in smokers vs. nonsmokers vs. exsmokers and, 80–1392
multiphasic screening in preventive medicine program, 80–0428
role in disease prevention, 80–1051
role of health insurance companies in prevention of coronary disease, 80–1043
seat-belt use in smokers vs. nonsmokers, 80–1761
smoking habit and, 80–0155, 80–0158
in the United States, 80–1052
use of cessation of smoking aids in the United States, 80–0815

HEARING
(*See also* **EARS**)
effect of carbon monoxide on auditory vigilance, 80–1131
effect of carboxyhemoglobin levels on discrimination, 80–0162
effect of smoking and occuptional exposure on impairment incidence, 80–0990

HEART
(*See also* **CARDIOVASCULAR SYSTEM**)
effect of nicotine on cyclic AMP levels in guinea pigs, 80–1136
effect of nicotine on cyclic nucleotide levels in rabbits, 80–0911
effect of nicotine on muscle in rats, 80–1640
effect of nicotine on myocardial contractility in cats, 80–1436
effect of smoke inhalation on enzyme activity in rats, 80–1350
effect of smoking and aging on echocardiographic examination, 80–1433
effect of tobacco smoke on muscle in rats, 80–1640

HEART DISEASES
(*See also* **ARRHYTHMIA; CARDIOVASCULAR DISEASES; CORONARY DISEASE; MYOCARDIAL INFARCT**)
evaluation of health education program, 80–0998
smoking habit in cardiomyopathy patients, 80–1451
smoking habit in hypertensives and, 80–0374

HEART FUNCTION
(*See also* **ARRHYTHMIA; BLOOD CIRCULATION**)
effect of carbon monoxide in nonsmokers, 80–1210
effect of coronary disease risk factor intervention program, 80–1703
effect of nicotine in dogs, 80–1351
effect of nicotine in guinea pigs, 80–1136
effect of smoking habit on ventricular premature beat incidence, 80–1212
effect of smoking in coronary disease patients, 80–1436

HEART RATE
effect of carbon monoxide and non-nicotine cigarettes in angina patients, 80–0782
effect of carbon monoxide in nonsmokers, 80–1625
effect of carbon monoxide in nonsmokers during exercise at high altitude, 80–0070
effect of cessation of smoking and exercise, 80–1708
effect of cigarette length reduction, 80–1751
effect of exercise in smokers vs. nonsmokers, 80–1195
effect of nicotine, 80–0064
effect of nicotine and heredity in mice, 80–1654
effect of nicotine content in cigarettes, 80–1138, 80–1206
effect of nicotine in cats, 80–1376
effect of nicotine in dogs, 80–1351

effect of nicotine in pregnant rats, 80–1338
effect of nicotine in rabbits, 80–0911
effect of nicotine in rats, 80–0734
effect of passive smoking, 80–1388
effect of pipe smoking, 80–0412
effect of rapid smoking, 80–0786
effect of smoking, 80–0149
effect of smoking, aging, and workload, 80–1716
effect of smoking and beta adrenoceptor blocking agents, 80–1465
effect of smoking and stress, 80–1784
effect of smoking in heavy- vs. light- vs. ex-smokers, 80–0785
effect of smoking in smokers vs. nonsmokers, 80–0784
smokers vs. nonsmokers, 80–0784

HEMATOCRIT
(*See also* **BLOOD; ERYTHROCYTES)**
effect of age on levels in smokers vs. nonsmokers, 80–0788
effect of carbon monoxide in smoking vs. nonsmoking submarine patrolmen, 80–1663
effect of cessation of smoking and exercise, 80–1708
effect of maternal smoking in neonate, 80–0385
effect of smoke inhalation in baboons, 80–1649
effect of smoking, 80–0973
smokers vs. nonsmokers, 80–0971

HEMODYNAMICS
(*See also* **BLOOD CIRCULATION)**
effect of smoking in coronary disease patients, 80–1436

HEMOGLOBIN
(*See also* **ERYTHROCYTES)**
affinity for oxygen in smokers vs. nonsmokers, 80–0731

HEMOGLOBIN LEVELS
(*See also* **CARBOXYHEMOGLOBIN LEVELS)**
effect of carbon monoxide in smoking vs. nonsmoking submarine patrolmen, 80–1663
effect of cessation of smoking and exercise, 80–1708
effect of maternal smoking during pregnancy, 80–1619
smokers vs. nonsmokers, 80–0971

HEREDITY
(*See also* **GENETICS)**
in coronary heart disease etiology, 80–0347
effect of gas phase cigarette smoke on mitotic recombination rate in yeast, 80–1628
effect of nicotine on behavior and electroencephalogram in mice, 80–1654
effect of smoke inhalation and genotype on behavior in mice, 80–1334
effect of smoke inhalation and genotype on nicotine levels in mice, 80–1334
effect on bronchogenic carcinoma incidence, 80–0921
effect on smoking behavior, 80–1064
in etiology of smoking-related disease, 80–1064
lipoprotein levels in smoking vs. nonsmoking myocardial infarct patients and relatives, 80–0371
in neoplasm etiology, 80–0921
paternal smoking and perinatal mortality, 80–0714
relative risk for coronary disease in smokers vs. nonsmokers and, 80–0126
as risk factor for ischemic heart disease and myocardial infarct, 80–0979

HISTAMINES
bronchial threshold in smokers vs. nonsmokers, 80–0102
effect on contractility of bronchi, bronchioles, and trachea, 80–0072

HORMONE LEVELS
calcitonin in smokers vs. nonsmokers vs. lung neoplasm patients, 80–1162
effect of exercise on insulin in coronary disease patients, 80–0375
effect of high- vs. low-nicotine cigarettes on vasopressin, 80–1653
effect of nicotine and carbon monoxide on corticosterone in rats, 80–0879
effect of smoking on neurophysin in blood, 80–0259
vasopressin in diabetes and inappropriate antidiuretic hormone secretion, 80–0140

HORMONES
effect of nicotine on growth hormone and prolactin levels in rats, 80–1365
effect of nicotine on prolactin release in rats, 80–0275
effect of nicotine on prostaglandins in lungs in rats, 80–0711
effect of smoke inhalation on calcitonin in rats, 80–0805
insulin requirements in smoking vs. nonsmoking diabetics, 80–1232
smoking and vasoconstrictor prostaglandins in coronary artery spasm etiology, 80–0967

HUMECTANTS
(*See also* **GLYCERIN; GLYCOLS)**
glycols, 80–1811

Hydrogen cyanide *See* **CYANIDES**

HYPERCHOLESTEREMIA
(*See also* **BLOOD CHOLESTEROL)**
effect of smoking, 80–0793

HYPERLIPIDEMIA
(*See also* **BLOOD LIPIDS)**

smoking in oral contraceptive users and, 80–1199

HYPERTENSION
(*See also* **BLOOD PRESSURE)**
antismoking campaigns in health education program for patients, 80–0380
effect of smoking on cardiovascular mortality risk in hypertensives, 80–1437
effect of smoking on mortality risk in patients, 80–1437
health education program for college students, 80–1197
interactive effect with smoking and oral contraceptives on myocardial infarct risk, 80–1215
renal artery stenosis in smoking vs. nonsmoking patients, 80–0360
smokers vs. nonsmokers, 80–0128
smokers vs. nonsmokers in Finland, 80–1056
smoking and, 80–1202, 80–1463
smoking habit and alcohol consumption in physicians and, 80–0828
smoking habit and heart disease in patients, 80–0374

HYPNOSIS
in cessation of smoking, 80–0817, 80–1011, 80–1799
self-hypnosis in cessation of smoking, 80–1251

HYPOXEMIA
effect of smoking on prognosis in cholecystectomy patients, 80–1179

IMMUNE SYSTEM
(*See also* **ANTIBODIES; ANTIGEN—ANTIBODY REACTIONS)**
effect of cigarette smoke on secondary response in mice, 80–0270
effect of maternal smoking in umbilical cord blood, 80–0800
effect of mitogens on lymphocyte transformation in asbestos-exposed smokers vs. nonsmokers vs. ex-smokers, 80–0716
effect of nitrogen oxides in mice, 80–0892
effect of smoking, 80–0462
effect of tobacco allergy on thromboangiitis obliterans incidence, 80–0790
IgA and IgG levels in smokers vs. nonsmokers, 80–1631
immunoglobulin levels in smokers vs. nonsmokers, 80–1611
immunoglobulin levels in smoking vs. nonsmoking grain handlers vs. teachers, 80–1348

IMMUNITY
(*See also* **ANTIBODIES; ANTIGEN—ANTIBODY REACTIONS)**
effect of smoke inhalation on susceptibility to respiratory tract infections in mice, 80–1639

INDOLES
determination in cigarette smoke condensate, 80–0710

INFANT MORTALITY
(*See also* **FETAL MORTALITY; NEONATE)**
effect of maternal smoking, 80–0134
effect of maternal smoking and body weight, 80–0130
effect of maternal smoking and socioeconomic status, 80–1738
effect of maternal smoking on sudden infant death syndrome incidence, 80–1223
effect of parental smoking on neonate, 80–1224
effect of paternal smoking on neonate, 80–1224

Infant, newborn *See* **NEONATE**

Influenza *See* **RESPIRATORY TRACT INFECTIONS**

Inspiratory reserve volume measurements *See* **RESPIRATORY FUNCTION TESTS**

INTERMITTENT CLAUDICATION
(*See also* **VASCULAR DISEASES)**
effect of smoking on postoperative prognosis, 80–1217
incidence in smokers vs. nonsmokers vs. ex-smokers, 80–1723
relative risk in smoking vs. nonsmoking hypertensives, 80–0368

INTESTINAL NEOPLASMS
(*See also* **STOMACH NEOPLASMS)**
smoking and, 80–0927
smoking and colonic neoplasm mortality in Great Britain, 80–0294

INTESTINES
(*See also* **COLON; GASTROINTESTINAL SYSTEM)**
effect of tobacco extracts on enzyme activity in rats, 80–1127

ISCHEMIA
(*See also* **CARDIOVASCULAR DISEASES; CORONARY DISEASE; MYOCARDIAL INFARCT)**
effect of smoking on mortality rate, 80–1207

Ischemic heart disease *See* **CORONARY DISEASE**

ISOPRENOIDS
in lamina vs. midrib cigarette smoke condensates, 80–0027
in tobacco, 80–0871
in tobacco leaf, effect of ozone on lipid fraction, 80–1819

Keratoacanthoma *See* **SKIN NEOPLASMS**

KETONE CONTENT
- in cigarette smoke, effect of paper perforation, 80–1264
- determination of dihydro-γ-pyrones in Burley vs. Oriental vs. Virginia varieties, 80–1076
- in tobacco, effect of agricultural practices, 80–1074
- in tobacco, effect of processing, 80–1074
- in tobacco leaf, effect of curing, 80–0012

KETONES
- cycloaliphatic derivatives for tobacco aroma and flavor enhancement, 80–1256
- determination in Burley tobacco, 80–0694
- determination of aliphatic and aromatic ketones in cigarette smoke condensate, 80–1319
- effect of benzo-1, 2-pyrone derivatives on tobacco aroma and flavor, 80–0593
- effect of benzophenones on sucker growth, 80–0478
- effect of butanone derivatives on tobacco aroma and flavor, 80–0603
- effect of 2-butyl-3, 5, 5-trimethyl-2-cyclohexen-1, 4-dione on tobacco aroma and flavor, 80–0601
- effect of butyrolactone derivatives on tobacco flavor, 80–0581
- effect of cyclohexadiene derivatives on tobacco aroma and flavor, 80–0599
- effect of 1, 2-cyclohexanedione on tobacco aroma and flavor, 80–0627
- effect of epoxy-β-ionone on tobacco aroma and flavor, 80–0575
- effect of hydroxy cyclohexenone derivatives on tobacco aroma and flavor, 80–0600
- effect of 2-hydroxymethylenecyclohexanones on tobacco aroma and flavor, 80–0583
- effect of 3-isopropyl-cyclopent-2-en-1-one on tobacco aroma and flavor, 80–0576
- effect of lactone derivatives on tobacco aroma and flavor, 80–0602
- effect of 2-methyl-5-(α-methyl-α-hydroxyethyl)-cyclohex-2-en-1-one on tobacco aroma and flavor, 80–0571
- effect of 6-methyl-hepta-3, 5-dien-2-one on tobacco aroma and flavor, 80–0573
- effect of nonane-2, 5, 8-trione on tobacco aroma and flavor, 80–0574
- effect of octa-3, 5-dien-2-one on tobacco aroma and flavor, 80–0572
- effect of octahydrotetramethyl acetonaphthone isomers on tobacco aroma and flavor, 80–0589
- effect of tricyclene-9-butenone on tobacco aroma and flavor, 80–0621
- effect of tricyclic derivatives on tobacco aroma and flavor, 80–0612
- identification of solanascone in Burley variety, 80–0695
- 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone in neoplasm induction in rats, 80–0722

KIDNEYS
- cadmium levels in smokers vs. nonsmokers, 80–1105
- effect of nicotine on adenosine triphosphatase activity and electrolyte transport in rabbits, 80–0724
- effect of nicotine on electrolyte transport in rats, 80–0724
- effect of smoke inhalation on aryl hydrocarbon hydroxylase activity in rats, 80–0912
- effect of smoke inhalation on enzyme activity in rats, 80–1107
- function in smoking vs. nonsmoking cadmium workers, 80–0334
- proteinuria in diabetic smokers vs. nonsmokers, 80–1490

LABELING
- Canadian Pharmaceutical Association warning on smoking and drug metabolism, 80–1582
- for oral contraceptives, 80–0352
- recommendations for carbon monoxide content in cigarettes in Great Britain, 80–0013
- recommendations in Australia, 80–1849
- recommendations of Federal Trade Commission for cigarette packages, 80–0861
- recommendations of Federal Trade Commission for rotating health warnings, 80–1306
- role of Federal Trade Commission, 80–0854

LACTATION
(*See also* **BREAST FEEDING)**
- effect of maternal smoking, 80–1479

LARYNGEAL NEOPLASMS
(*See also* **RESPIRATORY TRACT NEOPLASMS)**
- effect of smoking and dental X-ray exposure on risk, 80–0086
- effect of smoking on mortality rates in asbestos workers, 80–1668
- induced by benzo(*a*)pyrene and alcohol in hamsters, 80–0910
- induced by smoke inhalation in hamsters, 80–0883, 80–1141
- mortality in asbestos workers, 80–0292
- smoking, alcohol consumption, and occupational exposure and 80–0919
- smoking and, 80–0295
- smoking and alcohol consumption in etiology of, 80–0091

LARYNX
(*See also* **RESPIRATORY SYSTEM)**
- effect of smoke inhalation and vitamin A deficiency in hamsters, 80–0901

LEAD-210
(*See also* **METALS; RADIATION)**
bibliography of tobacco smoke carcinogenicity, 80–1385

LEAD CONTENT
in blood and placenta in smokers vs. nonsmokers, 80–0386
in blood and urine in smoking vs. nonsmoking ore dressing plant workers, 80–1104
in blood, effect of smoking, 80–0749
in bronchial epithelium of smokers vs. nonsmokers, 80–0737
in cigarettes in New Zealand brands, 80–1595
determination in cigarette smoke condensate, 80–1597
determination in particulate matter and gas phase cigarette smoke, 80–0034
in German cigarettes, 80–0229
in hair in smokers vs. nonsmokers, 80–1339
in tobacco, effect of acids, 80–1541

LEARNING
(*See also* **COGNITION)**
effect of carbon monoxide, 80–1092
effect of pipe smoking, 80–0412
effect of smoking, 80–1777, 80–1781
effect of smoking and marihuana in males vs. females, 80–1768
smoking as contagious behavior, 80–1503

LEGISLATION
(*See also* **BILLS)**
antismoking campaign in France and, 80–1834
control of advertising and smoking in public places in France, 80–0455
economic impact of tobacco price support program in the United States, 80–1040
effect on smoking habit in Norway, 80–0450
flammability standards for cigarettes, 80–1842
Howard County Indoor Clean Air Act, 80–0180
nonsmokers' rights for smoke-free workplaces, 80–1041
nonsmokers' rights in the United States, 80–1039
recommendations for antismoking campaigns in Great Britain, 80–1287
recommendations for drug abuse prevention in Australia, 80–0467
recommendations of American Medical Association for restriction of smoking on airplanes, 80–1278
recommendations of the Hunter Committee on tobacco additives and substitutes, 80–0856
recommendations of the Quebec Advisory Council on the Environment for prevention of smoking in public, 80–0847
restriction of smoking in public places in the United States, 80–0845
role of Federal Trade Commission in cigarette labeling, 80–0854
tobacco industry counterorganization for restriction of smoking, 80–1557
Wisconsin Clean Indoor Air Bill, 80–0846

LEUKOCYTES
(*See also* **BLOOD)**
benzo(*a*)pyrene metabolism in lymphocytes, 80–1095
benzo(*a*)pyrene metabolism in lymphocytes and monocytes, 80–1371
effect of cigarette smoke condensate on sister chromatid exchange rates in smokers vs. nonsmokers, 80–0723
effect of cigarette smoke inhalation and dust in lungs in guinea pigs, 80–1380
effect of cigarette smoke on alveolar macrophage release of a chemotactic factor for polymorphonuclear cells, 80–1118
effect of cigarette smoke on phagocytosis, 80–1354
effect of gas phase cigarette smoke on sister chromatid exchange rate in lymphocytes, 80–1628
effect of maternal smoking on count in cord blood, 80–1733
effect of maternal smoking on lymphocytes in umbilical cord blood, 80–0800
effect of mitogens on lymphocyte transformation in asbestos-exposed smokers vs. nonsmokers vs. exsmokers, 80–0716
effect of smoke inhalation on lymphocyte count in baboons, 80–1649
effect of smoking and anesthetics on sister chromatid exchange rates in lymphocytes in operating room personnel, 80–1119
effect of smoking and coffee consumption on DNA synthesis, 80–0271
effect of smoking on cell count in patients with cryptogenic fibrosing alveolitis, 80–1415
effect of smoking on function, 80–0258
effect of smoking on polymorphonuclear leukocyte chemotaxis in saliva and serum in smokers vs. nonsmokers, 80–1635
mutagenesis of cigarette smoke constituents in lymphocytes, 80–0714
sister chromatid exchange rates in lymphocytes of smokers vs. nonsmokers, 80–1616
smokers vs. nonsmokers, 80–1364

LEUKOPLAKIA
(*See also* **MOUTH NEOPLASMS)**
areca and tobacco chewing in etiology of, 80–0394
preleukoplakia in smokers vs. nonsmokers vs. tobacco chewers in India, 80–0989
smoking and oral epithelial keratinization in patients, 80–1486
snuff-dipping in etiology of, 80–0390

LIP NEOPLASMS
(*See also* **MOUTH NEOPLASMS)**
smoking and, 80–0308

LIVER
acetylaminofluorene hydroxylase, aryl hydrocarbon hydroxylase, and nicotine oxidase activity in animals, 80–1363
aryl hydrocarbon hydroxylase activity in smokers vs. nonsmokers, 80–0712
aryl hydrocarbon hydroxylase induction by benzo(*a*)pyrene and methylcholanthrene, 80–0052
cadmium levels in smokers vs. nonsmokers, 80–1105
effect of alcohol consumption on carcinoembryonic antigen levels in smoking vs. nonsmoking patients, 80–1335
effect of cigarette smoke on drug metabolism in microsomes in rats, 80–1620
effect of smoke inhalation on aryl hydrocarbon hydroxylase activity in rats, 80–1097
effect of smoke inhalation on epoxide hydrase activity in rats, 80–1097
effect of tobacco extracts on enzyme activity in rats, 80–1127

LIVER CIRRHOSIS
(*See also* **GASTROINTESTINAL DISEASES)**
smoking habit and alcohol consumption in male vs. female patients, 80–1399

LIVER NEOPLASMS
effect of phenobarbital on induction by dimethylnitrosamine in mice, 80–0744
induced by *N*-nitrosonornicotine and 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone in rats, 80–0722
smoking habit and alcohol consumption in patients, 80–1399

LOBELINE
effect on cough reflex in dogs, 80–0914
herbal tea ingestion and, 80–0395

LONGEVITY
coronary disease risk factors and, 80–0349
effect of health education in the United States, 80–1511
effect of maternal nicotine administration in offspring in rats, 80–1369
effect of smoking characteristics and habit, 80–1393
smokers vs. nonsmokers, 80–0917, 80–1393

LUNG DISEASES
(*See also* **PNEUMOCONIOSIS; PULMONARY ALVEOLAR PROTEINOSIS; PULMONARY FIBROSIS; RESPIRATORY TRACT DISEASES)**
antitrypsin deficiency and, 80–0944
attitudes in smokers vs. nonsmokers, 80–0161, 80–0955
mass media program for health education, 80–0161
pulmonary alveolar macrophage metabolism in etiology of, 80–1115

LUNG FUNCTION
(*See also* **PULMONARY CLEARANCE; RESPIRATORY FUNCTION TESTS)**
antitrypsin deficiency and, 80–0944
in antitrypsin deficient patients, 80–0946
in arthritis patients, 80–0941
asymptomatic blacks vs. whites, 80–1425
carbon monoxide exchange ratio in smokers vs. nonsmokers, 80–1166
in chimney sweeps, 80–0951
closing capacity in smokers vs. nonsmokers, 80–1421
effect of aging in smokers vs. nonsmokers, 80–1684
effect of air pollution in smokers vs. nonsmokers, 80–0092, 80–0098
effect of air pollution in smoking vs. nonsmoking industrial workers, 80–0763
effect of antitrypsin deficiency in smokers vs. nonsmokers, 80–0342
effect of asthma and bronchitis in infancy, 80–1431
effect of carboxyhemoglobin levels on single breath diffusing capacity in nonsmokers, 80–1186
effect of cessation of smoking and exercise, 80–1708
effect of cigarette consumption in obstructive lung disease patients, 80–0957
effect of codeine and promethazine on response to carbon dioxide in smoking vs. nonsmoking males vs. females, 80–0947
effect of dust exposure in sisal workers, 80–0336
effect of exercise in cigarette vs. marihuana smokers, 80–1641
effect of inhaling, 80–1190
effect of ipratropium bromide in obstructive airway disease patients, 80–0339
effect of neutrophil elastase in antitrypsin deficient patients, 80–0333
effect of nontobacco cigarettes in asthmatics, 80–1699
effect of passive smoking in asthmatics, 80–1697
effect of passive smoking in children, 80–1425
effect of passive smoking in nonsmokers, 80–1129, 80–1426
effect of passive smoking on small airways function in nonsmokers, 80–1190
effect of smoking, 80–0328, 80–0768, 80–1129
effect of smoking and aging, 80–0945
effect of smoking and dust exposure in textile workers, 80–0771
effect of smoking and respiratory tract diseases in adolescents and children, 80–1701
effect of smoking and sulfur dioxide in copper smelter workers, 80–1686

- effect of smoking cessation, 80–0094
- effect of smoking filtertipped vs. non-filtertipped cigarettes, 80–0781
- effect of smoking in adolescents, 80–0141
- effect of smoking in asbestos workers, 80–0776
- effect of smoking in cotton mill workers, 80–0766
- effect of smoking in smokers vs. nonsmokers, 80–1699
- effect of smoking in smokers vs. nonsmokers vs. ex-smokers, 80–1428
- effect of smoking in welders, 80–1681
- effect of smoking on prognosis for hypoxemia in cholecystectomy patients, 80–1179
- effect of sulfur dioxide in smokers vs. nonsmokers, 80–0098
- effect of water pipe smoking, 80–0956
- forced random noise impedance parameters in smokers vs. nonsmokers and, 80–0331
- nonsmokers, 80–0953
- in nonsmoking grain workers, 80–1682
- obstructive airway disease in smokers vs. nonsmokers, 80–0779
- obstructive airway diseases and, 80–1166
- obstructive airway diseases in smokers vs. nonsmokers and, 80–1169
- as predictor of susceptibility to bronchitis in smokers, 80–0099
- in small airways disease diagnosis in smokers vs. nonsmokers, 80–1680
- smokers vs. ex-smokers, 80–1170
- smokers vs. nonsmokers, 80–0774, 80–0938, 80–0945
- smokers vs. nonsmokers vs. asthmatics, 80–1168
- smokers vs. nonsmokers vs. ex-smokers, 80–0949, 80–1177, 80–1181, 80–1183, 80–1428
- smokers vs. nonsmokers vs. ex-smokers in a fertilizer plant, 80–0332
- smokers vs. nonsmokers vs. ex-smokers vs. bronchitics, 80–0335
- smokers vs. nonsmokers vs. ex-smokers with antitrypsin deficiency, 80–1417
- smokers vs. nonsmokers with α_1-antitrypsin deficiency, 80–0095
- smokers vs. nonsmokers with respiratory tract infections, 80–0337
- smoking and, 80–1171, 80–1189, 80–1694
- smoking and emphysema and, 80–0097
- smoking and obstructive airway diseases and, 80–0773
- smoking in adolescents and, 80–1693
- in smoking vs. nonsmoking cadmium workers, 80–0334
- in smoking vs. nonsmoking civic, grain elevator, and sawmill workers, 80–1167
- in smoking vs. nonsmoking coal miners, 80–0942
- in smoking vs. nonsmoking dairy farmers and factory workers, 80–0936
- in smoking vs. nonsmoking Eisenmenger syndrome patients, 80–1688
- in smoking vs. nonsmoking firefighters, 80–1185
- in smoking vs. nonsmoking gastroesophageal reflux patients, 80–0780
- in smoking vs. nonsmoking obstructive airway disease patients, 80–0322, 80–0957
- in smoking vs. nonsmoking polychlorinated biphenyl-exposed electrical workers, 80–0954
- in smoking vs. nonsmoking sisal ropemakers, 80–0093
- in smoking vs. nonsmoking vs. ex-smoking beryllium workers, 80–1698
- in smoking vs. nonsmoking vs. ex-smoking bronchitics, 80–1422
- in smoking vs. nonsmoking vs. ex-smoking dockyard workers, 80–1695
- spirometric standards for nonsmokers, 80–1175

LUNG NEOPLASMS

(*See also* **BRONCHIAL NEOPLASMS; CARCINOMA, BRONCHOGENIC; CARCINOMA, EPIDERMOID; RESPIRATORY TRACT NEOPLASMS)**

- 1980 American Cancer Society report, 80–0078
- antismoking campaigns as preventive medicine in Austria, 80–0306
- antismoking campaigns as preventive medicine in Great Britain, 80–0297
- aryl hydrocarbon hydroxylase activity in smokers vs. nonsmokers, 80–1675
- attitudes toward smoking as risk factor in smokers vs. nonsmokers, 80–0926
- benzo(*a*)pyrene metabolism in lung tissue of patients, 80–0884
- calcitonin levels in smokers vs. nonsmokers vs. patients, 80–1162
- cessation of smoking as preventive medicine in asbestos-exposed workers, 80–1280
- cigarette smoke and influenza virus in etiology of, 80–0285
- cigarette smoke condensate in etiology of, 80–0723
- effect of air pollution on mortality rates in Pennsylvania, 80–0320
- effect of bidi vs. cigarette smoking on risk, 80–0299
- effect of cessation of smoking on risk, 80–0925
- effect of less hazardous cigarettes on mortality rate, 80–1679
- effect of methylcholanthrene in rats, 80–1132
- effect of phenobarbital on induction by dimethylnitrosamine in mice, 80–0744
- effect of smoking and asbestos exposure on mortality rates, 80–1405
- effect of smoking and asbestos exposure on risk in shipyard workers, 80–1208
- effect of smoking and asbestos on risk, 80–1145

effect of smoking and occupational exposure on incidence in blacks vs. whites, 80–1147
effect of smoking and occupational exposure on risk, 80–1154, 80–1401
effect of smoking characteristics and habit on risk, 80–1679
effect of smoking on mortality rate in asbestos workers, 80–1668
effect of smoking on mortality rate in Great Britain, 80–0080
effect of smoking on mortality ratio in Japan, 80–0302
effect of smoking on relative risk, 80–0299, 80–0762
effect of smoking on risk in iron foundry workers, 80–0318
effect of vitamin A on relative risk in heavy vs. light smokers, 80–0089
epoxide hydratase and glutathione *S*-transferase activity in lungs in patients, 80–0888
high- vs. low-tar cigarette preference in female patients, 80–0930
induced by 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone in rats, 80–0722
induced by asbestos in hamsters, 80–1141
induced by benzo(*a*)pyrene, dimethylbenzanthracene, and 3-methylcholanthrene in mice, 80–0886
induced by benzo(*a*)pyrene in hamsters, 80–1360
induced by benzo(*a*)pyrene in rats, 80–1120
induced by benzopyrenes in mice, 80–0068
induced by cigarette smoke condensate in hamsters, 80–1360
induced by nitrosonornicotine in mice, 80–0024
metronidazole therapy in smokers vs. nonsmokers and, 80–0756
mortality in aluminum reduction plant workers in Quebec, 80–0083
mortality in asbestos workers, 80–0292
mortality in beryllium-exposed workers, 80–0931
mortality in coal miners and gold miners in Australia, 80–0073
mortality in males vs. females in Canada, 80–0298
mortality in Poland, 80–1406
mortality in smokers vs. nonsmokers in Sweden, 80–1149
mortality in smoking vs. nonsmoking asbestos workers, 80–1145, 80–1394
mortality in smoking vs. nonsmoking smelter workers in Sweden, 80–0314
mortality in Sweden, 80–0303
relative risk in asbestos workers, 80–0292
relative risk in smokers vs. nonsmokers in Czechoslovakia, 80–0106
relative risk in smoking vs. nonsmoking nickel smelter workers, 80–0310
risk in ex-smokers, 80–0925
role of physicians in prevention in asbestos-exposed workers, 80–0935
sex ratio and tobacco consumption and, 80–0421
smoking and, 80–0078, 80–0755, 80–0761, 80–0924, 80–1160
smoking and air pollution and, 80–0309
smoking and asbestos exposure and, 80–0929
smoking and enzyme activity in lungs in patients, 80–1125
smoking and occupational exposure and, 80–0304
smoking and occupational exposure in etiology of, 80–0081
smoking and radiation in etiology of, 80–0316
smoking habit and mortality in Austria, 80–1150
smoking habit in patients, 80–0089, 80–0307
smoking habit in patients in Kenya, 80–0300
smoking habit in women and, 80–0182
smoking in chloromethyl ether workers and, 80–0321
smoking in coal miners and gold miners and, 80–0073
smoking in etiology of, 80–0920, 80–1148
smoking in the Pacific Basin and, 80–1672
in smoking vs. nonsmoking nickel workers, 80–0305
in smoking vs. nonsmoking uranium miners, 80–0079
in smoking vs. nonsmoking women, 80–0930
tar exposure in aluminum reduction plant workers and, 80–0083

LUNGS
(*See also* **RESPIRATORY SYSTEM**)
acetaldehyde and formaldehyde metabolism in cells, 80–0904
aryl hydrocarbon hydroxylase induction by benzo(*a*)pyrene and methylcholanthrene, 80–0052
asbestos exposure and pleural plaques in smokers vs. nonsmokers, 80–0103
benzo(*a*)pyrene binding to DNA in rats, 80–1130
benzo(*a*)pyrene metabolism in cell cultures, 80–0060
benzo(*a*)pyrene metabolism in microsomal fractions, 80–1383
benzo(*a*)pyrene metabolism in rabbits, 80–0051, 80–0881, 80–0908
benzo(*a*)pyrene metabolism in rats, 80–0743
benzo(*a*)pyrene metabolism in tissue of lung neoplasm patients, 80–0884
benzo(*a*)pyrene metabolite binding to DNA and RNA in mice, 80–0727
bronchoalveolar cell count in smoking vs. nonsmoking vs. ex-smoking pulmonary fibrosis patients, 80–1176
carbon monoxide uptake in conducting airways, 80–1341
chest radiograph in smoking vs. nonsmoking asbestos workers, 80–1696
determination of alpha particle activity in smokers vs. nonsmokers vs. ex-smokers, 80–1379
distribution of polonium-210 from cigarette smoke, 80–0257

effect of antioxidants and steroids on aryl hydrocarbon hydroxylase activity in rats, 80–1377
effect of aromatic hydrocarbons on aryl hydrocarbon hydroxylase activity in cells in rats, 80–0742
effect of asbestos on chest radiograph in smoking vs. nonsmoking vs. ex-smoking dockyard workers, 80–1419, 80–1695
effect of benzo(*a*)pyrene on aryl hydrocarbon and prolyl hydroxylase activity in rats, 80–1627
effect of cannabis on bacteria in rats, 80–1116
effect of carbon monoxide, hydrogen cyanide, and nitric oxide in rabbits, 80–1353
effect of carbon monoxide, nitrogen dioxide, and sulfur dioxide in squirrels, 80–0062
effect of carbon monoxide on protein synthesis in rats, 80–1346
effect of cigarette smoke condensate on morphogenesis in chick embryo, 80–0889
effect of cigarette smoke gas phase on benzo(*a*)pyrene metabolism in rabbits, 80–1367
effect of cigarette smoke in emphysema etiology, 80–0053
effect of cigarette smoke inhalation and dust on free cells in guinea pigs, 80–1380
effect of cigarette smoke on angiotensin I conversion in rabbits, 80–0264
effect of cigarette smoke on benzo(*a*)pyrene metabolism in rabbits, 80–1367
effect of marihuana smoke and vitamin C on mitotic abnormality incidence in cells, 80–1637
effect of marihuana smoke inhalation in rats, 80–1651
effect of marihuana vs. tobacco smoke inhalation on defense system in rats, 80–1626
effect of methylcholanthrene on aryl hydrocarbon hydroxylase activity in rats, 80–0912, 80–1097, 80–1122
effect of methylcholanthrene on epoxide hydrase activity in rats, 80–1097
effect of methylcholanthrene on microsomal enzymes in rats, 80–1132
effect of nicotine on metabolism of benzo(*a*)pyrene in rats, 80–0067
effect of nicotine on prostaglandins in rats, 80–0711
effect of nitrogen oxides in mice, 80–0892
effect of smoke inhalation, acrolein, and dust in rats, 80–0730
effect of smoke inhalation on aryl hydrocarbon hydroxylase activity in rats, 80–0912, 80–1097, 80–1122
effect of smoke inhalation on elastic recoil in rats, 80–1372
effect of smoke inhalation on enzyme activity in rats, 80–1107, 80–1350
effect of smoke inhalation on epoxide hydrase activity in rats, 80–1097
effect of smoke inhalation on protein synthesis in rats, 80–1346
effect of smoke inhalation on pulmonary alveolar macrophage recruitment in mice, 80–0059
effect of smoking and asbestos exposure on chest radiograph in workers, 80–0937
effect of smoking in asbestos workers, 80–0776
effect of tobacco extracts on enzyme activity in rats, 80–1127
effect of tobacco smoke and vitamin C on mitotic abnormality incidence in cells, 80–1637
elastic recoil pressure in smokers vs. nonsmokers, 80–1432
epoxide hydratase and glutathione *S*-transferase activity, 80–0888
glycosidase and protease activity in pulmonary alveolar macrophages of light smokers vs. heavy smokers vs. nonsmokers, 80–1655
9-hydroxybenzo(*a*)pyrene binding to DNA in rats, 80–1130
lidocaine uptake in smokers vs. nonsmokers, 80–0736
morphology in smoking vs. nonsmoking cotton industry workers, 80–0948
morphology in smoking vs. nonsmoking cotton textile workers, 80–1692
phenol metabolism in cell cultures, 80–0060
protein content in lavage fluid in smokers vs. nonsmokers, 80–1643
smoking and enzyme activity in lung neoplasm patients, 80–1125

MAMMARY NEOPLASMS
carcinoembryonic antigen and L-fucose levels in diagnosis in smokers vs. nonsmokers, 80–1389

Marihuana *See* **CANNABIS**

MASS MEDIA
antismoking campaign in cardiovascular diseases prevention program, 80–0120
in antismoking campaigns for children in France, 80–1000
in antismoking campaigns in Finland, 80–1255
effect of advertising and actor models on smoking habit, 80–0147
in health education program for lung diseases, 80–0161
tobacco industry sponsorship of sports events on television in Great Britain, 80–1311

MATERNAL—FETAL EXCHANGE
cadmium, lead, and mercury in smokers vs. nonsmokers, 80–0386
effect of maternal smoking on oxygen transport, 80–1619
effect of transplacental cigarette smoke condensate on neoplasm induction in mother and offspring in rats, 80–1644

best copy available

- environmental pollutants, 80–1579

Maternal smoking *See* **SMOKING, MATERNAL**

MATHEMATICS
- dose—response relationship of smoking to ischemic heart disease mortality, 80–1207
- effect of cigarette smoke particulates on air pollution in indoor environments, 80–1378
- first passage times in model for carbon monoxide content in blood of smokers, 80–1126
- kinetic model of cadmium metabolism, 80–1362
- linear multiple regression model for cigarette consumption in Switzerland, 80–1825
- model for predicting cigarette smoke composition, 80–0017
- model for smoking in arteriosclerosis etiology, 80–1457
- model of diffusion and dilution of gaseous components through cigarette paper, 80–0230
- model of skin neoplasm induction by cigarette smoke condensate in mice, 80–0252
- multiple regression equation for predicting total volatile bases in flue-cured tobacco, 80–1085
- pressure drop measurements in estimation of cigarette smoke dilution, 80–1078
- secondary Poisson process models for carboxyhemoglobin levels in smokers, 80–1642

Maximum mid-expiratory flow rate measurements *See* **RESPIRATORY FUNCTION TESTS**

MEMORY
- effect of nicotine content in cigarettes on immediate accuracy in high- vs. low-arousal smokers, 80–1534
- effect of nicotine in rats, 80–1355
- effect of pipe smoking, 80–0412
- effect of smoking, 80–1777, 80–1781
- smokers vs. nonsmokers, 80–1009

MENTAL DISORDERS
- clomipramine blood levels and tolerance in depressed patients, 80–1368

MENTHOL
- (*See also* **TOBACCO ADDITIVES; TOBACCO AROMA; TOBACCO FLAVOR**)
- activated carbon filters, 80–0537
- α-substituted vinyl menthyl carbonate as flavor release agent, 80–0586
- cigarettes in dermatitis etiology, 80–0138
- effect of application by vaporization on tobacco flavor, 80–0567
- effect of polymeric menthol-release agents on tobacco aroma and flavor, 80–0610
- fixation methods for tobacco flavor enhancement, 80–0441
- 3-substituted-*p*-menthane derivatives as cooling agents, 80–0623, 80–0624

MESOTHELIOMA
- mortality rates in smoking vs. nonsmoking asbestos workers, 80–1394
- smoking and asbestos exposure and, 80–0929
- smoking habit and asbestos exposure in patients, 80–1146
- in smoking vs. nonsmoking vs. ex-smoking dockyard workers, 80–1419

METABOLISM
- effect of nicotine content in cigarettes, 80–1138
- smokers vs. nonsmokers, 80–1656

METALS
- (*See also* **CHROMIUM; LEAD-210; POLONIUM-210; URANIUM**)
- as binder constituents, 80–0648
- cadmium, lead, and mercury levels in blood and placenta in smokers vs. nonsmokers, 80–0386
- determination in particulate matter and gas phase of cigarette smoke, 80–0034
- determination of trace metals in cigarette paper, 80–0011
- effect of amine complexes on cyanide content in tobacco smoke, 80–0564
- effect of calcium fertilizer on tobacco leaf components, 80–0008
- effect of calcium fertilizer on tobacco yield, 80–0008
- effect of climate and soil on levels in tobacco in Zambia, 80–0683
- effect of fertilization on potassium levels in tobacco leaf, 80–0202
- effect of fertilizer and pH on levels in tobacco, 80–1587
- effect of iron and manganese air pollution on mortality rates in the United States, 80–0291
- effect of magnesium and potassium on nutrient levels and phenol content in tobacco, 80–0197
- effect of palladium on aromatic hydrocarbon content in tobacco smoke, 80–0563
- effect of smoking and age on cadmium and zinc levels in urine, 80–0261
- effect of transition metals on smoke constituent levels, 80–0566
- effect of zineb on zinc content and distribution in tobacco, 80–0682
- in German cigarettes, 80–0229
- in Iraqi vs. U.S. tobacco varieties, 80–0036
- magnesium silicate hydrate as adsorbent for cigarette smoke constituents, 80–0544
- oxides and oxyhydrates in cigarette filters and tobacco as filtering agents, 80–1807
- ruthenium catalyst for carbon monoxide and nitric oxide removal from smoke, 80–1820

sintered material in cigarette filters, 80–0671
tobacco smoke condensate binding, 80–0877
trace element levels in coronary disease patients, 80–0362
trace element levels in tobacco varieties in Yugoslavia, 80–0692
use with organic compounds and resins in cigarette filters, 80–0526
zinc as tracer for evaluation of hydrophobic coatings in reconstituted tobacco, 80–0498
zinc levels in blood in myocardial infarct patients, 80–0366

METHYLCHOLANTHRENE
(*See also* **AROMATIC HYDROCARBONS**)
in aryl hydrocarbon hydroxylase induction in liver and lungs in rodents, 80–0052
effect on aryl hydrocarbon hydroxylase activity in lung cells in rats, 80–0742
effect on aryl hydrocarbon hydroxylase activity in lungs in rats, 80–0912, 80–1097, 80–1122
effect on epoxide hydrase activity in lungs in rats, 80–1097
effect on pulmonary microsomal enzymes in rats, 80–1132
in lung neoplasm induction in mice and rats, 80–0886, 80–1132

MOISTURE CONTENT
in tobacco leaf, effect of fertilization, 80–0202

MORBIDITY
(*See also* **MORTALITY**)
absenteeism in smokers vs. nonsmokers, 80–1391
coronary disease in smokers vs. nonsmokers vs. ex-smokers, 80–1717
effect of annual health checkups, 80–0074
effect of maternal nicotine administration in offspring in rats, 80–1369
effect of maternal smoking and socioeconomic status in children, 80–1738
effect of maternal smoking during pregnancy on children, 80–0134
smoking and coffee consumption in Yugoslavia and, 80–0918

MORTALITY
(*See also* **FETAL MORTALITY; INFANT MORTALITY; MORBIDITY**)
alcohol consumption and smoking-related fires and, 80–1838
asbestos workers in Italy, 80–0292
asbestosis in smoking vs. nonsmoking workers, 80–1145
cardiovascular diseases and, 80–0085
cardiovascular diseases in Japan, 80–1456
cardiovascular diseases in Mormons vs. non-Mormons, 80–0119
cardiovascular diseases in the United States, 80–0752
coronary disease and myocardial infarct in smokers vs. nonsmokers, 80–0115
coronary disease in smokers vs. nonsmokers vs. ex-smokers, 80–1717
coronary disease in the United States, 80–1211, 80–1459
effect of annual health checkups, 80–0074
effect of asbestos exposure in workers, 80–1668
effect of cessation of smoking in myocardial infarct patients, 80–0356 80–1728
effect of cessation of smoking on ratios in the United States, 80–1395
effect of nickel exposure in workers, 80–0753
effect of smoking, 80–0077
effect of smoking and aging on ischemic heart disease risk, 80–1464
effect of smoking and air pollution in the United States, 80–0291
effect of smoking and blood sugar on coronary disease rates, 80–1200
effect of smoking and obesity, 80–1143
effect of smoking and oral contraceptives on cardiovascular disease rates, 80–0377
effect of smoking and other risk factors on mortality in myocardial infarct patients, 80–0365
effect of smoking and socioeconomic factors in the United States, 80–0075
effect of smoking habit in physicians, 80–1144
effect of smoking in older males, 80–0754
effect of smoking on cardiovascular disease rates, 80–1729, 80–1731
effect of smoking on cardiovascular ratios, 80–0916
effect of smoking on cardiovascular risk in hypertensives, 80–1437
effect of smoking on coronary disease rates, 80–0293
effect of smoking on ischemic heart disease rate, 80–1207
effect of smoking on neoplasm rates, 80–0928
effect of smoking on rates, 80–0290
effect of smoking on rates in physicians in Finland, 80–1666
effect of smoking on risk in hypertensives, 80–1437
health practices in smokers vs. nonsmokers vs. ex-smokers and, 80–1392
lung neoplasm rates in smokers vs. nonsmokers in Sweden, 80–1149
lung neoplasms in aluminum reduction plant workers in Quebec, 80–0083
lung neoplasms in coal miners and gold miners in Australia, 80–0073
lung neoplasms in males vs. females in Canada, 80–0298
lung neoplasms in Poland, 80–1406
lung neoplasms in smelter workers in Sweden, 80–0314

lung neoplasms in smoking vs. nonsmoking asbestos workers, 80–1145
lung neoplasms in Sweden, 80–0303
nonsmokers in the United States, 80–0915
respiratory tract neoplasms in male physicians in Finland, 80–0751
smokers vs. nonsmokers, 80–0917, 80–1390
smokers vs. nonsmokers vs. ex-smokers in the United States, 80–1395
smoking and, 80–0755
smoking and alcohol consumption and, 80–1142
smoking and coronary disease trends in the United States, 80–1721
smoking and death certificates and, 80–1584
smoking and lung neoplasms in beryllium-exposed workers, 80–0931
smoking and lung neoplasms in Great Britain, 80–0080
smoking as risk factor for cardiovascular diseases in black vs. white males vs. females, 80–1710
smoking, asbestos exposure, and lung neoplasms in shipyard workers and, 80–1208
smoking habit and neoplasms, 80–0085
smoking habit in diabetics and, 80–1741
smoking-related fires, 80–0470
smoking-related neoplasms in the United States, 80–1397
in smoking vs. nonsmoking vs. ex-smoking asbestos workers, 80–1394
sudden vs. nonsudden death after myocardial infarct in smokers vs. nonsmokers, 80–0123
trends in cigarette consumption and arteriosclerosis rate, 80–1450

MOTIVATION
(*See also* **ATTITUDES; BEHAVIOR)**
cessation of smoking and, 80–0999, 80–1540, 80–1801
cessation of smoking in medical students in the U.S.S.R. and, 80–1240
effect of smoking habit motivation on cessation of smoking method choice, 80–1011
effect of social influence and acceptability in smokers vs. ex-smokers, 80–0148
effect on purchase of cessation of smoking aids, 80–0815
smoking habit and oral behavior, 80–0820
smoking habit in adolescents, 80–1515
smoking habit in adolescents in France, 80–0424
smoking habit in colored vs. white adolescents in South Africa, 80–0141
smoking habit in nurses, 80–1775
smoking habit in university students in Romania and, 80–1508
university students in Romania, 80–1753

MOUTH
smoking and epithelial keratinization in leukoplakia patients, 80–1486

MOUTH DISEASES
effect of smoking on pain in alveolitis sicca dolorosa patients, 80–1743
localized osteitis in smoking vs. nonsmoking dental surgery patients, 80–0808, 80–0992
smoking habit in gingivitis patients, 80–0397

MOUTH MUCOSA
effect of areca and tobacco chewing, 80–0394
effect of cigarette smoke exposure on carbon monoxide content in blood in monkeys, 80–1657
effect of 3-substituted-*p*-menthane derivatives as cooling agents in tobacco products, 80–0623, 80–0624

MOUTH NEOPLASMS
(*See also* **LEUKOPLAKIA; LIP NEOPLASMS)**
areca and tobacco chewing in etiology of, 80–0394
areca chewing in etiology of, 80–0317
bidi smoking in etiology of, 80–0317
effect of smoking and alcohol consumption on risk for multiple neoplasms in patients, 80–0315
risk factors, 80–0317
role of dentists and hygienists in health education, 80–1844
smoking and alcohol consumption and, 80–1678
smoking and alcohol consumption as risk factors for tongue neoplasms, 80–0922
smoking and alcohol consumption in etiology of, 80–0091
smoking habit and alcohol consumption in textile workers and, 80–0933
smoking in etiology of, 80–0317
tobacco chewing in etiology of, 80–0317

Mouthpieces *See* **SMOKING ARTICLES**

MUSCLES
characteristics in smokers vs. nonsmokers, 80–0277
effect of nicotine and carbon monoxide in rats, 80–0879
effect of nicotine on heart in rats, 80–1640
effect of nicotine on myocardial contractility in cats, 80–1436
effect of nicotine on peristalsis in esophagus in opossums, 80–1100
effect of smoking, 80–0149
effect of smoking on finger tremor, 80–1124
effect of smoking on tonus, 80–0044
effect of tobacco smoke on heart in rats, 80–1640

MUTAGENESIS
benzo(*a*)pyrene in hamster cells, 80–0743

chromosome abnormalities in smoking vs. nonsmoking vinyl chloride workers, 80–1487
cigarette smoke condensate in *Salmonella typhimurium*, 80–1122
cigarette smoke constituents in lymphocytes and spermatozoa, 80–0714
cigarettes in bacterial vs. mouse skin response tests, 80–0251
effect of gas phase cigarette smoke on respiration in yeast, 80–1628
effect of pulmonary alvolar macrophages and benzo(*a*)pyrene, 80–0050
effect of sugars in cigarettes on smoke condensate in *Salmonella typhimurium*, 80–0280
marihuana smoke condensate in *Salmonella typhimurium*, 80–0715, 80–0746
polycyclic aromatic hydrocarbons from soot in *Salmonella typhimurium*, 80–0054
sister chromatid exchange rates in smokers vs. nonsmokers, 80–1616
tobacco smoke condensate in *Salmonella typhimurium*, 80–0746
urine of smokers vs. nonsmokers, 80–0283
urine of smokers vs. nonsmokers in *Salmonella typhimurium*, 80–1140
urine of smokers vs. nonsmokers vs. cirrhotics in *Salmonella*, 80–1347

MYOCARDIAL INFARCT
(*See also* **CARDIOVASCULAR DISEASES; CORONARY DISEASE; HEART DISEASES**)
apolipoprotein A-I levels in smoking vs. nonsmoking patients, 80–1440
cadmium levels in blood in smoking vs. nonsmoking patients, 80–0366
DNA content in skin of patients, 80–1434
effect of cessation of smoking on mortality rate in patients, 80–0356, 80–1728
effect of cessation of smoking on prognosis, 80–0376
effect of cessation of smoking on reinfarction incidence, 80–0789
effect of multifactorial intervention program on mortality rates in patients in Finland, 80–0356
effect of nicotine on size in dogs, 80–1370
effect of practolol on prognosis in smoking vs. nonsmoking vs. ex-smoking patients, 80–0965
effect of risk factors on prognosis in patients, 80–0363, 80–0365
effect of smoking and coffee consumption on risk in women, 80–1727
effect of smoking and exercise on prognosis in patients, 80–0968
effect of smoking and oral contraceptives on incidence, 80–0372
effect of smoking and other risk factors on incidence, 80–0783
effect of smoking, hypertension, and oral contraceptives on risk, 80–1215
effect of smoking on risk in oral contraceptive users, 80–1718
effect of smoking on thromboembolism incidence in patients, 80–0351
incidence in smokers vs. nonsmokers vs. ex-smokers, 80–1723
lipoprotein levels in smoking vs. nonsmoking patients and relatives, 80–0371
perception of smoking and stress as causes, 80–0824
psychiatric risk factors in smoking vs. nonsmoking patients, 80–0111
psychosocial risk factors, 80–0117
relative risk for coronary disease in first-degree relatives of patients, 80–0126
relative risk in smokers vs. nonsmokers, 80–0115
risk factors in patients, 80–0979
smoking and lipid abnormality in patients, 80–0346
smoking and oral contraceptive use in young women and, 80–0346
smoking and other risk factors in menopausal women, 80–1704
smoking and psychosocial stress as risk factors in patients, 80–1435
smoking habit and vascular occlusion in patients, 80–0794
smoking habit in young female patients, 80–1724
smoking in young adults and, 80–0346
sudden vs. nonsudden death in smoking vs. nonsmoking patients, 80–0123
zinc levels in blood in patients, 80–0366

NASAL MUCOSA
effect of cigarette smoke exposure on carbon monoxide content in blood in monkeys, 80–1657
effect of smoke inhalation in hamsters, 80–1094
effect of 3-substituted-*p*-menthane derivatives as cooling agents in tobacco products, 80–0623, 80–0624
histopathological changes in smoking vs. nonsmoking nickel workers, 80–1408

NASOPHARYNGEAL NEOPLASMS
(*See also* **PHARYNGEAL NEOPLASMS; RESPIRATORY TRACT NEOPLASMS**)
effect of smoking and other factors on risk, 80–1674
effect of smoking on risk, 80–1677
induced by *N*-nitrosonornicotine and 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone in rats, 80–0722
induced by nitrosonornicotine in rats, 80–0024
risk factors, 80–0087
smoking and, 80–0084, 80–0087
smoking habit in patients in Hong Kong, 80–1396

NEONATE
(*See also* **BIRTH WEIGHT; BODY HEIGHT; FETAL GROWTH; INFANT MORTALITY; PREMATURITY)**
effect of maternal smoking, 80–1222
effect of maternal smoking and socioeconomic status on morbidity and mortality, 80–1738
effect of maternal smoking on birth height and weight, 80–1225
effect of maternal smoking on birth weight and gestational age, 80–1736
effect of maternal smoking on cadmium, lead, and mercury levels in cord blood, 80–0386
effect of maternal smoking on hematocrit, 80–0385
effect of maternal smoking on immune system in umbilical cord blood, 80–0800
effect of maternal smoking on leukocyte count in cord blood, 80–1733
effect of maternal smoking on low birth weight and prematurity incidence, 80–0382
effect of maternal smoking on mortality rates, 80–1474
effect of maternal smoking on sucking latency and pressure, 80–1473
effect of nicotine on development in rats, 80–0890
effect of parental smoking, 80–1471
effect of parental smoking on birth weight and mortality, 80–1224
effect of paternal smoking on birth weight and mortality, 80–1224

NEOPLASMS
(*See also* **CARCCINOGENESIS; CARCINOMA, EPIDERMOID; MESOTHELIOMA)**
1980 American Cancer Society report, 80–0078
attitudes of teachers toward health education in Great Britain, 80–1756
effect of alcohol consumption on mortality ratio in Japan, 80–0302
effect of maternal nicotine administration on incidence in offspring in rats, 80–1369
effect of occupational exposure on risk, 80–1154
effect of smoking and alcohol consumption on risk, 80–1403
effect of smoking and occupation on relative risk, 80–0082
effect of smoking and occupational exposure on incidence, 80–0312
effect of smoking on mortality, 80–0928
effect of smoking on mortality ratio in Japan, 80–0302
effect of smoking on relative risk, 80–0313
effect of smoking on risk in mouth neoplasm patients, 80–0315
environment and heredity in etiology of, 80–0921
epidemiology in the Pacific Basin, 80–1402
glycoprotein levels in smokers vs. head and neck neoplasm patients, 80–0934
incidence in New Zealand, 80–1671
induced by transplacental administration of cigarette smoke condensate in mother and offspring in rats, 80–1644
morbidity and mortality in the United States, 80–0078
mortality rates in asbestos workers, 80–1394
mortality ratios in smokers vs. nonsmokers vs. ex-smokers in the United States, 80–1395
National Cancer Institute Journal research editorials, 80–1160
passive smoking and, 80–1404
relative risk for head and neck neoplasms in smokers vs. nonsmokers, 80–0923
smoking and, 80–0924, 80–1151
smoking and alcohol consumption and, 80–0924
smoking, diet, and occupational exposure in etiology of, 80–0319
smoking habits of patients, 80–0085
smoking-related mortality rates in the United States, 80–1397
tracheobronchial epithelial multinucleation in smoking vs. nonsmoking patients, 80–0758

NEURONS
effect of cholinergic antagonists on nicotine in snails, 80–0728
effect of nicotine and cotinine in brain in rats, 80–0887
effect of nicotine in rats, 80–1349
effect of nicotine on tuberoinfundibular dopamine neurons in rats, 80–1365

NEUROTICISM
coronary disease in smokers vs. nonsmokers and, 80–0121
effect of nicotine on vigilance performance, 80–1532
effect on recidivism, 80–0817
effect on smoking characteristics, 80–1532
smokers vs. nonsmokers, 80–1498
smoking habit in children and, 80–1525
in smoking vs. nonsmoking adolescents, 80–1774

Neurotransmitters *See* **SEROTONIN**

NICKEL
(*See also* **METALS)**
effect of exposure on mortality rates in workers, 80–0753
effect of occupational exposure on lung neoplasm incidence in smokers vs. nonsmokers, 80–0305
nasal mucosal histopathological changes in smoking vs. nonsmoking workers, 80–1408
nasal pathology in smoking vs. nonsmoking workers, 80–0110

relative risk for lung neoplasms in smoking vs. nonsmoking smelter workers, 80–0310

NICKEL CONTENT
in cigarettes rolled by nickel workers, 80–0110
in nasal biopsy material from workers, 80–0110
in Norwegian vs. U.S. cigarette brands, 80–0110

NICOTINE
(*See also* **ALKALOIDS, TOBACCO**)
addictive behavior and, 80–0064, 80–0151, 80–0154
antibody production in goats and rabbits, 80–1613
in arteriosclerosis and cardiovascular disease etiology, 80–1458
in arteriosclerosis etiology in pigs, 80–0272
binding sites in brain in rats, 80–1665
biosynthesis, 80–0231
as discriminative stimulus in rats, 80–0040
distribution in mainstream vs. sidestream smoke, 80–1332
effect of alcohol consumption on mother and offspring in rats, 80–1609
effect of body weight and self-regulation on uptake in rats, 80–0903
effect of cigarette length on delivery, 80–1246
effect of cigarette paper on delivery, 80–0512
effect of ingestion in grazing animals, 80–0395
effect of intravenous vs. inhaled doses on smoking characteristics, 80–1772
effect of intravenous vs. inhaled doses on smoking habit, 80–1772
effect of maternal administration on offspring in rats, 80–1369
effect of personality on intake, 80–1610
effect of pH on uptake in brain in rats, 80–1645
effect of pyrolysis, 80–0708
effect of smoking characteristics on delivery, 80–1501
effect on acidosis and hypoxia incidence in fetus in monkeys, 80–1384
effect on adenosine triphosphatase and electrolyte transport in kidneys in rabbits, 80–0724
effect on arousal and emotion, 80–1001, 80–1002
effect on behavior, 80–0860
effect on behavior in mice, 80–1654
effect on behavior in rats, 80–1135
effect on benzo(*a*)pyrene metabolism in lungs in rats, 80–0067
effect on birth weight in rats, 80–0733
effect on blastocyst implantation in rats, 80–0256
effect on blood circulation, 80–0064
effect on blood circulation and pressure in uterus in monkeys, 80–1384
effect on blood circulation, blood pressure, and heart rate in uterus in pregnant rats, 80–1338
effect on blood parameters and tissues in rats, 80–0879
effect on blood pressure, 80–0064
effect on blood pressure and heart rate in cats, 80–1376
effect on body weight and food intake in rats, 80–1662
effect on body weight and water intake in rats, 80–0276
effect on cerebral arteries in dogs, 80–1110
effect on contractility of bronchi, bronchioles, and trachea, 80–0072
effect on copper content in organs in rats, 80–1374
effect on cough reflex in dogs, 80–0914
effect on cyclic guanosine 3′, 5′-monophosphate levels in brain in rats, 80–1101
effect on cyclic nucleotide levels in heart in rabbits, 80–0911
effect on dopamine metabolism in rats, 80–1349
effect on electroencephalogram, 80–1610
effect on electroencephalogram in mice, 80–1654
effect on electrolyte transport in kidneys in rats, 80–0724
effect on embryo implantation, growth, and parturition in rats, 80–0049
effect on enzyme activity in epididymis and testes in rats, 80–1340
effect on fetal and neonate development in rats, 80–0890
effect on flicker fusion frequency, 80–0064
effect on gastric mucosa in rats, 80–1356
effect on growth hormone and prolactin levels in rats, 80–1365
effect on heart muscle in rats, 80–1640
effect on heart rate, 80–0064
effect on heart rate in rabbits, 80–0911
effect on heart rate in rats, 80–0734
effect on HeLa cell division, 80–1387
effect on hemodynamics in dogs, 80–1351
effect on 5-hydroxytryptamine metabolism in brain in rats, 80–0887
effect on ingestion in rats, 80–1093
effect on memory in rats, 80–1355
effect on metabolism of benzo(*a*)pyrene in lungs in rabbits, 80–0051
effect on myocardial contractility in cats, 80–1436
effect on myocardial infarct size in dogs, 80–1370
effect on noradrenaline release from hypothalamic synaptosomes, 80–0748
effect on norepinephrine release from adrenergic nerve endings in animals, 80–0267
effect on oxygen transport in heart in dogs, 80–1351
effect on pain response in rats, 80–0906
effect on peristalsis in esophagus in opossums, 80–1100
effect on prolactin release in rats, 80–0275
effect on prostaglandins in lungs in rats, 80–0711
effect on protein metabolism in adult vs. newborn rodent brain, 80–0063

- effect on reflexes in rats, 80–0887
- effect on retina in cats, 80–0725
- effect on seizure susceptibility in mice, 80–0048
- effect on skin temperature, 80–0064
- effect on smoking habit, 80–0151, 80–0154
- effect on steroidogenesis in testes in rats, 80–1336
- effect on sympathetic nerve terminals in spleen in cats, 80–1361
- effect on testes in rats, 80–0905
- effect on tyrosine hydroxylase activity in adrenals and ganglia in rats, 80–1386
- effect on vasopressin levels in blood, 80–1653
- effect on vigilance performance in smokers vs. nonsmokers, 80–1532
- in lamina vs. midrib cigarette smoke condensates, 80–0027
- metabolism in *Microsporum gypseum*, 80–0282
- mouthpiece for progressive reduction of levels in cigarette smoke, 80–0662
- pharmacokinetics in rats, 80–1652
- physiological and psychological effects in smokers vs. nonsmokers, 80–1357
- psychophysiologic effects, 80–0064
- synthesis of related compounds, 80–0221
- in tobacco substitute formulation, 80–0658
- transfer rate from tobacco leaf to smoke, 80–1324
- use of deuterated analogues for nuclear magnetic resonance spectral analysis, 80–0233, 80–0245

Nicotine chewing gum *See* **TOBACCO SUBSTITUTES**

NICOTINE CONTENT

- (*See also* **ALKALOID CONTENT; NORNICOTINE CONTENT**)
- in blood and urine, effect of levels in cigarettes, 80–1352
- in blood, effect of cigar smoking, 80–1137
- in blood, effect of cigarette length, 80–1246, 80–1751
- in blood, effect of ethanol in rats, 80–1091
- in blood, effect of inhaling in cigar and pipe smokers, 80–1302
- in blood, effect of levels in cigarettes, 80–1138, 80–1206
- in blood, effect of smoking characteristics, 80–1790
- in blood, effect of snuff-dipping, 80–1137
- in Canadian cigarette brands, 80–0869
- in chewing tobacco, effect of fertilization with nitrogen, 80–0686
- in cigar smoke, effect of polymers, 80–0554
- in cigarette butts, effect of cigarette length, 80–1751
- in cigarette smoke, effect of agricultural practices, 80–0207
- in cigarette smoke, effect of carboxymethyl cellulose filters, 80–0511
- in cigarette smoke, effect of cellulose acetate filters, 80–0538
- in cigarette smoke, effect of cigarette characteristics and tobacco stalk position, 80–1608
- in cigarette smoke, effect of cigarette paper, 80–0033
- in cigarette smoke, effect of Cytrel substitute, 80–1544
- in cigarette smoke, effect of fibrous filters, 80–0541, 80–0542
- in cigarette smoke, effect of filters, 80–0029
- in cigarette smoke, effect of laminated tobacco foil, 80–0484
- in cigarette smoke, effect of paper perforation, 80–1264
- in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
- in cigarette smoke, effect of thermoplastic polymeric filters, 80–0493
- in cigarette smoke, effect of tobacco pyrolysis, 80–0708
- in cigarette smoke, effect of ventilated filters, 80–1016
- in cigarette smoke, effect of wool filters, 80–0496
- in cigarette smoke, effect on smoking characteristics, 80–0400
- in cigarette smoke of Canadian brands, 80–1603
- in cigarette smoke of green vs. pale yellow tobacco varieties, 80–0709
- in cigarettes, effect of expansion and fiberization, 80–0499
- in cigarettes, effect on cerebral cortex electrical activity, 80–0255
- in cigarettes, effect on cessation of smoking, 80–1012
- in cigarettes, effect on circulatory and metabolic parameters, 80–1138
- in cigarettes, effect on cognitive decisionmaking and psychomotor performance, 80–0255
- in cigarettes, effect on cotinine and nicotine levels in blood and urine, 80–1352
- in cigarettes, effect on memory and psychomotor performance in high- vs. low-arousal smokers, 80–1534
- in cigarettes, effect on psychomotor performance, 80–1793
- in cigarettes, effect on reduction of smoking, 80–1012
- in cigarettes, effect on respiratory system, 80–1687
- in cigarettes, effect on smoking characteristics, 80–1757
- in cigarettes, effect on smoking characteristics and carbon monoxide content in alveolar air, 80–1760
- in cigarettes, effect on smoking habit, 80–0150, 80–0400, 80–0459, 80–0829, 80–1352, 80–1777
- in cigarettes, effect on vasopressin levels in blood, 80–1653
- in cigarettes, physiological and psychological effects, 80–1505
- determination by pyrolysis-gas chromatography, 80–1329
- determination in U.S. cigarette brands, 80–0220

determination in blood and milk in cattle, 80–1594
determination in blood and urine, 80–0219
determination in blood by radioimmunoassay, 80–1613
determination in blood, tissue, and urine post-mortem samples, 80–0061
determination in cigarette brands in Great Britain, 80–0700
effect of smoke inhalation and genotype in mice, 80–1334
effect on tobacco quality, 80–0030, 80–0035
in flue-cured tobacco, effect of fertilization and plant density, 80–0681
in flue-cured tobacco, in determination of total volatile bases, 80–1085
in flue-cured tobacco varieties in Pakistan, 80–0204
in mainstream cigarette smoke, effect of air resistance and paper porosity, 80–0246
in regenerated tobacco products, 80–0632
in smoke of cigarettes containing puffed milo and tobacco, 80–0649
in smoke of cigarettes containing puffed rice and tobacco, 80–0650
in smoking products containing popped corn, 80–0651
in tobacco, effect of agricultural practices, 80–0192, 80–0198, 80–1585
in tobacco, effect of bacterial treatment, 80–0560
in tobacco, effect of breeding, 80–0194
in tobacco, effect of climate and soil in Zambia, 80–0683
in tobacco, effect of fermentation and topping in Italian variety, 80–1260
in tobacco, effect of fertilizer and pH, 80–1587
in tobacco, effect of ozone, 80–1069
in tobacco, effect of ozonization, 80–1823
in tobacco, effect of planting time, 80–1067
in tobacco, effect of ripeness, 80–0009
in tobacco, effect of spacing and topping, 80–1588
in tobacco, effect of sucker control, 80–0864
in tobacco leaf and smoke, effect of tobacco varieties, 80–1086
in tobacco leaf, effect of aging and curing, 80–1258
in tobacco leaf, effect of curing on distribution, 80–1542
in tobacco leaf, effect of enzyme activity, 80–1381
in tobacco leaf, effect of fertilization, 80–0202
in tobacco leaf, effect of flue-curing in Indian varieties, 80–1263
in tobacco leaf, effect of maturation stage, 80–0697
in tobacco leaf, effect of ozone on lipid fraction, 80–1819
in tobacco leaf, effect of potato virus Y infection, 80–0209
in tobacco smoke, effect of agricultural practices, 80–0198
in tobacco smoke, effect of expansion vs. reconstitution, 80–0174
in tobacco smoke, effect of plant density, 80–0874
in tobacco smoke, effect of transition metals, 80–0566
in tobacco wastes, 80–1326
in urine, as verification of self-report in cessation of smoking, 80–0288
in urine, as verification of smoking, 80–1646
in urine, effect of smoking in males vs. females, 80–1656

NICOTINE DERIVATIVES
determination of nicotine n′-oxide in cigarette smoke condensate, 80–0018

NICOTINE METABOLISM
(*See also* **ALKALOID METABOLISM**)
in brain in cats, 80–1376
in dogs, 80–0897
effect of ethanol in rats, 80–1091
effect of iminium oxidase, 80–1612
effect of smoke inhalation in rats, 80–1652
pharmacokinetics in rats, 80–1652
in wild species of *Nicotiana*, 80–0273

NICOTINE METABOLITES
(*See also* **COTININE; NORNICOTINE**)
antibody production in goats and rabbits, 80–1613
carcinogenicity, formation, and metabolism, 80–0873
determination in blood and urine in dogs, 80–0897
effect of *N*-nitrosonornicotine on rodlet formation in salivary glands in mice, 80–1113
effect of ethanol on levels in blood in rats, 80–1091
use of deuterium for analysis of formation and structure, 80–0245

NICOTINE PHARMACOLOGY
effect of alcohol on cyclic guanosine 3′, 5′-monophosphate levels in brain in rats, 80–1101
effect of 4-aminophrydine on sympathetic nerve terminals in spleen in cats, 80–1361
effect of ascorbic acid on enzyme activity in urogenital system in rats, 80–1340
effect of atropine, carbamylcholine, and D-tubocurarine on receptors in brain in rats, 80–1665
effect of atropine on heart function in guinea pigs, 80–1136
effect of atropine on hemodynamics in dogs, 80–1351
effect of atropine on oxygen transport in heart in dogs, 80–1351
effect of cholinergic antagonists on neurons in snails, 80–0728
effect of mecamylamine hydrochloride on noradrenaline release from hypothalamic synaptosomes, 80–0748
effect of opiate agonists on receptors in bovine adrenal chromaffin cells, 80–0896

effect of pilocarpine on catecholamines in brain in rats, 80–1102
effect of pilocarpine on tremor induction in rats, 80–1102
effect of pimozide on growth hormone and prolactin levels in rats, 80–1365
effect of pimozide on tuberoinfundibular neurons in rats, 80–1365
effect of reserpine on cerebral arteries in dogs, 80–1110
effect of testosterone propionate on enzyme activity in urogenital system in rats, 80–1340
effect on 5-hydroxytryptamine neurons in brain in rats, 80–0887
effect on noradrenaline release and perfusion pressure in artery in rabbits, 80–0739

NICOTINE REDUCTION
effect of bacterial treatment of tobacco, 80–0550, 80–0551, 80–0553, 80–0559, 80–0560, 80–0561
effect of boiling, drying, and rehydration of tobacco, 80–0547, 80–0548
effect of filter additives on levels in cigarette smoke, 80–1017
effect of halogenated hydrocarbons as extraction solvents in tobacco, 80–0565
effect of tobacco processing with potassium, 80–1814
effect of wetting and drying tobacco, 80–0546

NICOTINISM
effect of herbal tea, 80–0395
in infants, 80–0398
valerian and vitamins in treatment of withdrawal symptoms, 80–0664

NITRATE CONTENT
determination in tobacco, 80–0704
in tobacco, effect of ozone, 80–1069
in tobacco, effect on nitrosamine formation during curing, 80–0024
in tobacco soil, effect of agricultural practices and seasonal variation, 80–0002

NITRATES
(*See also* **NITROSAMINES**)
effect on polycyclic aromatic hydrocarbon levels in tobacco smoke, 80–0562
potassium nitrate removal from tobacco, 80–0552, 80–0555
in tobacco leaf and smoke, 80–0867
WHO environmental health criteria, 80–0747

Nitric oxide *See* **NITROGEN OXIDES**

NITROGEN CONTENT
determination in tobacco, 80–0021
effect of fertilization on levels in tobacco leaf, 80–0190
effect on tobacco quality, 80–0030, 80–0035
in fertilizer, effect on polyphenol content in flue-cured tobacco, 80–0840
in flue-cured tobacco, in determination of total volatile bases, 80–1085
in flue-cured tobacco varieties in Pakistan, 80–0204
in Taiwan vs. United States tobacco varieties, 80–0025
in tobacco, effect of climate and soil in Zambia, 80–0683
in tobacco, effect of fertilizer and pH, 80–1587
in tobacco, effect of nutrient deficiency, 80–1315
in tobacco, effect of ozone, 80–1069
in tobacco, effect of planting time, 80–1067
in tobacco, effect of priming and topping, 80–1590
in tobacco, effect of spacing and topping, 80–1588
in tobacco, effect on tar content, 80–1812
in tobacco leaf, effect of agricultural practices, 80–0201, 80–1591
of tobacco leaf, effect of calcium fertilizer, 80–0008
in tobacco leaf, effect of fertilization with magnesium, 80–1072
in tobacco leaf, effect of flue-curing in Indian varieties, 80–1263
in tobacco leaf, effect of homogenized leaf curing, 80–1261
in tobacco leaf, effect of maturation stage, 80–0697
in tobacco leaf, effect of potato virus Y infection, 80–0209, 80–0210
in tobacco soil, effect of agricultural practices and seasonal variation, 80–0002
in tobacco varieties, effect of fertilization, 80–1313

NITROGEN DIOXIDE
determination in cigarette smoke, 80–0032
effect of ascorbic and erythorbic acid on levels in tobacco smoke, 80–0557
effect of boiling, drying, and rehydration on levels in tobacco, 80–0547, 80–0548
effect on lipid content in lungs and trachea in squirrels, 80–0062

NITROGEN OXIDE CONTENT
in cigarette smoke, effect of humidity and puff parameters, 80–1599

NITROGEN OXIDES
in cigarette smoke, effect of cigarette paper, 80–0033
determination in cigarette smoke, 80–0032
effect of nitric oxide on aorta, arteries, and lungs in rabbits, 80–1353
effect on aorta and coronary arteries in rabbits, 80–1117
effect on immune system in mice, 80–0892
effect on lungs in mice, 80–0892
in tobacco smoke, effect of ruthenium catalyst on levels, 80–1820

NITROSAMINE CONTENT
- determination of nitrosonornicotine in tobacco, chewing tobacco, and snuff, 80–0024
- in tobacco smoke, effect of ascorbic and erythorbic acid, 80–0557
- in tobacco smoke, effect of silicic acid—polystyrene filter, 80–1813

NITROSAMINE METABOLISM
- in esophageal cells, 80–0265

NITROSAMINES
- (*See also* **NITRATES)**
- carcinogenicity, formation, and metabolism, 80–0873
- DNA binding in esophageal cells, 80–0265
- effect of *N*-nitrosodiethylamine on metabolism in respiratory system in pregnant hamsters, 80–1134
- effect of cigarette smoke inhalation on retention in respiratory system, 80–0718
- effect of diethylnitrosamine on respiratory tract neoplasm induction in hamsters, 80–0721
- effect of phenobarbital on liver and lung neoplasm induction by dimethylnitrosamine in mice, 80–0744
- effect of tobacco pyrolysis and transfer rate on cigarette smoke content, 80–0022
- effect of transplacental *N*-nitrosodiethylamine on metabolism in fetal trachea in hamsters, 80–1134
- formation in tobacco smoke, 80–0032
- interactive effect with influenza virus in lung neoplasm etiology, 80–0285
- nitrosonornicotine in esophageal and nasal neoplasm induction in rats, 80–0024
- nitrosonornicotine in lung neoplasm induction in mice, 80–0024
- sources of exposure, 80–0878
- WHO environmental health criteria, 80–0747

NONSMOKERS
- effect of carbon monoxide on carboxyhemoglobin levels, 80–1358
- effect of carbon monoxide on content in expired air, 80–1358
- effect of carbon monoxide on heart function, 80–1210
- effect of carbon monoxide on psychomotor performance, 80–1625
- effect of carbon monoxide on work capacity at high altitude, 80–0070
- effect of carboxyhemoglobin levels on lung function, 80–1186
- effect of cigarette paper pyrolysis on carboxyhemoglobin levels, 80–1129
- effect of passive smoking, 80–0847
- effect of passive smoking and exercise on lung function, 80–1426
- effect of passive smoking on aggression, 80–0415
- effect of passive smoking on aggression and anxiety, 80–0823
- effect of passive smoking on annoyance response, 80–1789
- effect of passive smoking on carboxyhemoglobin levels, 80–0378
- effect of passive smoking on cardiovascular system, 80–0964
- effect of passive smoking on lung function, 80–1129, 80–1190
- lung function, 80–0953
- lung function in grain workers, 80–1682
- mortality in the United States, 80–0915
- perception of smokers, 80–0146
- rights of, attitudes of health department personnel in Hawaii, 80–0826
- rights of, in Howard County, Maryland, 80–0180
- rights of, in the United States, 80–1039
- rights of, in the workplace, 80–1041
- spirometric standards, 80–1175

Noradrenaline *See* **CATECHOLAMINES**

Norepinephrine *See* **CATECHOLAMINES**

NORNICOTINE
- (*See also* **ALKALOIDS, TOBACCO; NICOTINE METABOLITES)**
- biosynthesis, 80–0231
- determination in blood and urine in dogs, 80–0897
- effect of *N*-nitrosonornicotine on rodlet formation in salivary glands in mice, 80–1113
- effect on nicotine binding in brain in rats, 80–1665
- formation in *Microsporum gypseum*, 80–0282
- formation of nitroso derivative in tobacco, 80–0024
- metabolism in wild species of *Nicotiana*, 80–0273
- *N*-nitrosonornicotine in liver and nasopharyngeal neoplasm induction in rats, 80–0722
- synthesis of related compounds, 80–0221
- use of deuterium for analysis of formation and structure, 80–0245

NORNICOTINE CONTENT
- (*See also* **ALKALOID CONTENT; NICOTINE CONTENT)**
- determination by pyrolysis—gas chromatography, 80–1329
- in tobacco, effect of sucker control, 80–0864
- in tobacco leaf and smoke, effect of tobacco varieties, 80–1086
- in tobacco leaf, effect of enzyme activity, 80–1381

NOSE
- effect of nickel exposure in smokers vs. nonsmokers, 80–0110
- effect of passive smoking, 80–0809, 80–1748

NOSE NEOPLASMS
- (*See also* **RESPIRATORY TRACT NEOPLASMS)**
- in nickel workers, 80–0110

smoking in nickel workers and, 80–1408

Oat cell carcinoma *See* **CARCINOMA, BRONCHOGENIC**

OBESITY
(*See also* **BODY WEIGHT**)
effect of smoking on mortality, 80–1143
effect of weight loss on high density lipoprotein cholesterol levels, 80–0066
smokers vs. nonsmokers in Finland, 80–1056
smokers vs. nonsmokers in Poland, 80–0994

OBSTRUCTIVE AIRWAY DISEASES
(*See also* **BRONCHITIS; CHRONIC BRONCHOPULMONARY DISEASE; EMPHYSEMA; RESPIRATORY TRACT DISEASES; SMALL AIRWAYS DISEASE**)
antitrypsin deficiency as risk factor in smokers vs. nonsmokers vs. ex-smokers, 80–1417
cessation of smoking as preventive medicine in patients, 80–1423
effect of cigar smoke on carboxyhemoglobin levels in patients, 80–0353
effect of cigarette consumption on lung function in patients, 80–0957
effect of education and health awareness on cessation of smoking in patients, 80–0940
effect of ipratropium bromide on lung function and mucociliary clearance in patients, 80–0339
forced random noise impedance parameters in diagnosis of, 80–0331
lung function and, 80–1166
lung function in smokers vs. nonsmokers and, 80–0779
lung function in smokers vs. nonsmokers vs. ex-smokers, 80–1181
lung function in smoking vs. nonsmoking patients, 80–0322, 80–0773, 80–0957
manual for education of patients, 80–1165
respiratory function tests in diagnosis of, 80–0938, 80–1411
in smokers vs. nonsmokers in Micronesia, 80–0764
smoking and, 80–0330, 80–1694
smoking and antitrypsin deficiency and, 80–0100, 80–1172
smoking and incidence in psychiatric clinic patients, 80–1049
smoking and other risk factors, 80–1169
smoking in etiology of, 80–0328
in smoking vs. nonsmoking Fabry's disease patients, 80–1424
in smoking vs. nonsmoking machine building workers, 80–1182
in smoking vs. nonsmoking patients with antitrypsin deficiency, 80–0765
spirometry in diagnosis of, 80–0104

OCCUPATIONAL DISEASES
(*See also* **ASBESTOSIS; PNEUMOCONIOSIS**)
asbestosis, 80–0776
asbestosis in dockyard workers in Great Britain, 80–0107
asthma in electronics workers, 80–0096
bronchitis in dust-exposed workers, 80–0108
byssinosis in smoking vs. nonsmoking sisal workers, 80–0336
byssinosis in textile workers, 80–0771
effect of asbestos exposure in drywall construction workers, 80–0326
farmer's lung, 80–0109

OCCUPATIONS
aluminum reduction plant workers, 80–0083
asbestos workers, 80–0292, 80–0716, 80–0776, 80–0777, 80–0937, 80–1145, 80–1280, 80–1394, 80–1405, 80–1668, 80–1696
asphalt workers, 80–1187
battery factory workers, 80–0250
beryllium workers, 80–0931, 80–1698
cadmium workers, 80–0334
cedar sawmill workers, 80–1410
cement plant workers, 80–1683
chemical workers, 80–1227
chimney sweeps, 80–0951
chloromethyl ether workers, 80–0321
civic workers, 80–1167
coal miners, 80–0073, 80–0942, 80–1689
copper smelter workers, 80–1686
cosmetologists, 80–0775
cotton industry workers, 80–0948
cotton textile workers, 80–0766, 80–1692
dairy farmers, 80–0936
dentists, 80–1766
dockyard workers, 80–0107, 80–1419, 80–1695
drywall construction workers, 80–0326
electrical workers, 80–0954
electronics workers, 80–0096, 80–0963
engineers, 80–1245
factory workers, 80–0936
farm workers, 80–0109
fertilizer plant workers, 80–0332
firefighters, 80–1185
gold miners, 80–0073
grain elevator workers, 80–1167, 80–1410
grain handlers, 80–1348, 80–1682
health professionals, 80–0813
hospital employees, 80–1516
industrial workers, 80–0341, 80–1418
iron foundry workers, 80–0318
jewelry factory workers, 80–0880
journalists, 80–0403
lacquerers, 80–1669
machine building workers, 80–1182
machinists, 80–0763

mechanics, 80–1622
metallurgists, 80–0763
municipal workers, 80–1410
naval personnel in the United States, 80–1564
nickel smelter workers, 80–0310
nickel workers, 80–0110, 80–0305, 80–0753, 80–1408
nurses, 80–0160, 80–0182, 80–1241, 80–1775
operating room personnel, 80–1119
ore dressing plant workers, 80–1104
phosphate workers, 80–1702
physicians, 80–1144, 80–1245, 80–1509, 80–1666, 80–1766
police officers, 80–0071
railroad workers, 80–1493
safety personnel, 80–0127
sawmill workers, 80–1167, 80–1410
shipyard workers, 80–0935, 80–1208
sisal ropemakers, 80–0093
sisal workers, 80–0336
smelter workers, 80–0314
submarine patrolmen, 80–1663
tea blenders, 80–1700
teachers, 80–1348
textile workers, 80–0763, 80–0771, 80–0933
traffic policemen, 80–1614
uranium miners, 80–0079
vinyl chloride workers, 80–1487
welders, 80–1650, 80–1681
wire mill workers, 80–1690

Oral neoplasms *See* **MOUTH NEOPLASMS**

OSTEOPOROSIS
smoking in etiology of, 80–0805

Ovaries *See* **UROGENITAL SYSTEM**

OXYGEN TRANSPORT
carbon monoxide exchange ratio in lungs in smokers vs. nonsmokers, 80–1166
effect of cessation of smoking and exercise, 80–1708
effect of maternal smoking during pregnancy, 80–1619
effect of nicotine in heart in dogs, 80–1351
in heavy vs. light smokers vs. nonsmokers, 80–1359

PANCREATIC NEOPLASMS
acinar cell dysplasia in smokers vs. nonsmokers, 80–1157
smoking and, 80–0090
smoking habit in patients, 80–1409

Paper, cigarette *See* **CIGARETTE PAPER**

Paraffins *See* **ALIPHATIC HYDROCARBONS**

Particulate matter *See* **TARS, CIGARETTE; TARS, TOBACCO**

PASSIVE SMOKING
attitudes in the United States, 80–0862
effect of carbon monoxide on carboxyhemoglobin levels in nonsmokers, 80–0378
effect of cigarette paper pyrolysis on carboxyhemoglobin levels, 80–1129
effect of cigarette smoke particulates on air pollution in indoor environments, 80–1378
effect of parental smoking and air pollution on respiratory tract disease incidence in children, 80–0952
effect of sidestream smoke on eyes, nose, and throat, 80–0809
effect on aggression and anxiety in nonsmokers, 80–0823
effect on aggression in angered vs. nonangered nonsmokers, 80–0415
effect on annoyance response in nonsmokers, 80–1789
effect on asthma incidence in adolescents in New Zealand, 80–0778
effect on cardiovascular system in nonsmokers, 80–0964
effect on coronary disease patients, 80–0358
effect on eyes, heart rate, and respiration, 80–1388
effect on eyes, nose, and throat, 80–1748
effect on gastric secretion, 80–1226
effect on lung function and respiratory symptoms in asthmatics, 80–1697
effect on lung function in children, 80–1425
effect on lung function in nonsmokers, 80–1129, 80–1190, 80–1426
effect on nonsmokers, 80–0847
effect on respiratory symptom incidence in children in France, 80–1420
effect on respiratory tract infection incidence in children, 80–0801
effect on tonsillopharyngitis incidence in families in Canada, 80–1180
health effects for high-risk groups, 80–0180
neoplasms and, 80–1404
recommendations of the Quebec Advisory Council on the Environment for legislation, 80–0847
research needs, 80–1573
simulated cigarette and holder for reduction of, 80–1830

Parental smoking *See* **SMOKING, PARENTAL**

Paternal smoking *See* **SMOKING, PATERNAL**

Peak expiratory flow measurements *See* **RESPIRATORY FUNCTION TESTS**

PEER GROUPS
  effect on smoking habit, 80–0147
  effect on smoking habit in adolescents, 80–1243, 80–1518
  effect on smoking habit in children in Sweden, 80–0419
  in prevention of smoking in adolescents, 80–1583
  teenage counseling in antismoking campaigns for adolescents, 80–0468

Peptic ulcer *See* **ULCER, PEPTIC**

PERCEPTION
  (*See also* **COGNITION**)
  effect of masculine and feminine brand names on cigarette flavor evaluation in males vs. females, 80–1763
  effect of parental attitudes and behavior in children, 80–1752
  smoking and stress as causes of myocardial infarct, 80–0824
  subliminal perception in reduction of smoking, 80–1506

Perinatal mortality *See* **INFANT MORTALITY**

PERIODONTAL DISEASES
  (*See also* **TEETH**)
  gingivitis in smokers vs. nonsmokers, 80–0988
  snuff-dipping in etiology of, 80–0390

PERSONALITY
  (*See also* **BEHAVIOR**)
  cessation of smoking method selection and, 80–1010
  coronary disease and, 80–0117
  coronary disease in smokers vs. nonsmokers and, 80–0121
  effect of nicotine on arousal and emotion in introverts vs. extraverts, 80–1001
  effect of smoking control programs in adolescents, 80–1234
  effect of smoking on contingent negative variation, 80–1496
  effect on cessation of smoking, 80–1498
  effect on smoking characteristics, 80–1610
  effect on smoking habit, 80–1498
  effect on vigilance performance in smokers vs. nonsmokers, 80–1532
  extraversion and neuroticism in smokers vs. nonsmokers, 80–1498
  high- vs. low-arousal smokers, 80–1771
  locus of control scale in smokers vs. nonsmokers vs. ex-smokers, 80–0157
  smoking and, 80–1512
  smoking and arteriosclerosis and, 80–0970
  smoking habit in academic psychologists and, 80–0144
  smoking habit in children and, 80–1525
  smoking habit in male vs. female adolescents and, 80–1514
  smoking habit in males vs. females and, 80–1776
  smoking habit in psychosomatic disorder patients and, 80–0406
  in smoking vs. nonsmoking adolescents, 80–1518, 80–1774
  in smoking vs. nonsmoking alcoholics, 80–0830
  Type A behavior in coronary disease patients, 80–1705

PESTICIDE RESIDUES
  (*See also* **TOBACCO CONTAMINANTS**)
  DDT levels in flue-cured tobacco, 80–0186
  determination in tobacco, 80–1089, 80–1327, 80–1328
  determination of carbamates in tobacco, 80–0214, 80–0241
  determination of metobromuron in tobacco, 80–1082
  determination of nicotine in blood and milk in cattle, 80–1594
  determination of organochlorine insecticides in tobacco, 80–0235, 80–0236
  effect of acephate powder vs. emulsion on levels in tobacco, 80–0203
  effect of agricultural practices on butralin levels in tobacco, 80–0244
  effect of dosage on levels in tobacco leaves, 80–0191
  effect of maleic hydrazide on tobacco sales in West Germany, 80–1827
  effect of zineb on zinc content and distribution in tobacco, 80–0682
  maleic hydrazide levels in tobacco, 80–0864, 80–0865
  maleic hydrazide metabolism in tobacco, 80–0045
  methomyl in tobacco, 80–0046
  methoprene content in cigarette butt and smokestreams, 80–1266
  methoprene toxicity in rodents, 80–1266
  in soils and crops, 1971 National Soils Monitoring Program, 80–0193
  in tobacco, health effects in smokers, 80–0461
  in tobacco imports in Europe, 80–0001
  in tobacco soil in Ontario, 80–0703

PHAGOCYTOSIS
  effect of cigarette smoke on leukocytes, 80–1354
  effect of smoking on pulmonary alveolar macrophages, 80–1430
  effect of tobacco smoke vs. marihuana inhalation in rats, 80–1343

in pulmonary alveolar macrophages from smokers vs. nonsmokers, 80–0745

PHARYNGEAL NEOPLASMS
(*See also* **NASOPHARYNGEAL NEOPLASMS; RESPIRATORY TRACT NEOPLASMS)**
smoking and alcohol consumption and, 80–1678
smoking and alcohol consumption in etiology of, 80–0091
smoking habit and alcohol consumption in textile workers and, 80–0933

PHARYNGITIS
effect of smoking on incidence in families in Canada, 80–1180

PHARYNX
(*See also* **RESPIRATORY SYSTEM)**
bacterial adherence to cells in smokers vs. nonsmokers vs. bronchitics, 80–1685
effect of passive smoking, 80–1748
effect of smoking and air pollution on *H. influenzae* incidence in phosphate workers, 80–1702

PHENOL CONTENT
in cigarette smoke, effect of Cytrel substitute, 80–1544
in cigarette smoke, effect of filters, 80–1079
in cigarette smoke, effect of laminated tobacco foil, 80–0484
in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
in cigarette smoke, effect of protein granule filters, 80–0523
determination in cigarette smoke, 80–1079
determination in cigarette smoke condensate, 80–0037
determination in cigarette smoke of tobacco varieties, 80–1598
determination in tobacco by Folin-Ciocaltau reagent, 80–0247
effect of aging, curing, and fertilization on chlorogenic acid and rutin levels in flue-cured tobacco, 80–0840
effect of agricultural practices on polyphenol levels in chlorophyll-deficient varieties, 80–1071
polyphenols in cured tobacco leaf and seedlings, 80–0690, 80–0697
solanesol in tobacco wastes, 80–1326
in tobacco, effect of agricultural practices, 80–0197, 80–1074
in tobacco, effect of ozonization, 80–1823
in tobacco, effect of processing, 80–1074
in tobacco leaf, effect of curing, 80–1816
in tobacco leaf, effect of flue-curing in Indian varieties, 80–1263
in tobacco leaf, effect of ozone on lipid fraction, 80–1819
in tobacco leaf, effect of potato virus Y infection, 80–0209
in tobacco smoke, effect of plant density, 80–0874

PHENOLS
effect of tobacco curing on distribution in tobacco leaf, 80–1542
effect on metabolism of benzo(*a*)pyrene in lungs in rabbits, 80–0051
isolation from cigarette smoke condensate by gel chromatography, 80–1088
in lamina vs. midrib cigarette smoke condensates, 80–0027
metabolism in lung cell cultures, 80–0060
in Natu tobacco, 80–1325
in smoke condensate in tobacco varieties, 80–0876
in tobacco leaf and smoke, 80–0867

PHYSICAL ACTIVITY
(*See also* **EXERCISE)**
effect on heart rate in smokers vs. nonsmokers, 80–1195
smoking habit and, 80–0155
in smoking vs. nonsmoking patients and postoperative thrombosis, 80–1707

PHYSICAL FITNESS
effect of cessation of smoking and exercise in bronchitics, 80–1427
effect of smoking, aging, and workload on heart rate, 80–1716
smoking and body weight and, 80–1567

PIGMENTS, TOBACCO
carotene, chlorophyll, and xanthophyll levels during maturation in Indian varieties, 80–1077
effect of flue-curing on levels in Indian varieties, 80–1263
effect of insecticides on carotenoid and chlorophyll levels, 80–1068

Pipes *See* **SMOKING ARTICLES**

Pipe smoking *See* **SMOKING, PIPE**

Pipe tobacco smoke *See* **SMOKE, TOBACCO**

PLACENTA
benzo(*a*)pyrene metabolism, 80–1139
benzo(*a*)pyrene metabolism in smokers vs. nonsmokers, 80–1128
cadmium, lead, and mercury levels in smokers vs. nonsmokers, 80–0386
effect of maternal smoking, 80–1222, 80–1469
effect of maternal smoking and aging on abruptio placentae and placenta praevia incidence, 80–1474
effect of maternal smoking and weight gain on disorder incidence, 80–1475

effect of maternal smoking on aryl hydrocarbon hydroxylase activity, 80–1225
effect of maternal smoking on blood vessels, 80–1468
effect of maternal smoking on disorder incidence, 80–1476
effect of maternal smoking on morphology, 80–1732
effect of smoking in blacks vs. whites, 80–0391
enzyme activity as indicator of prenatal exposure to environmental pollutants, 80–1579

PNEUMOCONIOSIS
(*See also* **LUNG DISEASES; OCCUPATIONAL DISEASES**)
byssinosis in smoking vs. nonsmoking sisal workers, 80–0336
effect of smoking and dust exposure on byssinosis incidence in textile workers, 80–0771
smoking and respiratory symptoms in coal miners and, 80–1689
smoking in cotton industry workers and, 80–0948

POLONIUM-210
(*See also* **METALS**)
bibliography of tobacco smoke carcinogenicity, 80–1385
determination of alpha particle activity in lungs in smokers vs. nonsmokers vs. ex-smokers, 80–1379
distribution in lungs from cigarette smoke, 80–0257

POLONIUM-210 CONTENT
in bronchial epithelium of smokers vs. nonsmokers, 80–0737
determination in cigarette smoke condensate, 80–1597
in tobacco, effect of acids, 80–1541

Polycyclic aromatic hydrocarbons *See* **AROMATIC HYDROCARBONS**

Polyphenols *See* **PHENOLS**

PREGNANCY
(*See also* **FETUS; NEONATE; PREMATURITY**)
ectopic and myomatous type incidence in smokers vs. nonsmokers, 80–1735
effect of *N*-nitrosodiethylamine on metabolism in respiratory system in hamsters, 80–1134
effect of maternal nicotine administration on offspring in rats, 80–1369
effect of maternal smoking, 80–0387
effect of maternal smoking and aging on abruptio placentae and placenta praevia incidence, 80–1474
effect of maternal smoking and alcohol consumption on fetus, 80–1740
effect of maternal smoking and body weight on infant birth weight and growth, 80–0130
effect of maternal smoking on birth weight and gestational age, 80–1736
effect of maternal smoking on carboxyhemoglobin and hemoglobin levels, 80–1619
effect of maternal smoking on fetal risk, 80–0383
effect of maternal smoking on fetus, 80–1734
effect of maternal smoking on leukocyte count in cord blood, 80–1733
effect of maternal smoking on oxygen transport, 80–1619
effect of maternal smoking on spontaneous abortion incidence, 80–0133
effect of nicotine and alcohol consumption on food consumption in rats, 80–1609
effect of nicotine and alcohol consumption on length of gestation and litter size in rats, 80–1609
effect of nicotine on birth weight in rats, 80–0733
effect of nicotine on blood circulation, blood pressure, and heart rate in uterus in rats, 80–1338
effect of nicotine on litter size in rats, 80–0890
effect of smoking on mother and fetus, 80–0384
epidemiological tools for assessment of risks of maternal smoking, 80–0388
estrogen:creatinine ratio in prediction of fetal complications in smokers vs. nonsmokers, 80–1737
maternal smoking in planned vs. unplanned pregnancy, 80–1524
smoking and drug exposure history, 80–0129
smoking habit in obstetrical inpatients, 80–0818

PREMATURITY
(*See also* **BIRTH WEIGHT; FETUS; NEONATE; PREGNANCY**)
effect of maternal smoking, 80–0799, 80–0801, 80–1470
effect of maternal smoking and body weight on incidence, 80–0381
effect of maternal smoking on incidence, 80–0382, 80–1472

PREVENTION OF SMOKING
(*See also* **ANTISMOKING CAMPAIGNS; HEALTH EDUCATION**)
in children and adolescents, 80–1583
effect of life skills training in adolescents, 80–0811, 80–1533
methods, 80–0853
in the United States, 80–1304

PROSTATE
(*See also* **UROGENITAL SYSTEM**)
effect of smoking and alcohol consumption on function, 80–1488

PROTEIN CONTENT
in bronchoalveolar lavage fluid in smokers vs. nonsmokers, 80–1111
effect on tobacco quality, 80–0030
immunoglobulins and secretory component in lung lavage fluid in smokers vs. nonsmokers, 80–1643

in tobacco, effect of ozone, 80–1069
in tobacco, effect of planting time, 80–1067
in tobacco leaf, effect of aging and curing, 80–1258
in tobacco leaf, effect of agricultural practices, 80–0202, 80–1591
in tobacco leaf, effect of curing, 80–0012
in tobacco leaf, effect of fertilization with magnesium, 80–1072
in tobacco leaf, effect of flue-curing in Indian varieties, 80–1263
in tobacco leaf, effect of homogenized leaf curing, 80–1261
in tobacco leaf, effect of maturation stage, 80–0697

PROTEIN METABOLISM
effect of cigarette smoke on angiotensin I conversion in lungs in rabbits, 80–0264
effect of nicotine in adult vs. newborn rodent brain, 80–0063

PROTEINS
benzo(*a*)pyrene metabolite binding in embryo cells in hamsters, 80–1096
bovine milk whey and egg white granules as filter, 80–0523
determination of polypeptide composition of Fraction I proteins in tobacco by isoelectric focusing, 80–1607
effect of carbon monoxide on synthesis in rats, 80–1346
effect of smoke inhalation on synthesis in lungs in rats, 80–1346
identification of Fraction I polypeptides in tobacco by isoelectric focusing, 80–0875
neurophysins in diabetes and inappropriate antidiuretic hormone secretion, 80–0140
use of tobacco leaf proteins as food, 80–0443

PSYCHOMOTOR PERFORMANCE
effect of carbon monoxide, 80–1131
effect of carbon monoxide on vigilance in nonsmokers, 80–1625
effect of nicotine content in cigarettes on letter cancellation speed in high- vs. low-arousal smokers, 80–1534
effect of nicotine on vigilance, 80–1793
effect of pipe smoking, 80–0412
effect of smoking, 80–1004
effect of smoking and alcohol consumption, 80–0429, 80–1781
effect of smoking and urinary pH, 80–1530
effect of smoking on vigilance, 80–1777, 80–1793

PULMONARY ALVEOLAR MACROPHAGES
angiotensin I convertase and lysozyme activity in smokers vs. nonsmokers, 80–1174
antitrypsin activity in smokers vs. nonsmokers, 80–0329
benzo(*a*)pyrene metabolism, 80–0900, 80–1095
effect of cigarette smoke inhalation and dust in guinea pigs, 80–1380
effect of cigarette smoke on elastase activity, 80–0053
effect of cigarette smoke on release of a chemotactic factor for polymorphonuclear leukocytes, 80–1118
effect of marihuana vs. tobacco smoke inhalation in rats, 80–1626
effect of smoke inhalation in rats, 80–0893
effect of smoke inhalation on enzyme activity in rats, 80–0041
effect of smoke inhalation on metabolism and phagocytosis in rats, 80–0042
effect of smoke inhalation on microperoxisomes in rats, 80–1618
effect of smoke inhalation on migration in mice, 80–0058
effect of smoke inhalation on recruitment in lungs in mice, 80–0059
effect of smoking, 80–0769
effect of smoking on cell count in cryptogenic fibrosing alveolitis patients, 80–1415
effect of smoking on cell number, phagocytotic ability, and surface morphology, 80–1430
effect of smoking on morphology, 80–1114
effect of smoking on phagocytosis, membrane morphology, and migration ability, 80–0745
effect of tar content in cigarettes in rats, 80–1638
effect of tobacco smoke vs. marihuana inhalation in rats, 80–1343
effect of tobacco vs. tobacco substitute smoke in rats, 80–0732
effect on benzo(*a*)pyrene mutagenicity, 80–0050
elastase activity in dogs, 80–0047
elastase activity in smokers vs. nonsmokers, 80–0047, 80–1174
elastase, lactate dehydrogenase, and lysozyme activity in smokers vs. nonsmokers, 80–1112
glycosidase and protease activity in light smokers vs. heavy smokers vs. nonsmokers, 80–1655
metabolism in smokers vs. nonsmokers, 80–1115
in smoking vs. nonsmoking sarcoidosis patients, 80–0101, 80–0324
tetrazolium salt metabolism in smokers vs. nonsmokers, 80–1630

PULMONARY ALVEOLAR PROTEINOSIS
(*See also* **LUNG DISEASES**)
effect of smoking, 80–0745

PULMONARY ALVEOLI
effect of smoking on permeability, 80–0770

PULMONARY CLEARANCE
(*See also* **CILIARY ACTIVITY**)
effect of benzo(*a*)pyrene particle size in hamsters, 80–1345

effect of marihuana vs. tobacco smoke inhalation in rats, 80–1626
effect of smoke inhalation in rats, 80–1648
effect of smoking on permeability in alveoli, 80–0770

Pulmonary emphysema *See* **EMPHYSEMA**

PULMONARY FIBROSIS
(*See also* **LUNG DISEASES**)
antielastase activity of antitrypsin in lungs in smoking vs. nonsmoking patients, 80–0327
bronchoalveolar cell count in smoking vs. nonsmoking vs. ex-smoking patients, 80–1176
effect of smoke and uranium ore dust inhalation in animals, 80–0284
in smoking vs. nonsmoking dockyard workers in Great Britain, 80–0107
in smoking vs. nonsmoking vs. ex-smoking dockyard workers, 80–1419

Pulmonary function *See* **LUNG FUNCTION**

PULMONARY SURFACTANT
effect of smoke inhalation on yield in rats, 80–1123
effect of smoking on phospholipid levels, 80–0323

PYRIDINE CONTENT
in cigar smoke, effect of polymers, 80–0554
in cigarette smoke, effect of tobacco pyrolysis, 80–0708
determination in cigarette smoke condensate, 80–0018
in mainstream and sidestream cigarette smoke, 80–0015

PYRIDINES
effect of nicotine pyrolysis on formation, 80–0708
in lamina vs. midrib cigarette smoke condensates, 80–0027
in smoke condensate in tobacco varieties, 80–0876
in tobacco, 80–0871

Pyrolysis products *See* **TOBACCO PYROLYSIS PRODUCTS**

Pyrolysis temperature *See* **COMBUSTION TEMPERATURE**

RADIATION
determination of alpha activity in cigarette smoke condensate, 80–1597
determination of alpha activity in lungs in smokers vs. nonsmokers vs. ex-smokers, 80–1379
determination of uranium content in Indian cigarettes, 80–1596
effect of plutonium and smoking on bronchial epithelium, 80–0311
effect of smoking and dental X-ray exposure on laryngeal neoplasm risk, 80–0086
effect of smoking in lung neoplasm etiology, 80–0316

RADIUM-226
determination in cigarette smoke condensate, 80–1597

RECIDIVISM
attitudes and self-regulatory behavior and, 80–0152
in coronary disease intervention program, 80–0422
effect of behavior in cessation of smoking, 80–0169
effect of focused smoking, 80–0836
effect of multicomponent cessation method, 80–0837
effect of smoking habit and attitudes, 80–0817
effect of paraprofessional training and social support, 80–0434
in Five-Day Plan in Great Britain, 80–1805
in high- vs. low-fear appeal smoking cessation methods, 80–0171
self-control and, 80–0156
in self-help cessation of smoking methods, 80–0170
theory for relapse in cessation of smoking, 80–0440

Reconstituted tobacco *See* **TOBACCO, RECONSTITUTED**

Rectal neoplasms *See* **INTESTINAL NEOPLASMS**

REDUCTION OF SMOKING
(*See also* **CESSATION OF SMOKING; WITHDRAWAL CLINICS**)
effect of aversive therapy and group training, 80–1779
effect of coronary risk factor intervention program, 80–1726
effect of expectancy in self-control, 80–0413
effect of modification of smoking characteristics, 80–0816
effect of multicomponent treatment and maintenance strategies, 80–1499
methods, cigarette case with clockwork mechanism, 80–0661
methods, cigarette case with timer, 80–0663
methods, cognitive efficacy vs. symptom attribution, 80–1497
methods, locking safety device, 80–0665
methods, multicomponent, 80–1788
methods, nicotine fading vs. self-monitoring vs. American Cancer Society program, 80–1012
methods, pharmacological, 80–1057
methods, positive reinforcement, 80–0164
methods, subliminal perception, 80–1506
methods, thought-stopping, 80–0168
methods, time-delayed single cigarette dispenser, 80–0660
withdrawal symptoms and, 80–1802

REFLEXES
(*See also* **COUGH**)

effect of lobeline on cough in dogs, 80–0914
effect of nicotine and cotinine in rats, 80–0887
effect of nicotine on cough in dogs, 80–0914

RESPIRATION
(*See also* **LUNG FUNCTION; OXYGEN TRANSPORT; RESPIRATORY FUNCTION TESTS)**
effect of maternal smoking in fetus, 80–1223
effect of passive smoking on respiratory frequency, 80–1388
effect of smoking and stress, 80–1784
in heavy vs. light vs. nonsmokers, 80–1359

RESPIRATORY FUNCTION TESTS
(*See also* **LUNG FUNCTION; OXYGEN TRANSPORT; RESPIRATION)**
evaluation of sensitivity, 80–1171
evaluation of single breath nitrogen determination of residual volume, 80–1189
helium-oxygen spirometry in small airways disease diagnosis, 80–0942
in obstructive airway disease diagnosis, 80–1411
in obstructive airway disease prognosis, 80–0779
single breath nitrogen test in obstructive airway disease diagnosis, 80–0938
in small airways disease diagnosis, 80–1173, 80–1188, 80–1680
standards for nonsmokers, 80–1175

RESPIRATORY SYMPTOMS
(*See also* **COUGH)**
bronchial rales and cough in smokers vs. nonsmokers, 80–0774
effect of air pollution in smokers vs. nonsmokers, 80–0098
effect of air pollution on incidence in children in France, 80–1420
effect of cessation of smoking on incidence, 80–1441, 80–1577
effect of dust exposure in sisal workers, 80–0336
effect of familial smoking habit on incidence in smoking vs. nonsmoking adolescents in New Zealand, 80–0778
effect of parental smoking on incidence in children, 80–0802, 80–1416
effect of passive smoking on incidence in asthmatics, 80–1697
effect of passive smoking on incidence in children in France, 80–1420
effect of smoking habit on incidence, 80–1177
effect of smoking in adolescents, 80–0141
effect of smoking in asbestos workers, 80–0776
effect of smoking on incidence in welders, 80–1681
immunoglobulin levels in smokers vs. nonsmokers and, 80–1631
prevalence elicited by questionnaires, 80–0767
relative risk in smokers vs. nonsmokers, 80–1184
smokers vs. nonsmokers in Japan, 80–1429
smokers vs. nonsmokers in Norway, 80–1414
smokers vs. nonsmokers with α_1-antitrypsin deficiency, 80–0095, 80–0765
smoking, alcohol consumption, and housing in Denmark and, 80–1178
smoking and, 80–0767
smoking and incidence in lacquerers in Denmark, 80–1669
smoking and pneumoconiosis in coal miners and, 80–1689
in smoking vs. nonsmoking children, 80–1754
in smoking vs. nonsmoking civic, grain elevator, and sawmill workers, 80–1167
in smoking vs. nonsmoking cosmetologists, 80–0775
in smoking vs. nonsmoking dairy farmers and factory workers, 80–0936
in smoking vs. nonsmoking drywall construction workers, 80–0326
in smoking vs. nonsmoking wire mill workers, 80–1690

RESPIRATORY SYSTEM
(*See also* **BRONCHI; LARYNX; LUNGS; PHARYNX; TRACHEA)**
distribution of polonium-210 in smokers vs. nonsmokers vs. ex-smokers, 80–0257
effect of *N*-nitrosodiethylamine on metabolism in pregnant hamsters, 80–1134
effect of cigarette smoke inhalation, 80–1687
effect of cigarette smoke inhalation on nitrosamine retention, 80–0718
effect of nicotine and tar content in cigarettes, 80–1687
effect of smoke inhalation in rats, 80–0719
lidocaine pharmacokinetics in lungs in smokers vs. nonsmokers, 80–0736

RESPIRATORY TRACT DISEASES
(*See also* **ASTHMA; BRONCHITIS; CHRONIC BRONCHOPULMONARY DISEASE; DYSPNEA; LUNG DISEASES; OBSTRUCTIVE AIRWAY DISEASES; SMALL AIRWAYS DISEASE)**
bronchoalveolar lavage cell count in smokers vs. nonsmokers vs. ex-smokers with cryptogenic fibrosing alveolitis, 80–1415
effect of parental smoking and air pollution on incidence in children, 80–0952
effect of smoking on lung function in adolescents and children, 80–1701
mortality in smokers vs. nonsmokers, 80–0290
mortality in smokers vs. nonsmokers vs. ex-smokers in the United States, 80–1395
smoking and, 80–0755
smoking and air pollution in etiology of, 80–0188
smoking and farmer's lung, 80–0109

smoking and ferruginous bodies in asbestos workers, 80–0943
in smoking vs. nonsmoking cement plant workers, 80–1683

RESPIRATORY TRACT INFECTIONS
effect of parental smoking on incidence in children and mothers, 80–0801
effect of smoke inhalation on immunity in mice, 80–1639
H. influenzae incidence in smoking vs. nonsmoking phosphate workers, 80–1702
histamine metabolism in smoking vs. nonsmoking asthma patients and, 80–0772
lung function in smoking vs. nonsmoking patients, 80–0337
smoking and carcinoembryonic antigen levels and, 80–0760

RESPIRATORY TRACT NEOPLASMS
(*See also* **BRONCHIAL NEOPLASMS; LARYNGEAL NEOPLASMS; LUNG NEOPLASMS; NASOPHARYNGEAL NEOPLASMS; PHARYNGEAL NEOPLASMS; TRACHEAL NEOPLASMS**)
carcinoembryonic antigen levels in diagnosis of, 80–0760
effect of benzo(*a*)pyrene particle size on induction in hamsters, 80–1345
effect of smoke inhalation on incidence in rats, 80–0719
induced by benzo(*a*)pyrene and dibenz(*a, j*)acridine in hamsters, 80–1659
induced by benzo(*a*)pyrene and diethylnitrosamine in hamsters, 80–0721
induced by benzo(*a*)pyrene and silicon dioxide in hamsters, 80–1659
induced by cigarette smoke and uranium ore dust inhalation in animals, 80–0284
induced by cigarette smoke in hamsters, 80–0891
mortality in male physicians in Finland, 80–0751
mortality in nickel workers, 80–0753
mortality in smoking vs. nonsmoking asbestos workers, 80–1394
smoking as risk factor, 80–0757
sputum cytology in smokers vs. nonsmokers in diagnosis of, 80–0759

RNA
(*See also* **DNA**)
acetaldehyde and formaldehyde incorporation in lung cells, 80–0904
benzo(*a*)pyrene metabolite binding in embryo cells in hamsters, 80–1096
benzo(*a*)pyrene metabolite binding in liver and lungs in rodents, 80–0727

SALIVA
effect of areca chewing on constituent levels and flow rate, 80–1133
effect of smoking on polymorphonuclear chemotaxis in smokers vs. nonsmokers, 80–1635
effect of tobacco chewing on constituent levels and flow rate, 80–1133
immunoglobulin levels in smokers vs. nonsmokers, 80–1611, 80–1631

SALIVARY GLANDS
effect of *N*-nitrosonornicotine on rodlet formation in mice, 80–1113

SEROTONIN
effect of antidepressants on uptake in platelets in smokers vs. nonsmokers, 80–1366

SERUM
antigen—antibody reactions induced by tobacco extracts in smoking vs. nonsmoking coronary disease patients, 80–0976
effect of smoking on polymorphonuclear leukocyte chemotaxis in smokers vs. nonsmokers, 80–1635
effect of tobacco extracts on enzyme activity in rats, 80–1127
immunoglobulin levels in smokers vs. nonsmokers, 801611

SEX BEHAVIOR
in smoking vs. nonsmoking male vs. female adolescents, 80–1518

SEX RATIO
(*See also* **WOMEN**)
absenteeism in smokers vs. nonsmokers, 80–1391
attitudes toward health risk of smoking, 80–1792
bronchitis in smokers vs. nonsmokers in Czechoslovakia, 80–0106
bronchitis risk in smokers vs. nonsmokers, 80–1184
cessation of smoking success in the United States, 80–0996
effect of codeine and promethazine on respiratory response to carbon dioxide in smokers vs. nonsmokers, 80–0947
effect of masculine and feminine brand names on cigarette flavor evaluation, 80–1763
effect of parental smoking on smoking habit in adolescents and children in Great Britain, 80–1527
effect of smoking, aging, and workload on heart rate, 80–1716
effect of smoking habit on bladder neoplasm risk, 80–1152
effect of smoking on cotinine and nicotine excretion, 80–1656
effect of smoking on lung neoplasm mortality rate in Great Britain, 80–0080
lung neoplasm mortality in Canada, 80–0298

lung neoplasms in Norway, 80–0304
respiratory symptom risk in smokers vs. nonsmokers, 80–1184
risk factors for coronary disease, 80–0117
risk factors for sudden coronary death, 80–0791
smoking and pancreatic neoplasms and, 80–0090
smoking as risk factor for cardiovascular disease mortality in blacks vs. whites, 80–1710
smoking characteristics, 80–1520
smoking habit, alcohol consumption, and drug abuse in adolescents in Puerto Rico, 80–0427
smoking habit and drug abuse in high school students in the United States, 80–0414
smoking habit and personality, 80–1776
smoking habit and personality in adolescents, 80–1514
smoking habit in academic psychologists in the United States, 80–0144
smoking habit in adolescents in Finland, 80–0426
smoking habit in alcoholic users of health services, 80–1238
smoking habit in Canada, 80–0410
smoking habit in Finland, 80–0416
smoking habit in hospital employees in the United States, 80–1516
smoking habit in Latin America, 80–0343
smoking habit in liver cirrhosis vs. liver neoplasm patients, 80–1399
smoking habit in Sweden, 80–0418
smoking habit in teachers and school children in Great Britain, 80–0402
smoking habit in the United States, 80–0420
smoking habit in university students in Romania, 80–1753
sputum analysis in smokers vs. nonsmokers in rural vs. urban areas in Yugoslavia, 80–0340
stroke in smokers vs. nonsmokers, 80–0361
tobacco consumption and lung neoplasms, 80–0421

SILICOSIS
mortality in gold miners in Australia, 80–0073

SKIN
DNA content in myocardial infarct patients, 80–1434
effect of nicotine on temperature, 80–0064
effect of smoking on resistance level, 80–0149

SKIN DISEASES
menthol cigarettes in dermatitis etiology, 80–0138
tobacco smoke in atopic dermatitis etiology, 80–1233

SKIN NEOPLASMS
(*See also* **CARCINOMA, EPIDERMOID**)
induced by cigarette smoke condensate in mice, 80–0740
induced by cigarette smoke condensate in mice, 80–0252
occupations in Great Britain and, 80–0932

SKIN TESTS
in atopic dermatitis diagnosis, 80–1233
tobacco extract allergy in smoking vs. nonsmoking coronary disease patients, 80–0976

SLEEP
smokers vs. nonsmokers, 80–0807

SMALL AIRWAYS DISEASE
(*See also* **OBSTRUCTIVE AIRWAY DISEASES; RESPIRATORY TRACT DISEASES**)
respiratory function tests in diagnosis of, 80–0942, 80–1173, 80–1188, 80–1680
smokers vs. nonsmokers, 80–1421
in smoking vs. nonsmoking firefighters, 80–1185
in smoking vs. nonsmoking vs. ex-smoking bronchitics, 80–1422

SMOKE, CIGAR
(*See also* **SMOKING, CIGAR**)
effect of polymers on constituent levels, 80–0554
effect of reconstituted tobacco foam on tar content, 80–0497
effect on carboxyhemoglobin levels in lung disease patients, 80–0353

SMOKE, CIGARETTE
(*See also* **SMOKING**)
analysis by computer processing, 80–0699
aromatic hydrocarbon, nicotine, and tar content in green vs. pale yellow tobacco varieties, 80–0709
ashtray for filtration, 80–0680
carcinogenicity in bacterial vs. mouse skin response tests, 80–0251
cigarette lighter-extinguisher to reduce air pollution, 80–0670
Cytrel vs. tobacco toxicity tests, 80–1337
determination of acetic and formic acid content, 80–1087
determination of acids and phenols in semivolatile fraction of tobacco varieties, 80–1598
determination of ammonia, nicotine, and tar in low-profile vs. normal harvest tobacco, 80–0207
determination of carbon monoxide content, 80–0698
determination of components in condensates from lamina and midrib cigarettes, 80–0027
determination of components in total particulate matter, 80–0026
determination of constituents of water soluble condensate fraction, 80–0018
determination of cyanide content, 80–1075
determination of ethephon metabolites, 80–0260
determination of gas phase constituents in Canadian brands, 80–1603
determination of hydrogen sulfide content in gas phase by automated colorimetry, 80–0691
determination of nitric oxide content, 80–0032

determination of nitrogen dioxide content, 80–0032
determination of phenol content, 80–1079
determination of phenol content in condensate, 80–0037
determination of reactivity with thiols, 80–1602
determination of trace metals in particulate matter and gas phase, 80–0034
distribution of polonium-210 label in lungs, 80–0257
effect of air dilution on components, 80–0234
effect of alumina and silica filters on constituents, 80–0505
effect of calcium-alginate tobacco substitute on tar content, 80–0645
effect of carboxymethyl cellulose filters on nicotine and tar levels, 80–0511
effect of cellulose acetate filters on nicotine and tar levels, 80–0538
effect of cellulose and coniferous bark particle filters on constituents, 80–0543
effect of cigarette characteristics and tobacco stalk position on nicotine and tar content, 80–1608
effect of cigarette paper on chemical composition, 80–0033
effect of fibrous filters on nicotine levels, 80–0541, 80–0542
effect of filter additives on constituent levels, 80–1017
effect of filters on chemical composition, 80–0029
effect of filtration and ventilation on constituents, 80–0839
effect of hydrogen peroxide-containing filter on aldehyde content, 80–1817
effect of inhaling on nitrosamine retention in respiratory system, 80–0718
effect of inhaling on respiratory system, 80–1687
effect of iron sulfate in filters on visibility of hydrogen sulfide removal, 80–0527
effect of laminated tobacco foil on constituent levels, 80–0484
effect of nicotine and tar content on smoking characteristics, 80–0400
effect of organic oils and powders in filters on tar levels, 80–0510
effect of oronasal exposure on carbon monoxide content in blood in monkeys, 80–1657
effect of paper perforation on constituent levels, 80–1264
effect of particle size on chemical characteristics of mainstream smoke, 80–1323
effect of permeable polymer membrane filter on constituent levels, 80–0483
effect of physical characteristics on composition, 80–0017
effect of polymer-coated carbon particle filters on aldehyde levels, 80–0495
effect of polystyrene resins on aldehyde and hydrogen cyanide levels, 80–0841
effect of protein granule filters on benzo(*a*)pyrene, phenol, and tar levels, 80–0523
effect of pyrolysis and transfer on formation of oxygenated compounds, 80–0222
effect of pyrolysis on chlorofluoromethane degradation product levels, 80–0702
effect of pyrolysis on nicotine degradation product levels, 80–0708
effect of resins and organic and metal compound filters on tar content, 80–0526
effect of smoking characteristics on carbon monoxide, nicotine, and total particulate matter delivery, 80–1501
effect of smoking characteristics on exposure, 80–1832
effect of thermoplastic polymeric filters on nicotine and tar levels, 80–0493
effect of tobacco pyrolysis and transfer rate on nitrogenous compound content, 80–0022
effect of tobacco shreds on tar content, 80–1262
effect of tobacco varieties on composition, 80–0223
effect of tube-like smoke vector on constituents, 80–0516
effect of ventilated filters on constituent levels, 80–1016
effect of wool filters on nicotine and tar levels, 80–0496
effect on alveolar macrophage release of a chemotactic factor for polymorphonuclear leukocytes, 80–1118
effect on angiotensin I conversion in lungs in rabbits, 80–0264
effect on α_1-antitrypsin activity in lungs in rats, 80–0266
effect on benzo(*a*)pyrene metabolism in lungs in rabbits, 80–1367
effect on conjunctiva in hamsters, 80–0882
effect on drug metabolism in liver microsomes in rats, 80–1620
effect on elastase activity of pulmonary alveolar macrophages, 80–0053
effect on elastase and antitrypsin levels in dogs, 80–0274
effect on immune system in mice, 80–0270
effect on lung function and respiratory symptoms in asthmatics, 80–1697
effect on lungs in rats, 80–0730
effect on phagocytosis, 80–1354
effect on α_1-proteinase inhibitor, 80–0053
effect on pulmonary alveolar macrophage recruitment in lungs in mice, 80–0059
effect on retina in cats, 80–0725
in emphysema etiology, 80–0053
interactive effect with influenza virus in lung neoplasm etiology, 80–0285
in laryngeal neoplasm induction in hamsters, 80–0883

mathematical model for estimating dilution using pressure drop measurements, 80–1078
mathematical model for predicting composition, 80–0017
mouthpiece for flow regulation, 80–0521
mutagenicity in *Salmonella typhimurium*, 80–0746
mutagenicity in lymphocytes and spermatozoa, 80–0714
nonvolatile and semivolatile components, 80–0232
optical properties, 80–0707
physical mechanisms of formation, 80–0227
in respiratory tract neoplasm induction in hamsters, 80–0891
as source of nitrosamines, 80–0878
valve for filter-controlled delivery, 80–0524

Smoke condensates *See* **TARS, CIGARETTE; TARS, TOBACCO**

SMOKE INHALATION
(*See also* **SMOKING**)
in arteriosclerosis etiology in baboons, 80–1649
in arteriosclerosis etiology in pigs, 80–0272
deposition and clearance in gastrointestinal and respiratory system in rats, 80–1648
detection of particulates in animal exposure systems, 80–0023
effect of chronic exposure on respiratory tract neoplasm incidence in rats, 80–0719
effect of dust on free lung cells in guinea pigs, 80–1380
effect of genotype on behavior in mice, 80–1334
effect of genotype on nicotine levels in mice, 80–1334
effect of tar content in cigarettes on pulmonary alveolar macrophages in rats, 80–1638
effect of tobacco vs. tobacco substitutes on pulmonary alveolar macrophages in rats, 80–0732
effect of uranium ore dust on pulmonary disease incidence in animals, 80–0284
effect of vitamin A deficiency on larynx in hamsters, 80–0901
effect on α_1-antitrypsin activity in lung in rats, 80–0266
effect on arteries in pigs, 80–1647
effect on aryl hydrocarbon hydroxylase activity in kidney and lungs in rats, 80–0912
effect on aryl hydrocarbon hydroxylase activity in liver and lungs in rats, 80–1097
effect on aryl hydrocarbon hydroxylase activity in lungs in rats, 80–1122
effect on body weight in hamsters, 80–1141
effect on calcitonin in rats, 80–0805
effect on elastase and antitrypsin levels in dogs, 80–0274
effect on elastic recoil in lungs in rats, 80–1372
effect on enzyme activity in aorta, heart, and lungs in rats, 80–1350
effect on enzyme activity in blood in rats, 80–1350
effect on enzyme activity in kidneys and lungs in rats, 80–1107
effect on epoxide hydrase activity in liver and lungs in rats, 80–1097
effect on fetus in rats, 80–0799
effect on immunity to repiratory tract infections, 80–1639
effect on lungs in rats, 80–0730
effect on microperoxisomes in pulmonary alveolar macrophages in rats, 80–1618
effect on nasal mucosa in hamsters, 80–1094
effect on nicotine metabolism in rats, 80–1652
effect on phagocytosis by pulmonary alveolar macrophages in rats, 80–0042
effect on protein synthesis in lungs in rats, 80–1346
effect on pulmonary alveolar macrophage enzyme activity in rats, 80–0041
effect on pulmonary alveolar macrophage recruitment in lungs in mice, 80–0059
effect on pulmonary alveolar macrophages in rats, 80–0042, 80–0893, 80–1343, 80–1626
effect on pulmonary macrophage migration in mice, 80–0058
effect on pulmonary surfactant yield in rats, 80–1123
effect on retina in cats, 80–0725
effect on surface tension of washing fluid in lungs in rats, 80–1372
effect on tracheobronchial epithelium in guinea pigs, 80–0717
in laryngeal neoplasm induction in hamsters, 80–1141

SMOKE STREAMS
determination of ethephon metabolites in mainstream vs. sidestream, 80–0260
distribution of tobacco constituents in mainstream vs. sidestream, 80–1332
effect of air resistance and cigarette paper porosity on nicotine content in mainstream, 80–0246
effect of cigarette periphery and rod on formation, 80–0028
effect of particle size on chemical characteristics of mainstream smoke, 80–1323
effect of tobacco pyrolysis on nicotine content in sidestream, 80–0708
methoprene content, 80–1266
pyridine content of filter vs. nonfilter cigarettes, 80–0015

SMOKE, TOBACCO
(*See also* **SMOKE, CIGAR; SMOKING; SMOKING, PIPE**)
antigen—antibody reactions in rodents, 80–1636
in atopic dermatitis etiology, 80–1233
bibliography of lead-210 and polonium-210 carcinogenicity, 80–1385
cartridge for filtration in pipes, 80–0679

definition of "condensate" vs. "tar", 80–0465
determination of dihydro-γ-pyrones in Burley vs. Oriental vs. Virginia varieties, 80–1076
effect of agricultural practices on constituent levels, 80–0208
effect of agricultural practices on tar content, 80–0687
effect of ascorbic acid in tobacco on nitrogen dioxide and nitrosamine content, 80–0557
effect of erythorbic acid in tobacco on nitrogen dioxide and nitrosamine content, 80–0557
effect of fine and rough metal in mouthpiece on constituent levels, 80–0671
effect of gamma alumina filler in cigarette paper on constituents, 80–0556
effect of manufacturing practices on composition, 80–0223
effect of metal—amine complex and polyalkylene oxide in filters on hydrogen cyanide levels, 80–0514
effect of metal nitrates and zinc oxide on polycyclic aromatic hydrocarbon levels, 80–0562
effect of metal salt—amine complexes on cyanide content, 80–0564
effect of methylated silica—water filter on quality, 80–0504, 80–0506
effect of microporous styrene polymer filters on tar reduction, 80–0529
effect of palladium and ammonium salts on aromatic hydrocarbon content, 80–0563
effect of palladium on aromatic hydrocarbon levels, 80–0545
effect of plant density on constituent levels, 80–0874
effect of reconstitution vs. expansion processes, 80–0174
effect of selective breeding on constituent levels, 80–0867
effect of silicic acid—polystyrene filter on amine and nitrosamine content, 80–1813
effect of tobacco leaf components on composition, 80–0867
effect of tobacco varieties on constituent levels, 80–1086
effect of transition metals on constituent levels, 80–0566
effect of vitamin C on mitotic abnormality incidence in lung cells, 80–1637
effect on heart muscle in rats, 80–1640
effect on IgE production in smokers vs. nonsmokers, 80–0899
effect on pulmonary alveolar macrophages in rats, 80–0732
in laryngeal neoplasm induction in hamsters, 80–0883
mutagenicity in *Salmonella typhimurium*, 80–0746
pipe-like device for reuse and containment, 80–0675
as source of nitrosamines, 80–0878
transfer rate of nicotine from tobacco leaf, 80–1324

SMOKERS VS. NONSMOKERS
absenteeism in males vs. females, 80–1391
acinar cell dysplasia in pancreas, 80–1157
age at onset of menopause, 80–1230
alcohol consumption in university students in Australia, 80–0401
allergic response to tobacco extract in serum and skin in coronary disease patients, 80–0976
alpha$_1$-antitrypsin deficiency in bronchitics, 80–0105
alveolar macrophage release of a chemotactic factor for polymorphonuclear leukocytes, 80–1118
amount of pocket money in adolescents, 80–1774
angiotensin I convertase and lysozyme activity in pulmonary alveolar macrophages, 80–1174
antielastase activity of antitrypsin in lungs, 80–0327
antipyrine and paracetamol metabolism, 80–1103
antitrypsin activity in pulmonary alveolar macrophages, 80–0329
antitrypsin deficiency and neutrophil elastase as risk factors for lung function abnormalities, 80–0333
antitrypsin deficiency and respiratory symptoms, 80–0765
antitrypsin, α_2-globulin, and leukocyte levels, 80–1364
antitrypsin levels in dust-exposed workers, 80–1410
Apgar scores in infants of, 80–0130
apolipoprotein A-I levels in myocardial infarct patients, 80–1440
arrhythmia in healthy adults, 80–0958
aryl hydrocarbon hydroxylase activity in liver, 80–0712
aryl hydrocarbon hydroxylase activity in lung neoplasm patients, 80–1675
aryl hydrocarbon hydroxylase activity in placenta, 80–1225
asbestos-related lesions in dockyard workers in Great Britain, 80–0107
asbestosis in workers, 80–1696
asbestosis mortality in workers, 80–1145
asthma in α_1-antitrypsin patients, 80–0095
attitudes, 80–0146
attitudes of railroad workers toward restriction of smoking in the workplace, 80–1493
attitudes toward health effects of smoking, 80–0955, 80–1761
attitudes toward smoking and lung diseases, 80–0161
attitudes toward smoking as risk factor for lung neoplasms, 80–0926
attitudes toward smoking during social interaction, 80–0404
attributions about smoking, 80–0147
bacterial adherence to pharyngeal cells, 80–1685
bacterial flora in dental plaque, 80–1747
behavior of infants, 80–0136
benzo(*a*)pyrene metabolism in placenta, 80–1128
birth weight of infants, 80–0134

blood cholesterol levels, 80–1455
blood lipid and lipoprotein levels in adolescents, 80–0364
blood pressure, heart rate, and electrocardiogram, 80–0784
blood pressure in myocardial infarct patients, 80–0965
blood protein levels, 80–1664
blood sugar levels, 80–0975, 80–1629
blood viscosity, hematocrit, and hemoglobin levels, 80–0971
bone mineral content in diabetics, 80–1745
bronchial neoplasms in Hong Kong, 80–1670
bronchitis in α_1-antitrypsin deficient patients, 80–0095
bronchitis in asphalt workers, 80–1187
bronchitis in children, 80–1691
bronchitis in Czechoslovakia, 80–0106
bronchitis in dust-exposed workers, 80–0108
bronchitis in fertilizer plant workers, 80–0332
bronchitis incidence in copper smelter workers, 80–1686
bronchitis incidence in tea blenders, 80–1700
bronchoalveolar cell count in pulmonary fibrosis patients, 80–1176
bronchoalveolar lymphocytes and macrophages in sarcoidosis patients, 80–0324
byssinosis in sisal workers, 80–0336
cadmium and zinc levels in urine, 80–0261
cadmium, lead, and mercury levels in blood and placenta, 80–0386
cadmium levels, 80–1105, 80–1373
cadmium levels in blood, 80–1362
cadmium levels in blood and feces in battery factory workers, 80–0250
cadmium levels in blood and urine in jewelry factory workers, 80–0880
cadmium levels in blood, feces, tissues, and urine, 80–0269
cadmium levels in blood in myocardial infarct patients, 80–0366
cadmium levels in blood, tissue, and urine, 80–1634
cadmium levels in feces, 80–0895
caffeine pharmacokinetics, 80–1099
calcitonin levels, 80–1162
carbohydrate and lipid metabolism, and insulin requirements in diabetics, 80–1232
carbon monoxide exchange ratio in lungs, 80–1166
carboxyhemoglobin and thiocyanate levels in rural vs. urban areas, 80–1623
carboxyhemoglobin levels, 80–0885
carboxyhemoglobin levels and oxygen transport, 80–1359
carboxyhemoglobin levels in police officers, 80–0071
carboxyhemoglobin levels in welders, 80–1650
carcinoembryonic antigen and L-fucose levels in mammary neoplasm diagnosis, 80–1389
cardiovascular disease mortality, 80–0290
chest radiograph and lung function in dockyard workers, 80–1695
choice of bottle vs. breast feeding, 80–0159
cholesterol levels in Maoris in New Zealand, 80–0345
chromosome abnormalities in vinyl chloride workers, 80–1487
clomipramine blood levels and tolerance in depressed patients, 80–1368
coronary artery stenosis, 80–0118
coronary disease, 80–0115, 80–1723
coronary disease and hyperglycemia in Japan, 80–1216
coronary disease in Canada, 80–0355
coronary disease morbidity and mortality, 80–1717
coronary disease risk factors and stress, 80–1715
coronary disease risk factors and ventricular premature beats, 80–1712
cough in adolescents in France, 80–1412
delinquency and early marriage in males in Great Britain, 80–0417
depression in males vs. females, 80–1243
determination of alpha activity in lungs, 80–1379
determination of carbon monoxide content in expired air and blood, 80–0738
diabetic proteinuria and retinopathy, 80–1490
diazepam bioavailability, 80–1375
distribution of polonium-210 from cigarette smoke in lungs, 80–0257
drug abuse in university students in Nigeria, 80–0153
dysplasia, hyperplasia, and metaplasia in bronchi, 80–1159
ectopic and myomatous pregnancy incidence, 80–1735
effect of age on blood and plasma viscosity, 80–0788
effect of age on fibrinogen and hematocrit levels, 80–0788
effect of age on isoproterenol and propranolol responsiveness, 80–0913
effect of age on vision, 80–1483
effect of aging on bronchitis incidence, 80–1684
effect of aging on theophylline metabolism, 80–1617
effect of air pollution on bronchitis incidence in industrial workers, 80–1418
effect of air pollution on carboxyhemoglobin levels, 80–1621
effect of air pollution on carboxyhemoglobin levels in traffic policemen, 80–1614
effect of air pollution on lung function, 80–0092, 80–0098
effect of air pollution on lung function in industrial workers, 80–0763
effect of air pollution on respiratory symptoms, 80–0098

effect of alcohol consumption on bronchitis risk, 80–1413
effect of alcohol consumption on carcinoembryonic antigen levels in liver disease patients, 80–1335
effect of alcohol consumption on electroencephalogram, 80–1633
effect of alcohol consumption on mean red cell volume, 80–0961
effect of allopurinol on theophylline metabolism, 80–0263
effect of antidepressants on serotonin uptake in platelets, 80–1366
effect of artificial sweeteners on bladder neoplasm risk, 80–1398
effect of asbestos exposure on lung function and respiratory symptom incidence in workers, 80–0776
effect of asthma and bronchitis in infancy on lung function in children, 80–1431
effect of blood glucose levels on electrocardiogram abnormalities, 80–1445
effect of carbon monoxide exposure on carboxyhemoglobin levels in mechanics, 80–1622
effect of carbon monoxide on carboxyhemoglobin levels, 80–1198
effect of carbon monoxide on electrocardiogram, 80–1198
effect of carbon monoxide on red blood cell parameters in submarine patrolmen, 80–1663
effect of cimetidine and pirenzepine on healing rate in duodenal ulcer patients, 80–1482
effect of cimetidine in peptic ulcer patients, 80–0139
effect of codeine and promethazine on respiratory response to carbon dioxide in males vs. females, 80–0947
effect of coffee-drinking on DNA synthesis in leukocytes, 80–0271
effect of dietary fats on blood platelet function, blood pressure, and cholesterol levels, 80–0370
effect of enflurane and halothane on enzyme activity, 80–1106
effect of exercise on coronary disease incidence, 80–0972
effect of exercise on heart rate, 80–1195
effect of exercise on lung function, 80–1641
effect of fear arousal on attitudes and beliefs, 80–0810
effect of glutethimide on antipyrine metabolism, 80–1615
effect of health warning in cigarette advertisements on desire to smoke, 80–0822
effect of mitogens on lymphocyte transformation in asbestos workers, 80–0716
effect of nicotine on vigilance performance, 80–1532
effect of occupation on relative risk for neoplasms, 80–0082
effect of occupational exposure on hearing impairment incidence, 80–0990
effect of passive smoking on eyes, 80–1388
effect of practolol on prognosis in myocardial infarct patients, 80–0965
effect of smoke inhalation on lung function, 80–0102
effect of smoking on blood platelet aggregation, 80–0350
effect of smoking on blood pressure, heart rate, and electrocardiogram, 80–0784
effect of smoking on lung function, 80–1428, 80–1699
effect of smoking on polymorphonuclear leukocyte chemotaxis in saliva and serum, 80–1635
effect of sulfur dioxide on lung function in copper smelter workers, 80–1686
effect of tobacco leaf and smoke extracts on IgE production, 80–0899
effect of visual stimulation on electroencephalogram, 80–1632
effect of vitamin A on relative risk for lung neoplasms, 80–0089
effect of weight gain in pregnancy on placenta disorder incidence, 80–1475
elastase activity in pulmonary alveolar macrophages, 80–0047, 80–1174
elastase, lactate dehydrogenase, and lysozyme activity in pulmonary alveolar macrophages, 80–1112
elastic recoil pressure in lungs, 80–1432
electrocardiogram in electronics workers, 80–0963
electroencephalogram, 80–1632
emphysema incidence and severity, 80–0939
enzyme and protein content in bronchoalveolar lavage fluid, 80–1111
epinephrine and norepinephrine levels in urine in oral contraceptive users, 80–0750
erythrocyte count and volume, 80–0971
estrogen:creatinine ratio in pregnant women, 80–1737
extraversion, 80–1498
fatty streak incidence and conversion rates, 80–1457
fetal breathing movements, 80–0135
gastritis in peptic ulcer patients, 80–0806
gastrointestinal diseases in chemical workers, 80–1227
gingivitis and plaque formation, 80–0988
glossal central papillary atrophy in diabetics, 80–1742
glycoprotein levels, 80–0934
glycosidase and protease activity in pulmonary alveolar macrophages, 80–1655
health practices and mortality rates, 80–1392
hemoglobin affinity for oxygen, 80–0731
high density lipoprotein apoprotein levels, 80–0254
H. influenzae incidence in pharynx in phosphate workers, 80–1702
histamine metabolism in asthma patients, 80–0772
hypertension and obesity in Finland, 80–1056

hysterectomy prevalence, 80–1231
immunoglobulin levels and respiratory symptoms, 80–1631
immunoglobulin levels in grain handlers vs. teachers, 80–1348
immunoglobulin levels in saliva and serum, 80–1611
lead-210 and polonium-210 levels in bronchial epithelium, 80–0737
lead content in blood and urine in ore dressing plant workers, 80–1104
lead content in hair, 80–1339
leukocyte function, 80–0258
lidocaine uptake in lungs, 80–0736
life insurance pricing differentials, 80–0290, 80–0452
lipid levels in Maoris in New Zealand, 80–0345
lipoprotein levels in myocardial infarct patients and relatives, 80–0371
localized osteitis in dental surgery patients, 80–0808, 80–0992
locus of control scale, 80–0157
longevity, 80–0917
longevity in the United States, 80–1393
lung function, 80–0335, 80–0938, 80–0945, 80–0949, 80–1168, 80–1177, 80–1181, 80–1183, 80–1421, 80–1428
lung function and α_1-antitrypsin deficiency, 80–0095, 80–1417
lung function and forced random noise impedance parameters, 80–0331
lung function and obstructive airway diseases, 80–0779, 80–1169
lung function and respiratory symptoms, 80–0774
lung function and respiratory symptoms in welders, 80–1681
lung function in antitrypsin deficient patients, 80–0342
lung function in beryllium workers, 80–1698
lung function in bronchitics, 80–1422
lung function in cadmium workers, 80–0334
lung function in civic, grain elevator, and sawmill workers, 80–1167
lung function in coal miners, 80–0942
lung function in dairy farmers and factory workers, 80–0936
lung function in Eisenmenger syndrome patients, 80–1688
lung function in fertilizer plant workers, 80–0332
lung function in firefighters, 80–1185
lung function in gastroesophageal reflux patients, 80–0780
lung function in obstructive airway disease patients, 80–0322
lung function in obstructive lung disease patients, 80–0957
lung function in polychlorinated biphenyl-exposed electrical workers, 80–0954
lung function in respiratory tract infection patients, 80–0337
lung function in sisal ropemakers, 80–0093
lung neoplasm mortality in asbestos workers, 80–1145
lung neoplasm mortality in smelter workers in Sweden, 80–0314
lung neoplasm mortality rates in Sweden, 80–1149
lung neoplasms in nickel workers, 80–0305
lung neoplasms in uranium miners, 80–0079
lung neoplasms in women, 80–0930
malignant hypertension, 80–0128
manganese levels in blood, 80–0278
memory, 80–1009
mesothelioma and pulmonary fibrosis in dockyard workers, 80–1419
metabolism, 80–1656
mineral content in bone, 80–0805
morbidity, 80–0077
morbidity and mortality, 80–1057, 80–1305
morbidity and mortality of infants, 80–0134
morphology of lungs in cotton industry workers, 80–0948
mortality, 80–0077, 80–0290, 80–0917, 80–1057, 80–1390
mortality from coronary disease and myocardial infarct, 80–0115
mortality rates in asbestos workers, 80–1394
mortality ratios in the United States, 80–1395
mutagenicity of urine in *Salmonella typhimurium*, 80–1140, 80–1347
mutagens in urine, 80–0283
myeloperoxidase and alkaline phosphatase activity, 80–0258
myocardial infarct, 80–0115
myocardial infarct risk in coffee- drinking women, 80–1727
myocardial infarct risk in oral contraceptive users, 80–1718
myocardial reinfarction, 80–0789
nasal mucosal histopathological changes in nickel workers, 80–1408
nasal pathology in nickel workers, 80–0110
nephropathy incidence in diabetes mellitus patients, 80–0804
neuroticism, 80–1498
nicotine and cotinine levels in urine, 80–0288
obesity in Poland, 80–0994
obstructive airway disease in Fabry's disease patients, 80–1424
obstructive airway disease incidence in Micronesia, 80–0764
obstructive airway diseases in machine building workers, 80–1182
oral behavior, 80–0820
pepsin 1 secretion in peptic ulcer patients, 80–0993

peptic ulcer healing rate in patients treated with cimetidine and sucralfate, 80–1744
peptic ulcer site, 80–1489
personality and sex behavior in adolescents, 80–1518
personality in adolescents, 80–1774
personality in alcoholics, 80–0830
physical activity and postoperative thrombosis in patients, 80–1707
physiological and psychological effects of nicotine, 80–1357
physiological and psychological factors, 80–1505
placental characteristics in blacks vs. whites, 80–0391
placental ultrastructure, 80–1469
pleural plaques in asbestos-exposed patients, 80–0103
prepregnancy weight, 80–1481
protein content in lung lavage fluid, 80–1643
psychiatric risk factors in myocardial infarct patients, 80–0111
psychological risk factors for coronary disease, 80–0121
pulmonary alveolar macrophage cytology in sarcoidosis patients, 80–0101
pulmonary alveolar macrophage function and morphology, 80–0745
pulmonary alveolar macrophage inclusions in cryptogenic fibrosing alveolitis patients, 80–1415
pulmonary alveolar macrophage metabolism, 80–1115
pulmonary alveolar macrophages, 80–1430
pulmonary alveoli permeability, 80–0770
relative risk for bladder neoplasms in artificial sweetener users, 80–1158
relative risk for bronchitis and respiratory symptoms in males vs. females, 80–1184
relative risk for coronary disease, 80–0115
relative risk for coronary disease in relatives of myocardial infarct patients, 80–0126
relative risk for head and neck neoplasms, 80–0923
relative risk for intermittent claudication in hypertensives, 80–0368
relative risk for lung neoplasms, 80–0299, 80–0310
relative risk for lung neoplasms in occupationally exposed workers, 80–0081
relative risk of lung neoplasms in Czechoslovakia, 80–0106
relative risk of maternal smoking for high-risk pregnancy, 80–0383
renal artery stenosis in hypertensives, 80–0360
respiratory function tests in diagnosis of small airways disease, 80–1173, 80–1680
respiratory symptoms and sputum cytology in cosmetologists, 80–0775
respiratory symptoms in children, 80–1754
respiratory symptoms in civic, grain elevator, and sawmill workers, 80–1167
respiratory symptoms in drywall construction workers, 80–0326
respiratory symptoms in Japan, 80–1429
respiratory symptoms in Norway, 80–1414
respiratory symptoms in wire mill workers, 80–1690
respiratory tract disease mortality, 80–0290
respiratory tract diseases and lung function in adolescents and children, 80–1701
respiratory tract diseases in cement plant workers, 80–1683
seat-belt use, 80–1761
sister chromatid exchange rate in lung neoplasm patients, 80–0723
sister chromatid exchange rates, 80–1616
skeletal muscle characteristics, 80–0277
skin neoplasms in workers in Great Britain, 80–0932
sleep difficulty, 80–0807
smoking habit of significant others, 80–1243
sperm density and motility in infertile patients, 80–1480
spirometry in diagnosis of obstructive airway diseases, 80–0104
spontaneous abortions, 80–0133
sputum analysis in rural vs. urban areas in Yugoslavia, 80–0340
sputum cytology in respiratory tract neoplasm diagnosis, 80–0759
stroke incidence, 80–0361
subarachnoid hemorrhage, 80–0128
sudden vs. nonsudden death after myocardial infarct, 80–0123
taste acuity, 80–1484
tetrazolium salt metabolism in pulmonary alveolar macrophages, 80–1630
theophylline dosage requirements, 80–0056
theophylline metabolism, 80–0055
thiocyanate levels in blood, 80–0870, 80–0885, 80–1098
thiocyanate levels in mechanics, 80–1622
thioether levels in urine, 80–1140
tracheobronchial epithelial multinucleation in neoplasm patients, 80–0758

SMOKING
(*See also* **PASSIVE SMOKING; SMOKE, CIGAR; SMOKE, CIGARETTE; SMOKE INHALATION; SMOKE, TOBACCO; SMOKING, BIDI; SMOKING, CIGAR; SMOKING, MATERNAL; SMOKING, PARENTAL; SMOKING, PATERNAL; SMOKING, PIPE**)
advertising and, 80–1058, 80–1560
air pollution and lung neoplasms and, 80–0309
and air pollution in respiratory tract disease etiology, 80–0188
alcohol consumption and drug metabolism and, 80–1581

alcohol consumption and esophageal neoplasms and, 80–1153
alcohol consumption and fire mortality and, 80–1838
alcohol consumption and mortality and, 80–1142
alcohol consumption, housing, and respiratory symptoms in Denmark and, 80–1178
alcohol consumption, laryngeal neoplasms, and occupational exposure and, 80–0919
antitrypsin deficiency and obstructive airway diseases and, 80–0100, 80–1172
arousal mechanisms and, 80–1780
arrhythmia and, 80–1438
in arteriosclerosis and cardiovascular disease etiology, 80–1458
arteriosclerosis and personality and, 80–0970
in arteriosclerosis etiology, 80–1439, 80–1453
asbestos exposure and lung neoplasms and, 80–0929
and asbestos in neoplasm etiology, 80–0713
asymptomatic hyperglycemia and coronary heart disease and, 80–1730
attitudes of smoking vs. nonsmoking railroad workers toward restriction in the workplace, 80–1493
attitudes toward health effects in smokers vs. nonsmokers, 80–0955
attitudes toward health risk in males vs. females, 80–1792
attitudes toward restriction in the United States, 80–1839
attitudes toward risk factor for lung neoplasms in smokers vs. nonsmokers, 80–0926
biliary reflux and, 80–1228
blood sugar and coronary disease and, 80–1204, 80–1713, 80–1729, 80–1731
bronchitis and, 80–0099, 80–1156
bronchogenic carcinoma and, 80–0857, 80–1156
bronchogenic carcinoma incidence in France and, 80–1673
carcinoembryonic antigen levels and respiratory tract infections and, 80–0760
cardiovascular disease mortality and, 80–1445
cardiovascular disease mortality in Mormons vs. non-Mormons and, 80–0119
cardiovascular disease mortality in Switzerland and, 80–0076
cardiovascular disease mortality rates in the United States and, 80–0752
cardiovascular disease risk factor clustering, 80–1709
cardiovascular diseases and, 80–0792, 80–0857, 80–0959
as cause of fires in upholstered furniture, 80–0858
in chronic bronchopulmonary disease etiology, 80–0852
coffee consumption and morbidity in Yugoslavia and, 80–0918
and coffee consumption as cocarcinogens, 80–0085
colonic neoplasm mortality in Great Britain and, 80–0294
coronary disease and, 80–0127, 80–1711
coronary disease and hyperglycemia and, 80–1219
coronary disease and post-load blood sugar levels and, 80–1213
coronary disease and socioeconomic status and, 80–1203
coronary disease mortality trends in the United States States and, 80–1721
in coronary heart disease etiology, 80–0347
death certificates and, 80–1584
disease-specific mortality rates and, 80–0755
effect of adrenergic agonists on tracheal mucosa in bronchitics and, 80–0950
effect of aging and workload on heart rate, 80–1716
effect of aging on ischemic heart disease mortality risk, 80–1464
effect of anesthetics on sister chromatid exchange rates in lymphocytes in operating room personnel, 80–1119
effect of artificial sweeteners on bladder neoplasm risk, 80–1161, 80–1164
effect of asbestos exposure on chest radiograph in workers and, 80–0937
effect of beta adrenergic receptor blockers on blood circulation, blood pressure, and heart rate, 80–1465
effect of beta adrenergic receptor blockers on catecholamine levels in blood, 80–1465
effect of blood sugar on coronary disease mortality rates, 80–1200
effect of dust exposure in grain elevator and sawmill workers, 80–1167
effect of ethnic group and socioeconomic status on coronary disease risk, 80–1448
effect of exercise on prognosis in myocardial infarct patients and, 80–0968
effect of glucose tolerance on electrocardiogram and, 80–0962
effect of marihuana on learning, 80–1768
effect of mouthwash on teeth staining, 80–0393
effect of obesity on mortality, 80–1143
effect of occupational exposure on lung neoplasm risk, 80–1154
effect of pindolol on fibrinolysis and platelet adhesion, 80–1720
effect of pindolol on free fatty acid and glucose levels in blood, 80–1720
effect of plutonium irradiation on bronchial epithelium, 80–0311
effect of school regulations in adolescents, 80–1412
effect of stress on heart rate and respiration, 80–1784
effect of urinary pH on psychomotor performance, 80–1530
effect of vitamin A on relative risk for lung neoplasms in heavy vs. light smokers, 80–0089

effect on δ-aminolevulinic acid dehydratase activity, 80–0749
effect on α_1-antitrypsin activity in blood, 80–0266
effect on arteriosclerosis change rate, 80–1706
effect on behavior, 80–0860
effect on blood cholesterol and triglyceride levels, 80–1218
effect on blood lipids in oral contraceptive users, 80–0379
effect on blood pressure, 80–1444
effect on blood pressure, electrocardiogram, and heart rate in heavy- vs. light- vs. ex-smokers, 80–0785
effect on body weight in women, 80–1485
effect on bronchitis incidence in dust-exposed workers, 80–0341
effect on bronchoalveolar lavage cell count in cryptogenic fibrosing alveolitis patients, 80–1415
effect on bronchogenic carcinoma incidence, 80–0921
effect on byssinosis incidence in textile workers, 80–0771
effect on cadmium and lead levels in blood, 80–0749
effect on cadmium levels in blood in coronary disease patients, 80–0362
effect on carboxyhemoglobin levels, 80–0973
effect on cardiovascular disease risk, 80–1462
effect on cardiovascular disease risk in oral contraceptive users, 80–1719
effect on cardiovascular mortality ratios, 80–0916
effect on cardiovascular mortality risk in hypertensives, 80–1437
effect on cholesterol levels, 80–0793
effect on contingent negative variation, 80–1496
effect on coronary disease incidence, 80–1714
effect on coronary disease incidence in women in Great Britain, 80–0983
effect on coronary disease mortality rates, 80–0293
effect on coronary disease prevalence in Japanese in the United States, 80–1452
effect on coronary disease risk, 80–0966, 80–1201, 80–1454
effect on coronary disease risk in housewives vs. working women, 80–0787
effect on diazepam metabolism, 80–1108
effect on 2, 3-diphosphoglycerate levels in blood, 80–0726
effect on echocardiographic examination, 80–1433
effect on elastic recoil pressure in lungs, 80–1432
effect on electroencephalogram, 80–1610, 80–1633
effect on erythrocyte deformability, 80–1344
effect on erythrocytes, 80–1449
effect on eye movement, 80–1496
effect on finger tremor, 80–1124
effect on fire incidence in California, 80–0453
effect on gastric secretion, 80–1226
effect on health status score, 80–1309
effect on heart function in coronary disease patients, 80–1436
effect on heart rate, 80–0149
effect on hematocrit, 80–0973
effect on high density lipoprotein apoprotein levels, 80–0254
effect on high density lipoprotein cholesterol levels, 80–0066
effect on immune system, 80–0462
effect on intermittent claudication postoperative prognosis, 80–1217
effect on ischemic heart disease mortality rates, 80–1207
effect on 17-ketosteroid content in urine, 80–1488
effect on laryngeal neoplasm mortality rates in asbestos workers, 80–1668
effect on learning and memory, 80–1781
effect on learning, memory, and psychomotor performance, 80–1777
effect on lung function, 80–0328, 80–0768, 80–0781, 80–1129
effect on lung function in cotton mill workers, 80–0766
effect on lung function in smokers vs. nonsmokers, 80–1699
effect on lung neoplasm incidence in blacks vs. whites, 80–1147
effect on lung neoplasm mortality in asbestos workers, 80–1668
effect on lung neoplasm risk in iron foundry workers, 80–0318
effect on lung neoplasm risk in uranium miners, 80–0079
effect on mortality rates in asbestos workers, 80–1394
effect on mortality rates in nickel workers, 80–0753
effect on mortality rates in older males, 80–0754
effect on mortality rates in physicians in Finland, 80–1666
effect on mortality rates in the United States, 80–0075, 80–0291
effect on mortality risk in hypertensives, 80–1437
effect on multiple neoplasm risk in mouth neoplasm patients, 80–0315
effect on muscle tension, 80–0149
effect on muscle tonus, 80–0044
effect on nasopharyngeal neoplasm risk, 80–1674, 80–1677
effect on neoplasm incidence in occupationally exposed workers, 80–0312
effect on neoplasm mortality rates, 80–0928
effect on neoplasm mortality ratio in Japan, 80–0302
effect on neurophysin levels in blood, 80–0259
effect on phenytoin metabolism in epileptics, 80–1342
effect on phosphatase activity in blood, 80–1488
effect on plasma vasopressin and human neurophysin levels, 80–0140

effect on prognosis for hypoxemia in cholecystectomy patients, 80–1179
effect on prognosis in coronary bypass patients, 80–1205
effect on prognosis in myocardial infarct patients, 80–0363, 80–0365, 80–0965
effect on prostaglandin synthesis and reactive hyperemia, 80–0796
effect on prostate and testes function, 80–1488
effect on psychomotor performance, 80–0429, 80–1004, 80–1781, 80–1793
effect on pulmonary alveolar macrophage phagocytosis, membrane morphology, and migration ability, 80–0745
effect on pulmonary alveolar macrophages, 80–0769, 80–1114
effect on pulmonary surfactant phospholipid levels, 80–0323
effect on relative risk for bladder and lung neoplasms, 80–0762
effect on relative risk for neoplasms, 80–0313
effect on risk for lung neoplasms in shipyard workers, 80–1208
effect on skin resistance level, 80–0149
effect on social behavior in male psychiatric patients, 80–0425
effect on spermatozoa count and mobility, 80–1488
effect on teeth, 80–0396
effect on theophylline metabolism, 80–0268
effect on thromboembolism incidence in myocardial infarct patients, 80–0351
effect on tonsillopharyngitis incidence in families in Canada, 80–1180
effect on uric acid levels, 80–1624
effect on urinary tract neoplasm incidence in phenacetin abusers, 80–1400
emphysema and, 80–0097
enzyme activity in lungs in lung neoplasm patients and, 80–1125
farmer's lung and, 80–0109
fire-related injuries and mortality, 80–0470
gastroesophageal reflux and, 80–0389
health effects, 80–1054, 80–1057
health effects of pesticide residues in tobacco, 80–0461
hypertension and, 80–1202, 80–1463
IgD and IgG levels and, 80–0279
interactive effect with alcohol consumption on laryngeal neoplasm risk, 80–0086
interactive effect with alcohol consumption on neoplasm risk, 80–1403
interactive effect with alcohol consumption on relative risk for head and neck neoplasms, 80–0923
interactive effect with asbestos on asbestosis incidence, 80–0777
interactive effect with asbestos on lung neoplasm mortality rates, 80–1405
interactive effect with dental X-ray exposure on laryngeal neoplasm risk, 80–0086
interactive effect with hypertension and oral contraceptives on myocardial infarct risk, 80–1215
interactive effect with occupational exposure in lung neoplasm etiology, 80–0081
interactive effect with occupational exposure on lung neoplasm risk, 80–1401
interactive effect with oral contraceptives on cardiovascular disease incidence, 80–0354, 80–0372
interactive effect with oral contraceptives on cardiovascular disease mortality, 80–0377
interactive effect with oral contraceptives on cardiovascular disease risk, 80–0352
interactive effect with oral contraceptives on hemorrhagic stroke risk, 80–0803
intestinal neoplasms and, 80–0927
ischemia in women and, 80–0369
in ischemic heart disease etiology, 80–0796
as ischemic heart disease risk factor in males in Scotland and Sweden, 80–0359
laryngeal and lung neoplasm mortality in asbestos workers and, 80–0292
in laryngeal neoplasm etiology, 80–0091
laryngeal neoplasms and, 80–0295
lip neoplasms and, 80–0308
and lipid abnormality in myocardial infarct patients, 80–0346
lung function and, 80–1171
lung function and obstructive airway diseases and, 80–0773
lung function in adolescents and, 80–1693
lung function in antitrypsin deficient patients and, 80–0946
lung function in arthritis patients and, 80–0941
in lung neoplasm etiology, 80–0920, 80–1148
lung neoplasm mortality in Great Britain and, 80–0080
lung neoplasm mortality rates in beryllium-exposed workers and, 80–0931
lung neoplasms and, 80–0078, 80–0303, 80–0761
lung neoplasms and, 1980 American Cancer Society report, 80–0078
lung neoplasms and metronidazole and, 80–0756
lung neoplasms and occupational exposure and, 80–0304
lung neoplasms and sex ratio and, 80–0421
lung neoplasms in chloromethyl ether workers and, 80–0321
lung neoplasms in the Pacific Basin and, 80–1672
lung neoplasms in women and, 80–0182
effect on abruptio placentae and placenta praevia incidence, 80–1474

mathematical model for carbon monoxide content in blood, 80–1126
model for arteriosclerosis etiology, 80–1457
mortality in diabetics and, 80–1741
myocardial infarct in young adults and, 80–0346
myocardial infarct incidence in menopausal women and, 80–1704
nasopharyngeal neoplasms and, 80–0084, 80–0087
in neoplasm etiology, 80–0319
neoplasm mortality rates in the United States and, 80–1397
neoplasms and, 80–1151, 80–1402
obstructive airway diseases and, 80–0330, 80–1694
oral and pharyngeal neoplasms and, 80–1678
oral contraceptives and myocardial infarct in young women and, 80–0346
oral epithelial keratinization in leukoplakia patients and, 80–1486
in oral neoplasm etiology, 80–0091, 80–0317
in osteoporosis etiology, 80–0805
pancreatic neoplasms and, 80–0090
peptic ulcer and, 80–1746
as perceived cause of myocardial infarct, 80–0824
pharmaceutical label on drug metabolism and, 80–1582
pharmacological and psychological determinants, 80–1528
in pharyngeal neoplasm etiology, 80–0091
physiological and psychological effects, 80–1531
physiological effects, 80–1777
postoperative pain in alveolitis sicca dolorosa patients, 80–1743
radiation exposure in lung neoplasm etiology and, 80–0316
recommendations for prohibition in the workplace, 80–1297
recommendations of American Medical Association for restriction on airplanes, 80–1278
respiratory symptoms and, 80–0767
respiratory symptoms in lacquerers in Denmark and, 80–1669
respiratory tract neoplasm mortality rates in male physicians in Finland and, 80–0751
restrictions in hospitals in the United States, 80–1298, 80–1566
restrictions in Planned Parenthood clinics in the United States, 80–1764
restrictions in the workplace in the United States, 80–1285, 80–1495, 80–1833
as risk factor for arrhythmia, 80–0125
as risk factor for cardiovascular disease mortality in black vs. white males vs. females, 80–1710
as risk factor for cerebral infarction and transient ischemic attacks, 80–0795
as risk factor for coronary artery stenosis, 80–0348
as risk factor for coronary disease, 80–0114, 80–0373, 80–0974, 80–0980, 80–1214
as risk factor for coronary disease in nonagenarians, 80–0349
as risk factor for coronary disease in women, 80–0787
as risk factor for myocardial infarct, 80–0783
as risk factor for obstructive airway diseases, 80–1169
as risk factor for respiratory tract neoplasms, 80–0757
as risk factor for stroke, 80–0357
as risk factor for sudden death in coronary disease patients, 80–0791
as risk factor for tongue neoplasms, 80–0922
role of physicians in prevention of lung neoplasms in asbestos-exposed workers, 80–0935
secondary Poisson process models for carboxyhemoglobin levels, 80–1642
self-scoring risk test for coronary disease, 80–0113
small airways disease and, 80–1188
in thromboangiitis obliterans etiology, 80–0960
tooth discoloration and, 80–0392
vasoconstrictor prostaglandins in coronary artery spasm etiology and, 80–0967

SMOKING AND HEALTH
abstinence and altitude acclimatization and, 80–1575
attitudes in Great Britain, 80–1571
attitudes in the United States, 80–0848
attitudes of tobacco industry, 80–1053
in Austria, 80–1836, 80–1837
in Canada, 80–1059, 80–1826
Department of Health, Education, and Welfare objectives for 1990, 80–0471
in East Germany, 80–1279
effect of filter cigarettes, 80–1292
effect of genetic factors, 80–1064
Federal policy in the United States, 80–0848
Federal Trade Commission annual report, 80–0861
lifestyle intervention as preventive medicine, 80–1578
National Cancer Institute report on program for reduction of smoking related disease, 80–1060
in Nigeria, 80–1574
public affairs educational pamphlet, 80–1046
recommendation of National Institute for Occupational Safety and Health, 80–1281
recommendations of a WHO Expert Committee, 80–1290
recommendations of the WHO Expert Committee on Smoking Control, 80–1308
research funding, 80–1711
research grants by Council for Tobacco Research, 80–1847
research in Austria, 80–1570
Surgeon General's Report on Health Promotion and Disease Prevention, 80–1304, 80–1305
1979 Surgeon General's Report review, 80–1295
Third Report of the Royal College of Physicians, 80–0077

trends in manufacturing less hazardous cigarettes, 80–1565
trends in World Health Organization policy, 80–0469
in the U.S. Navy, 80–1564
in the U.S.S.R., 80–1277
WHO resolution, 80–0863
in Yugoslavia, 80–1299

SMOKING ARTICLES
cigarette filter holder, 80–0482, 80–0508, 80–0673
cigarette holder producing electric shock for cessation of smoking, 80–0666
cigarette lighter-extinguisher and smoke reducer, 80–0670
cigarette with mouthpiece for flow regulation, 80–0521
effect of ventilated cigarette holders on smoking characteristics, 80–1767
fluid-cooled pipe, 80–0678
liquid-filtered pipe, 80–0674, 80–0676, 80–0679, 80–1815
liquid-filtered pipe with ash receptacle, 80–0677
mouthpiece for progressive reduction of nicotine delivery, 80–0662
mouthpiece with fine and rough metal filters, 80–0671
pipe for use with fruit for tobacco aroma and flavor enhancement, 80–1806
pipe-like device with bag for smoke reuse and containment, 80–0675
pipe tamper and disc, 80–0672
simulated cigarette and holder for reduction of passive smoking in ex-smokers, 80–1830
ventilated cigarette holders, 80–1790

Smoking behavior *See* **BEHAVIOR; SMOKING CHARACTERISTICS; SMOKING HABIT**

SMOKING, BIDI
effect on lung neoplasm risk, 80–0299
effect on preleukoplakia incidence in India, 80–0989
in oral neoplasm etiology, 80–0317
thromboangiitis obliterans and, 80–0124

SMOKING CHARACTERISTICS
(*See also* **SMOKING CLASSIFICATION; SMOKING HABIT**)
assessment methods, 80–1762
effect of active charcoal filter on puff characteristics, 80–1765
effect of arousal manipulation in smoking taste test, 80–1755
effect of cigarette draw resistance, 80–1759
effect of cigarette holders vs. butt length on smoke exposure, 80–1832
effect of cigarette length on inhalation and puff rate, 80–1246
effect of cigarette length on puff parameters, 80–1751
effect of demographic and socioeconomic variables, 80–1520
effect of experimental and social conditions, 80–1500
effect of filter vs. nonfilter cigarettes on bladder neoplasm risk, 80–1152
effect of heroin administration on intercigarette intervals, 80–1005
effect of high- vs. low-delivery cigarettes, 80–1757
effect of inhaling on bladder neoplasm risk in pipe smokers, 80–1152
effect of inhaling on carbon monoxide content in alveolar air, 80–1785
effect of inhaling on carboxyhemoglobin levels in cigar and pipe smokers, 80–1302
effect of inhaling on lung function, 80–1190
effect of inhaling on lung neoplasm risk, 80–1679
effect of inhaling on mortality rates, 80–0851
effect of inhaling on nicotine levels in blood in cigar and pipe smokers, 80–1302
effect of inhaling on nitrosamine retention in respiratory system, 80–0718
effect of inhaling on respiratory system, 80–1687
effect of inhaling on thiocyanate levels in blood, 80–1098
effect of intravenous vs. inhaled nicotine, 80–1772
effect of less hazardous cigarettes, 80–0409
effect of neuroticism, 80–1532
effect of nicotine and tar content in cigarette smoke on puff duration, frequency, and volume, 80–0400
effect of nicotine content in cigarettes, 80–1760
effect of personality on nicotine intake, 80–1610
effect of puff characteristics on carbon monoxide, nicotine, and total particulate matter delivery, 80–1501
effect of social interaction, 80–0143
effect of tar content in cigarettes, 80–1785
effect of tar content on puff characteristics, 80–1750
effect of ventilated cigarette holders on compensatory response, 80–1790
effect of ventilated cigarette holders on puff number and rate, 80–1767
effect of verbal instructions on puff frequency and duration, 80–0816
effect on carbon monoxide content in alveolar air, 80–1760
effect on contingent negative variation, 80–1496
effect on longevity in the United States, 80–1393
in evaluation of less hazardous cigarettes, 80–0409
instrumentation for monitoring and duplicating, 80–1758

SMOKING, CIGAR
(*See also* **CIGARS; SMOKE, CIGAR**)
adults in the United States, 80–1522
alcohol consumption, housing, and respiratory symptoms in Denmark and, 80–1178

effect of inhaling on carboxyhemoglobin levels, 80–1302
effect of inhaling on mortality rates, 80–0851
effect of inhaling on nicotine content in blood, 80–1302
effect on blood cholesterol and triglyceride levels, 80–1218
effect on lung morphology in cotton industry workers, 80–0948
effect on nicotine levels in blood, 80–1137
effect on relative risk for bladder neoplasms, 80–1152

SMOKING CLASSIFICATION
(*See also* **SMOKING CHARACTERISTICS; SMOKING HABIT**)
attitudes toward lung neoplasm risk in smokers vs. nonsmokers, 80–0926
effect of cigar vs. cigarette vs. pipe smoking on bladder neoplasm risk, 80–1152
effect on morphology in lungs in cotton industry workers, 80–0948
effect on neoplasm mortality rates, 80–0928

SMOKING CONTROL PROGRAMS
(*See also* **CESSATION OF SMOKING; HEALTH EDUCATION**)
in acutely ill patients, 80–0433
effect of coronary disease intervention program on smoking habit in Finland, 80–0367
effect of health education on onset of smoking in children, 80–0408
health education in schools in Australia, 80–1526
in hospitals in the United States, 80–1566
Kaiser-Permanente Medical Care Program, 80–1803
life skills training for adolescents, 80–0811
in schools in East Germany, 80–1507
in schools in the United States, 80–1234
Stop Smoking Clinic, 80–0167
in the workplace in the United States, 80–1249, 80–1285, 80–1495

SMOKING HABIT
(*See also* **SMOKING CHARACTERISTICS; SMOKING CLASSIFICATION**)
academic psychologists in the United States, 80–0144
as addiction vs. arousal modulation, 80–1777
as addictive behavior, 80–1239, 80–1782
administrators in schools for health professionals in the United States, 80–1236
adolescents and adults in Canada, 80–0410
adolescents and adults in the United States, 80–0448
adolescents and children in secondary schools in Great Britain, 80–1527
adolescents and psychosocial variables in prediction of, 80–1492
adolescents in East Germany, 80–1507, 80–1515
adolescents in Ethiopia, 80–0995
adolescents in Finland, 80–0426
adolescents in France, 80–0424, 80–1412
adolescents in Great Britain, 80–1693
adolescents in Switzerland, 80–1244
adolescents in the United States, 80–0468
adolescents in West Germany, 80–1774
adults in Finland, 80–0416, 80–1056, 80–1220
adults in Great Britain, 80–0423
adults in Japan, 80–1766
adults in Sweden, 80–0981
adults in the United States, 80–0420, 80–0996, 80–1052, 80–1522
alcohol consumption and, 80–0155, 80–0827
alcohol consumption and cardiovascular diseases in physicians and, 80–0828
alcohol consumption and drug abuse in adolescents in Puerto Rico and, 80–0427
alcohol consumption and drug abuse in military recruits in Switzerland and, 80–1494
alcohol consumption in college students and, 80–1008
alcohol consumption in oral and pharyngeal neoplasm patients and, 80–0933
alcohol consumption in university students in Australia and, 80–0401
alienation and, 80–0157
arterial diseases and, 80–1196
assessment methods, 80–1762
athletes in Sweden, 80–0457
attitudes and motivation in university students in Romania, 80–1508
attitudes in adolescents in Great Britain, 80–1504
attributions of smokers vs. nonsmokers vs. ex-smokers, 80–0147
in Australia, 80–0467
behavior and, 80–0155
in bladder vs. lung neoplasm patients, 80–0307
in bronchial neoplasm patients, 80–0296, 80–1676
bronchitis and lung function and, 80–0338
bronchitis patients, 80–1155
bronchogenic carcinoma patients, 80–1155
cervix neoplasm patients, 80–1407
children and parents in Great Britain, 80–1754
children in France, 80–1000
children in Sweden, 80–0419
coal miners and gold miners in Australia, 80–0073
college students in the United States, 80–1778
as contagious behavior, 80–1503
coronary disease incidence in Aborigines in Australia and, 80–0344
coronary disease mortality in marathoners and, 80–0122
coronary disease patients, 80–1155
coronary disease patients in Canada, 80–0355
cotinine levels in blood as verification of self-report, 80–0430

dentists and physicians in Japan, 80–1766
as deviant behavior, 80–1839
diabetes mellitus patients, 80–0804
disability retirees in Australia, 80–1667
drug abuse in the elderly and, 80–0142
effect of advertising in Canada, 80–1826
effect of alcohol consumption and marihuana smoking, 80–1519
effect of antismoking campaigns in coronary disease intervention program in Belgium, 80–0969
effect of antismoking campaigns in France, 80–1834
effect of antismoking campaigns in adolescents in France, 80–0405
effect of cardiovascular disease risk factor reduction program in Israel, 80–1749
effect of cigarette consumption on bladder neoplasm risk in males vs. females, 80–1152
effect of cigarette consumption on lung function in obstructive lung disease patients, 80–0957
effect of cigarette consumption on recidivism, 80–0817
effect of cigarette length on consumption, 80–1246
effect of coronary disease intervention program in Belgium, 80–1447
effect of coronary disease intervention program in Finland, 80–0367
effect of daily consumption on respiratory symptom incidence and sputum production, 80–1177
effect of genetic factors, 80–1064
effect of health education in adolescents in Switzerland, 80–1787
effect of health education in the United States, 80–1511
effect of health education on onset in children, 80–0408
effect of heroin administration, 80–1005
effect of hypertension control program in college students, 80–1197
effect of intravenous vs. inhaled nicotine, 80–1772
effect of ischemic heart disease risk factor reduction program, 80–1229
effect of legislation in Norway, 80–0450
effect of life skills training on onset in adolescents, 80–0811
effect of nicotine, 80–0154
effect of nicotine and tar content in cigarettes, 80–0150, 80–0400
effect of nicotine content in cigarettes, 80–0459, 80–0829, 80–1352, 80–1777
effect of parental and peer smoking in adolescents, 80–1518
effect of parental attitudes in children, 80–1055
effect of parental smoking and school regulations in adolescents, 80–1412
effect of parental smoking in children, 80–1000
effect of parental smoking on adolescents, 80–0405
effect of participation in American Cancer Society Stop Smoking Program, 80–0436
effect of personality, 80–1498
effect of physicians' smoking habits, 80–0454
effect of role stress in female academic psychologists, 80–0144
effect of School Health Curriculum Project in adolescents in the United States, 80–1510
effect of significant others in adolescents, 80–1243
effect of smoking control programs in adolescents in the United States, 80–1234
effect of social influence and acceptability, 80–0148
effect of social interaction, 80–0143
effect of socioeconomic status, 80–0819
effect of spouse's smoking on cessation success, 80–1801
effect of stress, 80–0149
effect of stress in journalists, 80–0403
effect of tar content in cigarettes, 80–1294, 80–1750
effect of teachers' smoking in adolescents, 80–0405
effect of teachers' smoking on school children in Great Britain, 80–0402
effect of vomiting, 80–1779
effect of weight loss, 80–0991
effect on arteriosclerotic heart disease mortality rates, 80–1209
effect on coronary disease mortality rates, 80–1194
effect on glycosidase and protease activity in pulmonary alveolar macrophages, 80–1655
effect on longevity in the United States, 80–1393
effect on lung neoplasm risk, 80–1679
effect on mortality rates in physicians, 80–1144
effect on ventricular premature beat incidence, 80–1212
electrocardiogram abnormalities and, 80–1196
engineers in Finland, 80–1245
ethnic groups in Hawaii, 80–1402
gingivitis patients, 80–0397
health practices and, 80–0155, 80–0158
heart disease patients, 80–1451
high school students in the United States, 80–0414
high- vs. low-tar cigarette preference in female lung neoplasm patients, 80–0930
ischemic heart disease and myocardial infarct patients, 80–0979
in Latin America, 80–0343
lawyers and physicians in Rhode Island, 80–0997
life insurance pricing and, 80–0290
low-nicotine cigarettes and, 80–1528
lung neoplasm mortality in Austria and, 80–1150
lung neoplasm patients in Kenya, 80–0300
male vs. female alcoholic users of health services, 80–1238
male vs. female hospital employees in the United States, 80–1516

male vs. female liver cirrhosis vs. liver neoplasm patients, 80–1399
marihuana use in the United States and, 80–1523
medical school faculty in England, 80–1237
medical students in the U.S.S.R., 80–1240, 80–1242
medical students in the United States, 80–0814
mesothelioma patients, 80–1146
motivating factors, 80–0165
motivation and oral behavior, 80–0820
myocardial infarct patients, 80–1435
naval personnel in the United States, 80–1564
in New Zealand, 80–1402, 80–1671
nicotine and cotinine levels in urine as verification of, 80–0288
nicotine content in urine as verification of, 80–1646
in Nigeria, 80–1491, 80–1574
in Norway, 80–1006
nurses, 80–1775
nurses in New Zealand, 80–0160
nursing students, 80–1241
obstetrical inpatients, 80–0818
oral contraceptive users, 80–1463
pancreatic neoplasm patients, 80–1409
in the People's Republic of China, 80–1561
peptic ulcer patients, 80–0993
personality in children and, 80–1525
personality in high- vs. low-arousal smokers, 80–1771
personality in male vs. female adolescents and, 80–1514
personality in males vs. females and, 80–1776
physicians in Australia, 80–1502
physicians in Finland, 80–0751, 80–1245
physicians in New Zealand, 80–1509
pregnant women, 80–1470
print media readers and cigarette advertising in Australia, 80–1831
psychiatric clinic patients, 80–1049
psychiatric hospital patients and staff in Finland, 80–0411
psychophysiologic effects of nicotine, 80–0064
psychosomatic disorder patients, 80–0406
questionnaire for assessment as coronary disease risk factor, 80–0982
as risk-taking behavior, 80–0812
in schools in Ohio, 80–1770
self-categorization and cognitive dissonance, 80–1791
sex ratio in Sweden, 80–0418
in smokers accepting vs. rejecting smoking cessation treatment, 80–0145
smokers' self-perception and, 80–0146
social behavior in male psychiatric patients and, 80–0425
statewide mortality rates and, 80–0291
stomach neoplasm patients, 80–1163
technical university students in Norway, 80–1007
thromboangiitis obliterans patients, 80–0960, 80–1467
trends in cigarette consumption and arteriosclerosis mortality rate, 80–1450
Type A behavior in coronary disease patients and, 80–1705
in the U.S.S.R., 80–1277
university students in Nigeria, 80–0153
university students in Romania, 80–1753
in urban and suburban areas in Boston, 80–0158
white vs. colored adolescents and their parents in South Africa, 80–0141
women in the United States, 80–1569
young female angina pectoris and myocardial infarct patients, 80–1724

SMOKING MACHINES
cigarette puff flow, duration, and interval testing, 80–0669
detection of particulates, 80–0023

SMOKING, MATERNAL
abnormalities and fetal mortality and, 80–0984
drug exposure history in pregnancy and, 80–0129
effect of alcohol consumption on fetus, 80–1740
effect of diet on birth weight, 80–1478
effect of 48-hour abstinence from smoking on oxygen availability during pregnancy, 80–0720
effect of maternal weight gain on placenta disorder incidence, 80–1475
effect on Apgar scores in infants, 80–0130
effect on aryl hydrocarbon hydroxylase activity in placenta, 80–1225
effect on asthma incidence in smoking vs. nonsmoking adolescents in New Zealand, 80–0778
effect on attitudes and behavior in children, 80–1752
effect on birth height and weight, 80–1225
effect on birth weight, 80–0134
effect on birth weight and gestation, 80–0387, 80–1736
effect on birth weight and prematurity, 80–0801
effect on birth weight in blacks vs. whites, 80–0798
effect on birth weight, perinatal mortality, and infant morbidity and mortality, 80–1738
effect on blood vessels in placenta and umbilical cord, 80–1468
effect on breast feeding and lactation, 80–1479
effect on cadmium, lead, and mercury levels in blood and placenta, 80–0386
effect on cadmium, lead, and mercury levels in cord blood in neonate, 80–0386
effect on carboxyhemoglobin and hemoglobin levels during pregnancy, 80–1619
effect on carboxyhemoglobin levels in mother and infant, 80–0137
effect on childhood morbidity, 80–0134
effect on choice of bottle vs. breast feeding, 80–0159

effect on cleft lip and cleft palate incidence in infants, 80–0131
effect on estrogen:creatinine ratio in urine, 80–1737
effect on fetal and neonatal mortality rates, 80–1474
effect on fetal and placental disorder incidence, 80–1476
effect on fetal breathing movements, 80–0135, 80–1223
effect on fetal cardiovascular system, 80–1732
effect on fetal growth and prematurity, 80–1470
effect on fetal mortality, 80–0133, 80–1222
effect on fetal movement, 80–0987
effect on fetal reactivity, 80–0986
effect on fetus, 80–0799, 80–1222, 80–1734
effect on fetus and children, 80–0985
effect on fetus and neonate, 80–1471
effect on hematocrit in neonate, 80–0385
effect on immune system in umbilical cord blood, 80–0800
effect on infant behavior, 80–0136
effect on leukocyte count in cord blood, 80–1733
effect on low birth weight and prematurity incidence in neonate, 80–0382
effect on maternal body weight, 80–0387
effect on mother and fetus, 80–0384
effect on neonate, 80–1222
effect on oxygen transport during pregnancy, 80–1619
effect on perinatal mortality, 80–1222
effect on placental characteristics in blacks vs. whites, 80–0391
effect on placental ultrastructure, 80–1469
effect on prematurity incidence, 80–0381, 80–1472
effect on prepregnancy weight, 80–1481
effect on relative risk for high-risk pregnancy, 80–0383
effect on respiratory symptom incidence in children, 80–0802, 80–1416
effect on spontaneous abortion incidence, 80–0133
effect on sucking latency and pressure in neonates, 80–1473
effect on sudden infant death syndrome incidence, 80–1223
effect on thiocyanate levels in blood, 80–0385
epidemiological tools for risk assessment in pregnancy, 80–0388
fetal alcohol syndrome and, 80–0797
fetal mortality and, 80–0133
induced abortion and, 80–1736
obstetrical inpatients, 80–0818
and occupational exposure, effect on incidence of abortion and abnormalities, 80–0132
planned vs. unplanned pregnancy, 80–1524
pregnancy in oral contraceptive users and, 80–1739
spontaneous abortion and, 80–0133
teratogenesis and, 80–1735

SMOKING, PARENTAL
effect on allergy-related illnesses in mothers, 80–0801
effect on asthma incidence in adolescents in New Zealand, 80–0778
effect on attitudes and behavior in children, 80–1752
effect on birth weight and neonatal mortality, 80–1224
effect on fetus and neonate, 80–1471
effect on respiratory disease prevalence in children, 80–0952
effect on respiratory symptom incidence in children, 80–0801, 80–1416
effect on respiratory tract infection incidence in children and mothers, 80–0801
effect on smoking habit, 80–0147
effect on smoking habit in adolescents, 80–0405, 80–1412, 80–1518
effect on smoking habit in adolescents and children in Great Britain, 80–1527
effect on smoking habit in adolescents in South Africa, 80–0141
effect on smoking habit in children, 80–1000, 80–1055, 80–1583

SMOKING, PATERNAL
effect on asthma incidence in adolescents in New Zealand, 80–0778
effect on birth weight and neonatal mortality, 80–1224
effect on fetus and neonate, 80–1471
effect on respiratory symptom incidence in children, 80–0802
perinatal mortality and, 80–0714

SMOKING, PIPE
(*See also* **SMOKE, TOBACCO; TOBACCO, PIPE**)
adults in the United States, 80–1522
effect of hookah smoking on preleukoplakia incidence in India, 80–0989
effect of inhaling on bladder neoplasm risk, 80–1152
effect of inhaling on carboxyhemoglobin levels, 80–1302
effect of inhaling on mortality rates, 80–0851
effect of inhaling on nicotine content in blood, 80–1302
effect of water pipe smoking on lung function, 80–0956
effect on blood cholesterol and triglyceride levels, 80–1218
effect on heart rate, learning, and psychomotor performance, 80–0412
effect on lung morphology in cotton industry workers, 80–0948
oral epithelial keratinization in leukoplakia patients and, 80–1486

SMOKING SURVEYS
- academic psychologists in the United States, 80–0144
- administrators in schools for health professionals in the United States, 80–1236
- adolescents and adults in New Zealand, 80–0778
- adolescents and adults in the United States, 80–0399
- adolescents in Ethiopia, 80–0995
- adolescents in Finland, 80–0426
- adolescents in France, 80–0405, 80–1412
- adolescents in Switzerland, 80–1244
- adults and adolescents in Canada, 80–0410
- adults in Finland, 80–0416
- adults in Great Britain, 80–0423
- adults in Sweden, 80–0418
- adults in the United States, 80–0420, 80–0996
- attitudes of health department personnel in Hawaii toward control of smoking in public places, 80–0826
- attitudes toward antismoking campaigns in France, 80–1235
- in Austria, 80–0926, 80–1150
- children in France, 80–1000
- college students in the United States, 80–1778
- control programs and restrictions in the workplace, 80–1495
- in Europe, 80–0857
- health professionals in the United States, 80–0813
- high school students in the United States, 80–0414
- in Japan, 80–1766
- medical students in the U.S.S.R., 80–1240
- medical students in the United States, 80–0814
- in Nigeria, 80–1574
- in Norway, 80–1006
- physicians in Australia, 80–1502
- physicians in New Zealand, 80–1509
- physicians in the United States, 80–0828
- psychiatric hospital patients and staff in Finland, 80–0411
- in schools in Ohio, 80–1770
- in Switzerland, 80–1494
- technical university students in Norway, 80–1007
- U.S. National Clearinghouse for Smoking and Health report on adults in the United States, 80–1522
- in the United States, 80–0819
- university students in Australia, 80–0401
- university students in Romania, 80–1508, 80–1753
- in urban and suburban areas in Boston, 80–0158
- white vs. colored adolescents and their parents in South Africa, 80–0141

SNUFF
- determination of nitrosonornicotine content, 80–0024
- fluoride and sugar content, 80–0239, 80–0696
- production and sales in the United States, 80–1829

SNUFF-DIPPING
- effect on nicotine levels in blood, 80–1137
- in leukoplakia etiology, 80–0390
- oral epithelial keratinization in leukoplakia patients and, 80–1486
- in periodontal disease etiology, 80–0390
- role of physicians in opposition to advertising, 80–1045

SOCIOECONOMIC STATUS
- effect of health education on attitudes toward smoking in children, 80–1786
- effect of smoking and employment status on risk for coronary disease in women, 80–0787
- effect of smoking and ethnic group on coronary disease risk, 80–1448
- effect on birth weight, perinatal mortality, and infant morbidity and mortality, 80–1738
- effect on coronary disease risk, 80–0966
- effect on mortality rate in the United States, 80–0075
- effect on smoking characteristics, 80–1520
- effect on smoking habit in the United States, 80–0819, 80–0996
- smoking habit in cervix neoplasm patients and, 80–1407
- tobacco farmers in Kentucky, 80–1038

SPERMATOZOA
- (*See also* **UROGENITAL SYSTEM**)
- density and motility in smoking vs. nonsmoking infertile patients, 80–1480
- effect of smoking and alcohol consumption on count and mobility, 80–1488
- mutagenicity of cigarette smoke constituents, 80–0714

Spirometric measurements *See* **RESPIRATORY FUNCTION TESTS**

SPUTUM ANALYSIS
- in respiratory tract neoplasm diagnosis in smokers vs. nonsmokers, 80–0759
- smokers vs. nonsmokers in rural vs. urban areas in Yugoslavia, 80–0340
- smoking and ferruginous bodies in asbestos workers, 80–0943
- in smoking vs. nonsmoking cosmetologists, 80–0775

SPUTUM PRODUCTION
- effect of smoking habit, 80–1177

STATISTICAL ANALYSIS
- methodology for risk evaluation of less hazardous cigarettes, 80–1283

STEROIDS
- effect of nicotine on steroidogenesis in testes in rats, 80–1336
- effect of smoking and alcohol consumption on 17-ketosteroid content in urine, 80–1488

- effect on aryl hydrocarbon hydroxylase activity in rats, 80–1377

STEROL CONTENT
- in green vs. pale yellow tobacco varieties, 80–0709
- in tobacco, effect of ozonization, 80–1823
- in tobacco leaf, effect of agricultural practices, 80–0242, 80–0243
- in tobacco leaf, effect of ozone on lipid fraction, 80–1819

STEROLS
- distribution of phytosterols in mainstream vs. sidestream smoke, 80–1332

STOMACH
- effect of nicotine on mucosa in rats, 80–1356
- effect of smoking on mucosa in peptic ulcer patients, 80–0806

STOMACH NEOPLASMS
(*See also* **INTESTINAL NEOPLASMS**)
- effect of smoking, alcohol consumption, and diet on incidence, 80–1163

STRESS
- coronary disease and, 80–0117
- coronary disease in smokers vs. nonsmokers and, 80–0121
- coronary disease risk factors in smokers vs. nonsmokers and, 80–1715
- effect of smoking on heart rate and respiration, 80–1784
- effect on smoking characteristics, 80–1755
- effect on smoking habit, 80–0149
- effect on smoking habit in journalists, 80–0403
- as perceived cause of myocardial infarct, 80–0824
- physiological and psychological effects of high- vs. low- nicotine cigarettes, 80–1505
- as risk factor for ischemic heart disease and myocardial infarct, 80–0979
- smoking and psychosocial stress as risk factors in myocardial infarct patients, 80–1435
- smoking habit and, 80–1528
- smoking habit in psychosomatic disorder patients and, 80–0406

STUDENTS, COLLEGE
- effect of hypertension control program on smoking habit, 80–1197
- smoking habit and alcohol consumption and, 80–1008
- smoking habit and alcohol consumption in Australia, 80–0401
- smoking habit and attitudes in the United States, 80–1778
- smoking habit in Norway, 80–1007
- smoking habit in Romania, 80–1753
- smoking habit in the U.S.S.R., 80–1242

STUDENTS, MEDICAL
- smoking habit and attitudes in the United States, 80–0814
- smoking habit in the U.S.S.R., 80–1240, 80–1242

SUGAR CONTENT
(*See also* **CARBOHYDRATES**)
- in cigar, cigarette, pipe, and snuff tobacco, 80–0696
- in cigarettes, effect on mutagenicity of smoke condensate in *Salmonella typhimurium*, 80–0280
- determination in tobacco using high pressure liquid chromatography, 80–1317
- determination of fructose, glucose, and sucrose in tobacco, 80–1604
- effect of fertilization on levels in tobacco leaf, 80–0190
- effect on tobacco quality, 80–0030, 80–0035
- in flue-cured tobacco, effect of fertilization and plant density, 80–0681
- in flue-cured tobacco varieties in Pakistan, 80–0204
- glucose and sucrose in chewing tobacco vs. snuff vs. tobacco leaf, 80–0239
- in Taiwan vs. United States tobacco varieties, 80–0025
- in tobacco, effect of agricultural practices, 80–0866, 80–1586
- of tobacco, effect of alpha-amylase and amyloglucosidase, 80–0558
- in tobacco, effect of climate and soil in Zambia, 80–0683
- in tobacco, effect of climatic conditions, 80–1592
- in tobacco, effect of fertilizer and pH, 80–1587
- in tobacco, effect of growth regulator, 80–1314
- in tobacco, effect of nutrient deficiency, 80–1315
- in tobacco, effect of priming and topping, 80–1590
- in tobacco, effect of sucker control agents, 80–1070
- in tobacco leaf, effect of agricultural practices, 80–1591
- of tobacco leaf, effect of calcium fertilizer, 80–0008
- in tobacco leaf, effect of curing, 80–1816
- in tobacco leaf, effect of fertilization, 80–0202
- in tobacco leaf, effect of flue-curing in Indian varieties, 80–1263
- in tobacco leaf, effect of homogenized leaf curing, 80–1261
- in tobacco leaf, effect of irrigation, 80–0684
- in tobacco leaf, effect of maturation stage, 80–0697

SULFUR COMPOUNDS
- determination of sulfate levels in tobacco, 80–0704
- determination of thiol reactivity with cigarette smoke, 80–1602
- effect of air pollution on mortality rates in the United States, 80–0291

effect of iron sulfate in filters on hydrogen sulfide removal from smoke, 80–0527
effect of sulfides on tobacco aroma and flavor, 80–0615

SULFUR DIOXIDE
effect of air pollution on mortality rates in the United States, 80–0291
effect of smoking on bronchitis incidence in copper smelter workers, 80–1686
effect of smoking on lung function in copper smelter workers, 80–1686
effect on lipid content in lungs and trachea in squirrels, 80–0062
effect on lung function and respiratory symptoms in smokers vs. nonsmokers, 80–0098
effect on lung function in smokers vs. nonsmokers and, 80–0092

TAR CONTENT
in cigar smoke, effect of polymers, 80–0554
in cigar smoke, effect of reconstituted tobacco foam wrapper, 80–0497
in cigarette smoke, effect of agricultural practices, 80–0207
in cigarette smoke, effect of calcium-alginate tobacco substitute, 80–0645
in cigarette smoke, effect of carboxymethyl cellulose filters, 80–0511
in cigarette smoke, effect of cellulose acetate filters, 80–0538
in cigarette smoke, effect of cigarette characteristics and tobacco stalk position, 80–1608
in cigarette smoke, effect of cigarette paper, 80–0033
in cigarette smoke, effect of Cytrel substitute, 80–1544
in cigarette smoke, effect of filters, 80–0029
in cigarette smoke, effect of laminated tobacco foil, 80–0484
in cigarette smoke, effect of organic oils and powders in filters, 80–0510
in cigarette smoke, effect of paper perforation, 80–1264
in cigarette smoke, effect of permeable polymer membrane filter, 80–0483
in cigarette smoke, effect of protein granule filters, 80–0523
in cigarette smoke, effect of resins and organic and metal compound filters, 80–0526
in cigarette smoke, effect of thermoplastic polymeric filters, 80–0493
in cigarette smoke, effect of tobacco shreds, 80–1262
in cigarette smoke, effect of ventilated filters, 80–1016
in cigarette smoke, effect of wool filters, 80–0496
in cigarette smoke, effect on smoking characteristics, 80–0400
in cigarette smoke, effect on smoking habit, 80–0400
in cigarette smoke of Canadian brands, 80–0869, 80–1603
in cigarette smoke of green vs. pale yellow tobacco varieties, 80–0709
in cigarettes, effect of expansion and fiberization, 80–0499
in cigarettes, effect on pulmonary alveolar macrophages in rats, 80–1638
in cigarettes, effect on respiratory symptom incidence and sputum production, 80–1177
in cigarettes, effect on respiratory system, 80–1687
in cigarettes, effect on smoking characteristics, 80–1750, 80–1757, 80–1785
in cigarettes, effect on smoking habit, 80–0150, 80–1294, 80–1750
determination in cigarette brands in Great Britain, 80–0700
determination in cigarette brands in the United States, 80–0220
determination in less hazardous cigarettes, 80–1606
in herbal cigarettes in Australia, 80–1090
prediction in tobacco, 80–1081
in smoke of cigarettes containing puffed milo and tobacco, 80–0649
in smoke of cigarettes containing puffed rice and tobacco, 80–0650
in smoking material from entangled tobacco fibers, 80–1259
in smoking products containing popped corn, 80–0651
in tobacco, effect of ash alkalinity and total nitrogen content, 80–1812
in tobacco, effect of curing and leaf level, 80–1812
in tobacco smoke, effect of agricultural practices, 80–0687
in tobacco smoke, effect of expansion vs. reconstitution processes, 80–0174
in tobacco smoke, effect of tobacco varieties, 80–1086
in tobacco smoke, effect of transition metals, 80–0566

TAR REDUCTION
in cigarettes in Canada, 80–1036
effect of boiling, drying, and rehydration of tobacco, 80–0547, 80–0548
effect of microporous styrene polymer cigarette filters, 80–0529
effect of tobacco processing with potassium, 80–1814

TARS, CIGARETTE
analysis of ether-soluble neutral fraction, 80–1330
analysis of water-soluble fraction, 80–1331
antigens in extracts, 80–0262
carcinogenicity in bacterial vs. mouse skin response tests, 80–0251
chromosome aberrations in lung neoplasm etiology, 80–0723

- determination of acetic and formic acid content, 80–1087
- determination of aliphatic and aromatic ketones, 80–1319
- determination of alpha radioactivity, 80–1597
- determination of catechols, resorcinols, and hydroquinones, 80–0037
- determination of components by gas chromatography, 80–0026
- determination of components from lamina vs. midrib cigarettes, 80–0027
- determination of indoles and carbazoles, 80–0710
- determination of multi-alkylated polynuclear aromatic hydrocarbons, 80–0038
- determination of polyaromatic hydrocarbons, 80–0228
- determination of trace metals, 80–0034
- effect of cigarette paper on delivery, 80–0512
- effect of cigarette smoke on levels in indoor environments, 80–1378
- effect of particle size on chemical characteristics of mainstream smoke, 80–1323
- effect of passive smoking on eyes, nose, and throat, 80–0809, 80–1748
- effect of smoking characteristics on delivery, 80–1501
- effect of sugars in cigarettes on mutagenicity in *Salmonella typhimurium*, 80–0280
- effect of transplacental administration on neoplasm induction in mother and offspring in rats, 80–1644
- effect on enzyme activity in liver in rats, 80–1107
- effect on mitochondria in brown fat cells in hamsters, 80–0735
- effect on morphogenesis in lungs in chick embryo, 80–0889
- identification of components in lamina and midrib cigarettes by trimethylsilylation, 80–1322
- interaction with acetone during chemical analysis, 80–0020
- isolation of phenols by gel chromatography, 80–1088
- in lung neoplasm induction in hamsters, 80–1360
- mutagenicity in *Salmonella typhimurium*, 80–1122
- in skin neoplasm induction in mice, 80–0252, 80–0740

TARS, TOBACCO
- constituent levels in tobacco varieties, 80–0876
- definition of "condensate" vs. "tar", 80–0465
- detection of particulates in animal smoke inhalation exposure systems, 80–0023
- determination of norsequiterpenes, 80–0215
- effect of agricultural practices on levels in smoke, 80–0198
- effect of cigarette periphery and rod on smokestream formation, 80–0028
- effect of colloidal alumina and silica on retention in filters, 80–0503
- effect of curing on levels in smoke, 80–0198
- effect on DNA repair in yeast, 80–1109
- metal binding, 80–0877
- musk compounds in Oriental variety, 80–0216
- nonvolatile and semivolatile components, 80–0232

TASTE
- acuity in smokers vs. nonsmokers, 80–1484
- evaluation of smoking taste test using arousal manipulation, 80–1755

TAXATION
- cigarette sale revenues in West Germany, 80–0446
- effect on cigarette consumption and revenues in Kentucky, 80–1032
- effect on tobacco substitute sales in Great Britain, 80–0177
- history of moisture and inorganic content in tobacco assessment in Great Britain, 80–0855
- proposals for "health tax" on cigarette packages, 80–0452
- tobacco revenues in the United States, 1863-1979, 80–1275

TEETH
- bacterial flora in plaque in smokers vs. nonsmokers, 80–1747
- effect of cessation of smoking on bruxism, 80–0821
- effect of smoking, 80–0396
- effect of smoking and mouthwash on staining, 80–0393
- plaque formation in smokers vs. nonsmokers, 80–0988
- smoking in discoloration etiology, 80–0392

TERATOGENESIS
- maternal smoking and, 80–1735

TERPENE CONTENT
- determination in Bulgarian oriental tobacco, 80–0225
- determination of norsequiterpenes in tobacco condensate, 80–0215
- in green vs. pale yellow tobacco varieties, 80–0709

TERPENES
- identification of solanascone in Burley variety, 80–0695

TERPENOIDS
- determination in Burley tobacco, 80–0694
- distribution of neophytadiene in mainstream vs. sidestream smoke, 80–1332
- diterpenoids in Greek tobacco, 80–0211
- in Greek tobacco, 80–0213
- identification of monoterpene diol in tobacco, 80–0689
- isolation and identification of labdanic diterpenoids in tobacco, 80–1333
- synthesis from (12*E*)-abienol, 80–0248
- synthesis from (12*Z*)-abienol, 80–0249

in tobacco, 80–0871
in tobacco leaf and smoke, 80–0867

Testes *See* **UROGENITAL SYSTEM**

THIOCYANATE LEVELS
(*See also* **CYANIDE CONTENT**)
in blood, and self-reported reduction of smoking, 80–0164
in blood, as outcome predictor in cessation of smoking, 80–0172
in blood in smokers vs. nonsmokers, 80–1098
determination in blood in smokers vs. nonsmokers, 80–0870
effect of maternal smoking, 80–0385
effect of smoke inhalation in baboons, 80–1649
smokers vs. nonsmokers, 80–0885, 80–1622
in smokers vs. nonsmokers in rural vs. urban areas, 80–1623

THORIUM-228 CONTENT
determination in cigarette smoke condensate, 80–1597

THROMBOANGIITIS OBLITERANS
(*See also* **ARTERIAL DISEASES; THROMBOSIS**)
bidi smoking and, 80–0124
smoking habit in patients, 80–0960, 80–1467
surgical treatment with adrenalectomy and sympathectomy, 80–0124
tobacco allergy in etiology of, 80–0790

THROMBOEMBOLISM
(*See also* **CEREBROVASCULAR DISORDERS; VASCULAR DISEASES**)
effect of smoking and oral contraceptives on incidence, 80–0372
effect of smoking on incidence in myocardial infarct patients, 80–0351

THROMBOSIS
(*See also* **CARDIOVASCULAR DISEASES; CEREBROVASCULAR DISORDERS; CORONARY DISEASE; THROMBOANGIITIS OBLITERANS; VASCULAR DISEASES**)
physical activity in smoking vs. nonsmoking postoperative patients and, 80–1707
smoking habit in myocardial infarct patients and, 80–0794

TISSUES
cadmium levels in smokers vs. nonsmokers, 80–1634

TOBACCO
(*See also* **TOBACCO, CIGAR; TOBACCO, PIPE**)
alkaloids and isoprenoids, 80–0871
allergy in thromboangiitis obliterans etiology, 80–0790
ash content in flue-cured varieties in Pakistan, 80–0204
bidi, effect of fertilization and fumigation on yield and quality, 80–0006
carotenoid-related compound in Japanese variety, 80–0240
chewing tobacco production and sales in the United States, 80–1829
consumption in Egypt, 80–1553
determination of alkaloid content by nonaqueous conductimetric titration, 80–0705
determination of alkaloid content by spectrophotometry, 80–0701
determination of aroma constituents, 80–0872
determination of aromatic constituents in acid fraction, 80–1080
determination of carbamate residues, 80–0214
determination of carbohydrates, 80–0218
determination of condensate potential by macropyrolysis, 80–1593
determination of fatty acids and nonvolatile organic acids, 80–0016
determination of hydrocarbons by partition column chromatography, 80–0217
determination of nicotine and nornicotine by pyrolysis-gas chromatography, 80–1329
determination of nitrate and sulfate levels, 80–0704
determination of nitrogen content, 80–0021
determination of nitrosonornicotine content, 80–0024
determination of organochlorine insecticide residues, 80–0235, 80–0236
determination of paraffinic hydrocarbons, 80–0031
determination of pesticide residues, 80–1082, 80–1089, 80–1327, 80–1328
determination of phenol content by Folin-Ciocaltau reagent, 80–0247
determination of polypeptide composition of Fraction 1 proteins by isoelectric focusing, 80–1607
determination of sugar content, 80–1604
determination of sugar content using high pressure liquid chromatography, 80–1317
determination of total volatile bases from nitrogen and nicotine content of flue-cured variety, 80–1085
effect of acids on lead-210 and polonium-210 content, 80–1541
effect of agricultural practices on alkaloid and sugar content, 80–1586
effect of agricultural practices on constituents and yield, 80–0864, 80–0866
effect of agricultural practices on flecking, growth, and chemical composition of flue-cured tobacco, 80–0007
effect of agricultural practices on growth and disease resistance, 80–0208
effect of agricultural practices on growth and yield, 80–1589

effect of agricultural practices on nicotine content, 80–0192

effect of agricultural practices on physical and chemical characteristics, 80–0195

effect of agricultural practices on quality and ripening, 80–1585

effect of agricultural practices on quality and yield, 80–0201, 80–1586

effect of alkaloid content on ash formation, 80–0237

effect of alpha-amylase and amyloglucosidase on sugar content, 80–0558

effect of bacterial treatment on nicotine levels, 80–0550, 80–0551, 30–0553, 80–0559, 80–0560, 80–0561

effect of benzylthiolcarbamate derivatives on sucker growth, 80–0476

effect of boiling on carbon monoxide, nicotine, nitrogen dioxide, and tar levels, 80–0547

effect of boiling on constituent levels, 80–0548

effect of breeding on nicotine content, 80–0194

effect of calcium fertilizer on leaf components, 80–0008

effect of calcium fertilizer on yield, 80–0008

effect of carbetamide on sucker growth, 80–0473

effect of catalase—hydrogen peroxide reaction on expansion and quality, 80–0531

effect of climate and soil on chemical composition, quality, and yield in Zambia, 80–0683

effect of climatic conditions on alkaloid and sugar content, 80–1592

effect of crop rotation on cigar wrapper quality and yield, 80–0685

effect of curing and leaf level on tar content, 80–1812

effect of dialdehydes on stiffness and filling capacity, 80–0519

effect of dicarboxylic acids and derivatives on expansion, 80–0500

effect of 2, 6-dinitroaniline derivatives on sucker growth, 80–0475

effect of diphenyl sulfides and diphenyl ethers on sucker growth, 80–0481

effect of environmental and genetic factors on alkaloid content, 80–0238

effect of fatty alcohols and 1, 2-dihydro-pyridazine-3, 6-dione salts on sucker growth, 80–0477

effect of fertilization and seasonal variation on leaf and paddy yield, 80–0003

effect of fertilization and seasonal variation on leaf components, 30–0003

effect of fertilization on agronomic and chemical characteristics, 80–1313

effect of fertilization on boron content and yield in *N. tabacum* L., 80–0199

effect of fertilization on chemical composition and quality, 80–0205

effect of fertilization on quality and yield, 80–1073

effect of fertilization with chlorine on growth and quality, 80–0004

effect of fertilization with magnesium on quality and yield, 80–1072

effect of fertilization with nitrogen on quality and yield of chewing tobacco, 80–0686

effect of fertilizer and pH on chemical composition, 80–1587

effect of foliar nutrition on quality and yield, 80–0200

effect of genetic manipulation on constituents, 80–0196

effect of glycols on moisture content, 80–1811

effect of growth regulator on agronomic and physicochemical characteristics, 80–1314

effect of herbicide on yield and quality, 80–0010

effect of Indian variety ingestion in herbal tea, 80–0395

effect of insecticides on enzyme activity and pigment levels, 80–1068

effect of irrigation on growth and weight, 80–0684

effect of leaf extracts on IgE production in smokers vs. nonsmokers, 80–0899

effect of maleic hydrazide residues on sales in West Germany, 80–1827

effect of *N*-alkyl-*N*-phenylsulfenyl-*N'*-phenyl-formamidine derivatives on sucker growth, 80–0472

effect of *N*-benzyl-2, 6-dinitro-3-amino-4-trifluoromethylanilines on sucker growth, 80–0480

effect of nematicide application on seed germination, 80–1316

effect of ozonization on constituent levels, 80–1823

effect of pesticide dosage on residue levels in leaves, 80–0191

effect of phosphorothionoamidate derivatives on sucker growth, 80–0479

effect of physical and chemical characteristics on quality, 80–0030, 80–0035

effect of planting time on quality and yield, 80–1067

effect of potassium content on quality, 80–0226

effect of priming and topping on quality and yield, 80–1590

effect of ripeness on quality, 80–0009

effect of shreds on cigarette smoke condensate yields, 80–1262

effect of sucker control agent on maleic hydrazide residues, 80–0865

effect of wetting and drying on nicotine levels, 80–0546

effect of zineb on zinc content and distribution, 80–0682

ethephon metabolism, 80–0260

exports, imports, and production in the Dominican Republic, 80–1037

exports in the United States, 80–1276

fluoride and sugar content, 80–0696

- fluoride and sugar content in chewing tobacco vs. leaf vs. snuff, 80–0239
- halogenated hydrocarbons as nicotine extraction solvents, 80–0565
- history of origin and usage, 80–1835
- identification of Fraction 1 protein polypeptides by isoelectric focusing, 80–0875
- identification of monoterpene diol, 80–0689
- identification of nor-carotenoids, 80–0212, 80–0688, 80–1318
- identification of phytuberol, 80–1605
- identification of quinone, 80–0693
- industry's viewpoint of research needs, 80–1289
- isolation and identification of labdanic diterpenoids, 80–1333
- lipid extraction, 80–1545
- metabolism of maleic hydrazide residues, 80–0045
- methomyl uptake in plants, 80–0046
- moisture and inorganic content in revenue assessment in Great Britain, 80–0855
- nicotine, organic acid, and solanesol content in waste material, 80–1326
- pesticide residue levels in soil in Ontario, 80–0703
- pesticide residues in imports in Europe, 80–0001
- polyphenol content in seedlings, 80–0690
- potassium nitrate removal, 80–0552
- prediction of tar content, 80–1081
- production and exports in Pakistan, 80–1824
- production in Spain, 80–1274
- production of blond vs. dark varieties in France, 80–1272
- production quotas in the United States, 80–1035
- sales of chewing tobacco in the United States, 80–1273
- smoking material from entangled fibers, 80–1259
- taxation in the United States, 1863-1979, 80–1275
- terpenoids in Greek variety, 80–0211, 80–0213
- worldwide expenditures and taxation, 80–1026
- worldwide production figures for 1978, 80–0179

TOBACCO ADDITIVES

(*See also* **TOBACCO AROMA; TOBACCO FLAVOR**)

- 1-acetyl-3, 3-dimethyl cyclohexane derivatives for aroma and flavor enhancement, 80–0590
- alkyl-2, 3-dihydro-3(1′-hydroxyalkylidene)-2-oxo-5-alkyl-furan-4-carboxylates for flavor enhancement, 80–0625
- alkyl-2-methyl-3, 4-pentadienoates for aroma and flavor enhancement, 80–0588
- 2-alkyl-4-phenyl-dihydropyran derivatives for aroma and flavor enhancement, 80–0619
- 2-alkyl-4-phenyl-dihydropyrans for aroma and flavor enhancement, 80–0616, 80–0618
- alpha-substituted acetaldehyde for aroma and flavor enhancement, 80–0607
- alpha-substituted vinyl menthyl carbonate ester polymers for aroma and flavor enhancement, 80–0586
- alumina or zirconia fibers as filtering agents, 80–0507
- amides for aroma and flavor enhancement, 80–0577
- benzo-1, 2-pyrone derivatives for aroma and flavor enhancement, 80–0593, 80–0613
- beta-hydroxy esters and derivatives for aroma and flavor enhancement, 80–0613
- butanone derivatives for aroma and flavor enhancement, 80–0603
- 2-butyl-3, 5, 5-trimethyl-2-cyclohexen-1, 4-dione for aroma and flavor enhancement, 80–0601
- butyrolactone derivatives for flavor enhancement, 80–0581
- carbonate ester resins for aroma and flavor enhancement, 80–0585
- carboxaldehydes for aroma and flavor enhancement, 80–0591
- caryophyllenepoxides and caryophyllene alcohols for aroma and flavor enhancement, 80–0570
- catalase and hydrogen peroxide for expansion, 80–0530
- cinnamic derivatives for aroma and flavor enhancement, 80–0568, 80–0569
- cycloaliphatic ketones, 80–1256
- cyclogeraniol and derivatives for aroma and flavor enhancement, 80–0594
- cyclohexadiene derivatives for aroma and flavor enhancement, 80–0599
- 1, 2-cyclohexanedione for aroma and flavor enhancement, 80–0627
- dialdehydes for increasing stiffness and filling capacity, 80–0519
- effect of ascorbic acid on nitrogen dioxide and nitrosamine levels in smoke, 80–0557
- effect of erythorbic acid on nitrogen dioxide and nitrosamine levels in smoke, 80–0557
- effect of zinc oxide and nitrates on polycyclic aromatic hydrocarbon levels in tobacco smoke, 80–0562
- effect of metal salt—amine complexes on cyanide content in smoke, 80–0564
- effect of palladium and ammonium salts on aromatic hydrocarbon content in smoke, 80–0563
- effect of palladium on aromatic hydrocarbon content in smoke, 80–0545
- effect of urea on smoke quality, 80–0605
- effect on flavor, 80–1257
- enol esters for aroma and flavor enhancement, 80–0606
- epoxy-β-ionone for aroma and flavor enhancement, 80–0575
- 3-ethoxy-4-hydroxybenzaldehyde 2, 2-dimethyl-propanediol acetate for aroma and flavor enhancement, 80–0595
- glycols as humectants, 80–1811

hydroxy cyclohexenone derivatives for aroma and flavor enhancement, 80–0600
2-hydroxymethylenecyclohexanones for aroma and flavor enhancement, 80–0583
imidazole and benzimidazole derivatives for aroma and flavor enhancement, 80–0611
inorganic salts for burn rate modification, 80–0634
isobutyl-substituted heterocyclic compounds for aroma and flavor enhancement, 80–1810
isopropenyl ester polymers for aroma and flavor enhancement, 80–0584
3-isopropyl-cyclopent-2-en-1-one for aroma and flavor enhancement, 80–0576
2-isopropyl-4-phenyl-dihydropyran for aroma and flavor enhancement, 80–0617
isovaleric and methyl valeric acids for flavor enhancement, 80–0628
lactone derivatives for aroma and flavor enhancement, 80–0602
meerschaum as adsorbent for cigarette smoke constituents, 80–0544
menthol esters for aroma and flavor enhancement, 80–0610
menthol fixation methods for flavor enhancement, 80–0441
menthol for flavor enhancement, 80–0567
metal oxides and oxyhydrates as filtering agents, 80–1807
2-methyl-5(α-methyl-α-hydroxyethyl)-cyclohex-2-en-1-one for aroma and flavor enhancement, 80–0571
6-methyl-hepta-3, 5-dien-2-one for aroma and flavor enhancement, 80–0573
2-methyl-4-pentenoic acid for aroma and flavor enhancement, 80–0592
methylated silica for filter efficiency, 80–0505
4-(methylthio)butane derivatives for aroma and flavor enhancement, 80–0608
nonane-2, 5, 8-trione for aroma and flavor enhancement, 80–0574
norbornane derivatives for aroma and flavor enhancement, 80–0596
octa-3, 5-dien-2-one for aroma and flavor enhancement, 80–0572
octahydrotetramethyl acetonaphthone isomers for aroma and flavor enhancement, 80–0589
oxocylic pyrimidines for flavor enhancement, 80–0582
p-menthane-3-carboxamide derivatives as cooling agents in nasal and oral mucosa, 80–0624
pectin as filling agent, 80–0528
pentadienoates for aroma and flavor enhancement, 80–0587
2-phenyl-3-(fur-2-yl)-prop-2-en-1-al for flavor enhancement, 80–0578
pipe for use with fruit for aroma and flavor enhancement, 80–1806
polymeric carbonate ester derivatives for aroma and flavor enhancement, 80–0614
polymers for cigar smoke constituent alteration, 80–0554
pullulan and derivatives for aroma and flavor enhancement, 80–0520
pyrrole derivatives for aroma and flavor enhancement, 80–0579
reaction product of carbonyl compounds, sugar, and valine for flavor enhancement, 80–0622
recommendations of the Hunter Committee for use of flavorings, humectants, and sugars, 80–0856
resin as a strengthening agent in expansion, 80–0532
ruthenium catalyst for carbon monoxide and nitric oxide removal from smoke, 80–1820
silica for filter efficiency, 80–0506
sodium carboxymethyl cellulose as binder, 80–1265
spirane derivatives for flavor enhancement, 80–0609
3-substituted-*p*-menthane derivatives as cooling agents in nasal and oral mucosa, 80–0623
sulfides for aroma and flavor enhancement, 80–0615
tamarind gum adhesive as binding agent for reconstituted tobacco, 80–0534
3, 7, 11, 11-tetramethyl-spiro(5, 5) undeca-8-ene-1-one for aroma and flavor enhancement, 80–0604
thiazoline derivatives for aroma and flavor enhancement, 80–0620
transition metals for alteration of smoke constituent levels, 80–0566
tricyclene-9-butenone for aroma and flavor enhancement, 80–0621
tricyclic alcohols for aroma and flavor enhancement, 80–0597, 80–0598, 80–1808
tricyclic ketone derivatives for aroma and flavor enhancement, 80–0612
2, 4, 6-triisobutyl-1, 3, 5-trioxane for aroma and flavor enhancement, 80–0626
volatile compounds for flavor enhancement, 80–0525
zinc as tracer for evaluation of hydrophobic coatings, 80–0498

TOBACCO AGING
effect on constituent levels, 80–1258
effect on polyphenol content in flue-cured tobacco, 80–0840

TOBACCO AROMA
(*See also* **TOBACCO ADDITIVES; TOBACCO FLAVOR)**
determination of carotenoid- and thunberganoid-related compounds in Burley tobacco, 80–0694
determination of constituents in acid fraction of tobacco, 80–1080
determination of constituents in tobacco, 80–0872
determination of dihydro-γ-pyrone in Burley vs. Oriental vs. Virginia varieties, 80–1076

effect of α-substituted vinyl menthyl carbonate ester polymers, 80–0586
effect of 1-acetyl-3, 3-dimethylcyclohexane derivatives, 80–0590
effect of alkyl-2-methyl-3, 4-pentadienoates, 80–0588
effect of 2-alkyl-4-phenyl-dihydropyran derivatives, 80–0619
effect of 2-alkyl-4-phenyl-dihydropyrans, 80–0616, 80–0618
effect of alpha-substituted acetaldehyde, 80–0607
effect of amides, 80–0577
effect of benzo-1, 2-pyrone derivatives, 80–0593
effect of beta-hydroxy esters and derivatives, 80–0613
effect of butanone derivatives, 80–0603
effect of 2-butyl-3, 5, 5-trimethyl-2-cyclohexen-1, 4-dione, 80–0601
effect of carbon-filled paper substitute, 80–0642
effect of carbonate ester resins, 80–0585
effect of carboxaldehydes, 80–0591
effect of caryophyllenepoxides and caryophyllene alcohols, 80–0570
effect of C_1-C_6 alkyl-2-methyl-3, 4-pentadienoates, 80–0587
effect of cinnamic derivatives, 80–0568, 80–0569
effect of cycloaliphatic ketones, 80–1256
effect of cyclogeraniol and derivatives, 80–0594
effect of cyclohexadiene derivatives, 80–0599
effect of 1, 2-cyclohexanedione, 80–0627
effect of Cytrel substitute, 80–1544
effect of enol esters, 80–0606
effect of epoxy-β-ionone, 80–0575
effect of 3-ethoxy-4-hydroxybenzaldehyde 2, 2-dimethyl propanediol acetal, 80–0595
effect of hydroxy cyclohexenone derivatives, 80–0600
effect of 2-hydroxymethylenecyclohexanones, 80–0583
effect of imidazole and benzimidazole derivatives, 80–0611
effect of isobutyl-substituted heterocyclic compounds, 80–1810
effect of isopropenyl ester polymers, 80–0584
effect of 3-isopropyl-cyclopent-2-en-1-one, 80–0576
effect of 2-isopropyl-4-phenyl-dihydropyran, 80–0617
effect of lactone derivatives, 80–0602
effect of menthol esters, 80–0610
effect of 2-methyl-5-(α-methyl-α-hydroxyethyl)-cyclohex-2-en-1-one, 80–0571
effect of 6-methyl-hepta-3, 5-dien-2-one, 80–0573
effect of 2-methyl-4-pentenoic acid and ethyl ester derivatives, 80–0592
effect of 4-(methylthio)butane derivatives, 80–0608
effect of 1-(3-(methylthio)butyryl)-2, 6, 6-trimethylcyclohexene and 1-3-cyclohexadiene analog, 80–1809
effect of nonane-2, 5, 8-trione, 80–0574
effect of octa-3, 5-dien-2-one, 80–0572
effect of octahydrotetramethyl acetonaphthone isomers, 80–0589
effect of polymeric carbonate ester derivatives, 80–0614
effect of protein in tobacco substitutes, 80–0629
effect of pullulan and derivatives in shaped tobacco, 80–0520
effect of pyrrole derivatives, 80–0579
effect of reconstituted tobacco foam in cigars, 80–0497
effect of substituted norbornane derivatives, 80–0596
effect of sulfides, 80–0615
effect of 3, 7, 11, 11-tetramethyl-spiro(5, 5)undeca-8-ene-1-one, 80–0604
effect of thiazoline derivatives, 80–0620
effect of tobacco processing, 80–1546
effect of tricyclene-9-butenone, 80–0621
effect of tricyclic alcohols, 80–0597, 80–0598, 80–1808
effect of tricyclic ketone derivatives, 80–0612
effect of 2, 4, 6-triisobutyl-1, 3, 5-trioxane, 80–0626
effect of urea, 80–0605
pipe for use with fruit, 80–1806

TOBACCO CHEWING
(*See also* **ARECA CHEWING**)
and areca chewing, in leukoplakia etiology, 80–0394
and areca chewing, in mouth neoplasm etiology in Pakistan, 80–0394
effect on preleukoplakia incidence in India, 80–0989
effect on relative risk for bladder neoplasms, 80–1152
effect on risk for esophageal neoplasms, 80–0024
effect on saliva constituents and flow rate, 80–1133
in oral neoplasm etiology, 80–0317
role of physicians in opposition to advertising, 80–1045

TOBACCO, CIGAR
fluoride and sugar content, 80–0696

TOBACCO CONTAMINANTS
(*See also* **PESTICIDE RESIDUES**)
DDT levels in flue-cured tobacco, 80–0186
determination of carbamate residues, 80–0214, 80–0241
determination of organochlorine insecticide residues, 80–0235, 80–0236
determination of pesticide residues, 80–1327
effect of acephate powder vs. emulsion on levels, 80–0203
effect of agricultural practices on butralin residue levels, 80–0244
effect of ethephon on alkaloid and sugar content, 80–1586
effect of ethephon on quality and yield, 80–1586
pesticide residues in imports in Europe, 80–0001

pesticide residues in soils and crops, 1971 National Soils Monitoring Program, 80–0193

TOBACCO CURING
- effect of flue-curing on leaf components in Indian varieties, 80–1263
- effect of flue curing on lipid levels in leaves, 80–1543
- effect of growth regulator, 80–1314
- effect of homogenized leaf curing on tobacco leaf components, 80–1261
- effect of wilt-accelerating agents, 80–0474
- effect on alkaloid, amino acid, carbonyl, and protein content, 80–0012
- effect on constituent levels, 80–1258, 80–1816
- effect on maleic hydrazide distribution, 80–0045
- effect on nicotine and polyphenol distribution in tobacco leaf, 80–1542
- effect on nitrosonornicotine content, 80–0024
- effect on polyphenol content, 80–0840
- effect on tar content in tobacco smoke, 80–0198, 80–0687, 80–1812

TOBACCO EXTRACTS
- allergic response in smoking vs. nonsmoking coronary disease patients, 80–0976
- antigen—antibody reactions in rodents, 80–1636
- antigens in green leaf, cured leaf, and cigarette smoke condensate, 80–0262
- determination of alkylating activity, 80–1320
- effect on DNA synthesis in lymphocytes and tumor cells, 80–0289
- effect on enzyme activity in rats, 80–1127
- mutagenicity in *Salmonella typhimurium*, 80–0746
- potassium nitrate removal, 80–0555

TOBACCO FLAVOR
(*See also* **TOBACCO ADDITIVES; TOBACCO AROMA**)
- determination of dihydro-γ-pyrones in Burley vs. Oriental vs. Virginia varieties, 80–1076
- effect of α-substituted vinyl menthyl carbonate ester polymers, 80–0586
- effect of 1-acetyl-3, 3-dimethylcyclohexane derivatives, 80–0590
- effect of additives, 80–1257
- effect of alkyl-2, 3-dihydro-3(1′-hydroxyalkylidene)-2-oxo-5-alkyl-furan-4- carboxylates, 80–0625
- effect of alkyl-2-methyl-3, 4-pentadienoates, 80–0588
- effect of 2-alkyl-4-phenyl-dihydropyran derivatives, 80–0619
- effect of 2-alkyl-4-phenyl-dihydropyrans, 80–0616, 80–0618
- effect of alpha-substituted acetaldehyde, 80–0607
- effect of amides, 80–0577
- effect of benzo-1, 2-pyrone derivatives, 80–0593
- effect of beta-hydroxy esters and derivatives, 80–0613
- effect of butanone derivatives, 80–0603
- effect of 2-butyl-3, 5, 5-trimethyl-2-cyclohexen-1, 4-dione, 80–0601
- effect of butyrolactone derivatives, 80–0581
- effect of carbon-filled paper substitute, 80–0642
- effect of carbonate ester resins, 80–0585
- effect of carboxaldehydes, 80–0591
- effect of caryophylleneepoxides and caryophyllene alcohols, 80–0570
- effect of C_1-C_6 alkyl-2-methyl-3, 4-pentadienoates, 80–0587
- effect of cinnamic derivatives, 80–0568, 80–0569
- effect of cycloaliphatic ketones, 80–1256
- effect of cyclogeraniol and derivatives, 80–0594
- effect of cyclohexadiene derivatives, 80–0599
- effect of 1, 2-cyclohexanedione, 80–0627
- effect of Cytrel substitute, 80–1544
- effect of 8, 13:13, 20-diepoxy-15, 16-dinorlabdane, 80–0580
- effect of enol esters, 80–0606
- effect of epoxy-β-ionone, 80–0575
- effect of 3-ethoxy-4-hydroxybenzaldehyde 2, 2-dimethyl propanediol acetate, 80–0595
- effect of hydroxy cyclohexenone derivatives, 80–0600
- effect of 2-hydroxymethylenecyclohexanones, 80–0583
- effect of imidazole and benzimidazole derivatives, 80–0611
- effect of isobutyl-substituted heterocyclic compounds, 80–1810
- effect of isopropenyl ester polymers, 80–0584
- effect of 3-isopropyl-cyclopent-2-en-1-one, 80–0576
- effect of 2-isopropyl-4-phenyl-dihydropyran, 80–0617
- effect of isovaleric and methyl valeric acids in tobacco substitutes, 80–0628
- effect of lactone derivatives, 80–0602
- effect of menthol, 80–0441
- effect of menthol applied by vaporization, 80–0567
- effect of menthol esters, 80–0610
- effect of 2-methyl-5-(α-methyl-α-hydroxyethyl)-cyclohex-2-en-1-one, 80–0571
- effect of 6-methyl-hepta-3, 5-dien-2-one, 80–0573
- effect of 2-methyl-4-pentenoic acid and ethyl ester derivatives, 80–0592
- effect of 4-(methylthio)butane derivatives, 80–0608
- effect of 1-(3-(methylthio)butyryl)-2, 6, 6-trimethylcyclohexene and 1, 3-cyclohexadiene analog, 80–1809
- effect of nonane-2, 5, 8-trione, 80–0574
- effect of octa-3, 5-dien-2-one, 80–0572
- effect of octahydrotetramethyl acetonaphthone isomers, 80–0589
- effect of oxocyclic pyrimidines, 80–0582
- effect of 2-phenyl-3-(fur-2-yl)-prop-2-en-1-al, 80–0578
- effect of polymeric carbonate ester derivatives, 80–0614

effect of protein in tobacco substitutes, 80–0629
effect of pullulan and derivatives in shaped tobacco, 80–0520
effect of pyrrole derivatives, 80–0579
effect of reaction product of sugar, valine, and carbonyl compounds, 80–0622
effect of reconstituted tobacco foam in cigars, 80–0497
effect of spirane derivatives, 80–0609
effect of substituted norbornane derivatives, 80–0596
effect of sulfides, 80–0615
effect of 3, 7, 11, 11-tetramethyl-spiro(5, 5)undeca-8-ene-1-one, 80–0604
effect of thiazoline derivatives, 80–0620
effect of tobacco processing, 80–1546
effect of tricyclene-9-butenone, 80–0621
effect of tricyclic alcohols, 80–0597, 80–0598, 80–1808
effect of tricyclic ketone derivatives, 80–0612
effect of 2, 4, 6-triisobutyl-1, 3, 5-trioxane, 80–0626
effect of urea, 80–0605
effect of ventilated cigarettes, 80–0525
identification of musk compounds in Oriental variety, 80–0216
pipe for use with fruit, 80–1806

TOBACCO INDUSTRY
advertising and antismoking campaigns in Great Britain and, 80–1572
advertising in the United States, 80–0448
antismoking campaign opposition by the Tobacco Action Network, 80–1042
attitudes toward antismoking campaigns, 80–1286
attitudes toward smoking and health, 80–1053
cigar, cigarette, and tobacco exports in the United States, 80–1276
cigar production and sales in Holland, 80–1268
cigarette advertising in Great Britain, 80–1300
cigarette sales in the United States, 80–1028
cigarette sales in West Germany, 80–0446
counterorganization to prevent restriction of smoking legislation, 80–1557
in the Dominican Republic, 80–1037
economics in Thailand, 80–0444
effect of advertising restrictions on marketing techniques, 80–1066
effect of antismoking campaigns in Finland and Sweden, 80–1062
effect of diversification on nontobacco income, 80–0447
effect of filter additives on manufacturing and production practices, 80–1017
effect of government programs on benefit distribution, price stability, and production, 80–1021
effect of maleic hydrazide residues on sales in West Germany, 80–1827
effect of mechanization on laborers in United States, 80–1550
effect of price and quality on exports in the United States, 80–1270
effect of price support program on economy in the United States, 80–1282
effect of price support termination, 80–1022
effect of Roper Organization report, 80–0861, 80–0862
effect of tariff rates on exports in the United States, 80–1549
effect of taxation on cigarette consumption and revenues in Kentucky, 80–1032
effect on economy in Kentucky, 80–1019
effect on economy in the United States, 80–1023, 80–1027, 80–1034
effect on population in the United States, 80–1020
effect on producers in Kentucky, 80–1024
in Egypt, 80–1553
exports of Burley tobacco in the United States, 80–1033
factors affecting marketing, 80–1551
funding of smoking and health research, 80–1711
in Great Britain, 80–1301
international oligopoly, 80–1548
in Italy, 80–1556
leaf exports in the United States, 80–1828
low-tar cigarette sales in the United States, 80–1555
management and economics in Turkey, 80–0178
marketing in Third World countries, 80–1843
1979 Maxwell Report on cigar sales, 80–0843
in Nigeria, 80–1574
outlook in the United States, 80–1286
in Pakistan, 80–1824
policy for the 1970's in the United States, 80–1061, 80–1063
political support in the United States, 80–0189
production of blond vs. dark varieties in France, 80–1272
production quotas for flue-cured tobacco in the United States, 80–1035
production trends in Europe, 80–0445
production units in the United States, 80–1025
research needs for production of quality tobacco, 80–1289
role of Tobacco Board in India, 80–0842, 80–0844
sales in Great Britain, 80–1554
sales in health-related businesses, 80–1547
sales of chewing tobacco in the United States, 80–1273
sales of low-tar cigarettes in Canada, 80–1036
sales trends of cigarettes, cigarillos, and cigars in Great Britain, 80–0176
socioeconomic status of farmers in Kentucky, 80–1038
in Spain, 80–1274

sponsorship of sports events on television in Great Britain, 80–1311
technology in the United States, 80–1031
tobacco taxation in the United States, 1863-1979, 80–1275
trends in consumption and production in the United States, 80–1029
trends in manufacturing and processing practices in the United States, 80–1015
worldwide production figures for 1978, 80–0179
worldwide trends in cigarette sales, 80–1271

TOBACCO LEAF COMPONENTS
antigens in extracts, 80–0262
characterization of glycophosphoceramide, 80–0224
determination of fatty acids, 80–1084
determination of nitrate and sulfate levels, 80–0704
determination of nitrogen content, 80–0021
determination of phenols in Natu tobacco, 80–1325
determination of trace metals in Iraqi vs. United States tobacco varieties, 80–0036
effect of aging and curing, 80–1258
effect of agricultural practices, 80–0201, 80–1591
effect of agricultural practices on sterol content, 80–0242, 80–0243
effect of calcium fertilizer, 80–0008
effect of curing, 80–1816
effect of curing on alkaloid, amino acid, carbonyl, and protein content, 80–0012
effect of environmental factors on chemical composition, 80–0005
effect of fertilization, 80–0190, 80–0197, 80–0202, 80–0205
effect of fertilization and seasonal variation, 80–0003
effect of fertilization with chlorine, 80–0004
effect of fertilization with magnesium on nitrogen and protein content, 80–1072
effect of fertilization with nitrogen on nicotine content in chewing tobacco, 80–0686
effect of flue-curing on chlorogenic acid, flavonoid, and nicotine distribution, 80–1542
effect of flue-curing on levels in Indian varieties, 80–1263
effect of flue curing on lipid levels, 80–1543
effect of foliar nutrition, 80–0200
effect of homogenized leaf curing, 80–1261
effect of irrigation on alkaloid and rutin levels, 80–0684
effect of irrigation on chlorine levels, 80–0195
effect of maturation stage, 80–0697
effect of nutrient deficiency in tobacco, 80–1315
effect of ozone on lipid fraction, 80–1819
effect of plant density on lipid levels, 80–0874
effect of planting time, 80–1067
effect of potato virus Y infection, 80–0209, 80–0210
effect of selective breeding on constituent levels, 80–0867
effect of varieties on constituent levels, 80–1086
effect on tobacco smoke composition, 80–0867
in flue-cured varieties in Pakistan, 80–0204
fluroide and sugar content, 80–0239
identification of *N*′-formylanabasine and *N*′-formylanatabine, 80–1601
isolation of phosphosphingolipids, 80–1600
mechanical method for reduction of lipids, 80–0549
nicotine transfer rate to smoke, 80–1324
paraffinic hydrocarbons in *Natu* tobacco, 80–0031
polyphenol content, 80–0690
in Taiwan vs. United States tobacco varieties, 80–0025
use of protein as food, 80–0443

TOBACCO MOSAIC VIRUS
identification in German cigarettes, 80–0286

Tobacco pigments *See* **PIGMENTS, TOBACCO**

TOBACCO, PIPE
fluoride and sugar content, 80–0696
production and sales in the United States, 80–1829

TOBACCO PROCESSING
determination of cut tobacco distribution, 80–1018
effect of acids on lead-210 and polonium-210 content, 80–1541
effect of boiling on reduction of carbon monoxide, nicotine, nitrogen dioxide, and tar, 80–0547
effect of boiling with potassium on nicotine, tar, and resin content, 80–1814
effect of catalase and hydrogen peroxide on expansion, 80–0530, 80–0531
effect of dialdehydes on stiffness and filling capacity, 80–0519
effect of dicarboxylic acids and derivatives on expansion, 80–0500
effect of dust and fiber size on aroma and flavor, 80–1546
effect of expansion and fiberization on nicotine and tar levels, 80–0499
effect of fermentation on nicotine content in Italian variety, 80–1260
effect of pectin as filling agent, 80–0528
effect of pullulan and derivatives as coating and binding agents for shaped tobacco, 80–0520
effect of reconstitution vs. expansion on smoke composition, 80–0174
effect of resin as a strengthening agent in expansion, 80–0532
effect of wilt-accelerating agents on curing and drying, 80–0474
effect of zinc in evaluation of hydrophobic coatings in reconstituted tobacco, 80–0498
effect on carcinogenicity reduction, 80–0460
effect on ketone and phenol content in tobacco, 80–1074

filter rod manufacture by split rod technique, 80–0535
hydrophobic coating for reconstituted tobacco products, 80–0502
laminated tobacco foil production, 80–0484
lipid extraction, 80–1545
metal oxides and oxyhydrates in cigarette filters and tobacco as filtering agents, 80–1807
potassium nitrate removal, 80–0555
in production of less hazardous cigarettes, 80–1846
sodium carboxymethyl cellulose as binder, 80–1265

TOBACCO PYROLYSIS PRODUCTS
carbon oxide formation, 80–0868
in cigarette smoke, effect of cigarette paper, 80–0033
in cigarette smoke, effect of filters, 80–0029
determination of alkylating activity, 80–1320
determination of nicotine degradation compounds, 80–0708
effect of ozone on aromatic hydrocarbons, 80–1819
effect of plant density, 80–0874
effect of polysaccharide wrapper in cigarettes and cigars, 80–0509
effect on nitrogenous compound content in cigarette smoke, 80–0022

TOBACCO, RECONSTITUTED
effect of foamed sheet on cigar aroma and flavor, 80–0497
effect of hydrophobic coating on aroma, flavor, and quality, 80–0502
tamarind gum adhesive as binding agent, 80–0534
zinc as tracer for evaluation of hydrophobic coatings, 80–0498

Tobacco smoke *See* SMOKE, TOBACCO

TOBACCO SUBSTITUTES
aliphatic acid esters and carbohydrates, 80–0659
alkaline inorganic matter for increased nicotine delivery, 80–0630
β-1, 3-glucan-type polysaccharides, 80–0641
boric oxide or boron oxyacids with organic binder and metal salt constituents, 80–0648
calcium-alginate and cellulosic fibers, 80–0644
calcium-alginate fibers, 80–0645
carbohydrates and aldol condensation products with inorganic filler, 80–0635
carbon-filled paper, 80–0642
carbon monoxide and tar content in herbal cigarettes in Australia, 80–1090
cellulose binders and water insoluble inorganic fillers, 80–0640
chitin as tobacco extender, 80–0631
coffee cherries, 80–0638
Cytrel vs. tobacco toxicity tests, 80–1337
effect of boron compounds and tobacco fines on ash blooming, 80–0647
effect of Cytrel on smoke constituent levels, 80–1544
effect of protein on smoke aroma and flavor, 80–0629
effect of smoke on pulmonary alveolar macrophages in rats, 80–0732
fibrous carbonaceous fuel, 80–0636
gramineous plants and bean and nut shells, 80–0632
herbal leaves in non-nicotine cigarettes, 80–0782
inorganic salts, 80–0634
nicotine chewing gum in cessation of smoking, 80–1535
nicotine in formulation, 80–0658
peanut shells, 80–0639
plant cellulosic materials, 80–0646
plant parts in regenerated tobacco production, 80–0633
popped corn, 80–0651
puffed barley, 80–0655
puffed millet, 80–0652
puffed milo, 80–0649
puffed oats, 80–0656
puffed rice, 80–0650
puffed rye, 80–0657
puffed triticale, 80–0654
puffed wheat, 80–0653
recommendations of the Hunter Committee for use of Cytrel and New Smoking Material, 80–0856
sales in Great Britain, 80–0177
sodium carbonate for increased nicotine delivery, 80–0630
thermally degraded carbohydrates, 80–0628, 80–0643
thermally degraded cellulosic material, 80–0637

TOBACCO VARIETIES
blond vs. brown tobacco consumption in pancreatic neoplasm patients, 80–1409
carotenoid-related compound in Japanese variety, 80–0240
chemical composition of Taiwan vs. United States varieties, 80–0025
determination of acids and phenols in cigarette smoke, 80–1598
determination of alcohols, carbohydrates, and diterpenes in Bulgarian oriental variety, 80–0225
determination of carotenoid- and thunberganoid-related compounds in Burley variety, 80–0694
determination of dihydro-γ-pyrone content in Burley vs. Oriental vs. Virginia varieties, 80–1076
determination of phenols in Natu tobacco, 80–1325
effect of aging, curing, and fertilization on polyphenol content in flue-cured varieties, 80–0840
effect of agricultural practices on polyphenol content in chlorophyll-deficient varieties, 80–1071
effect of breeding on alkaloid content and ash formation, 80–0237
effect of climate and soil on chemical composition, quality, and yield in Zambia, 80–0683

effect of enzyme activity on nicotine and nornicotine content, 80–1381
effect of fermentation and topping on nicotine content in Italian variety, 80–1260
effect of fertilization on agronomic and chemical characteristics, 80–1313
effect of flue-curing on leaf components in Indian varieties, 80–1263
effect of potato virus Y infection on quality and yield of Burley tobacco, 80–0209, 80–0210
effect of sucker control on agronomic and chemical characteristics of Burley variety, 80–0206
effect on chemical and physical characteristics of cigarette smoke, 80–0223
effect on nicotine, anabasine, and nornicotine content in tobacco leaf and smoke, 80–1086
effect on nicotine, carbon monoxide, and tar content in tobacco leaf and smoke, 80–1086
effect on tar content in tobacco smoke, 80–0687
exports of Burley variety in the United States, 80–1033
growth characteristics and yield of flue-cured varieties in Pakistan, 80–0204
identification of *N'*-formylanabasine and, 80–1601
identification of musk compounds in Oriental variety, 80–0216
identification of solanascone in Burley variety, 80–0695
leaf components in Pakistan variety, 80–0204
lipid levels in green vs. pale yellow varieties, 80–0709
metabolism of nicotine and nornicotine in wild species of *Nicotiana*, 80–0273
pigment levels during maturation in Indian varieties, 80–1077
production of blond vs. dark varieties in France, 80–1272
smoke condensate constituent levels, 80–0876
terpenoids in Greek variety, 80–0211, 80–0213
trace metal levels in varieties in Yugoslavia, 80–0692
trace metals in Iraqi vs. United States varieties, 80–0036
worldwide production figures for 1978, 80–0179

TONGUE DISEASES
glossal central papillary atrophy in smoking vs. nonsmoking diabetics, 80–1742

TRACHEA
(*See also* **RESPIRATORY SYSTEM)**
effect of carbon monoxide, nitrogen dioxide, and sulfur dioxide in squirrels, 80–0062
effect of dimethylbenz(*a*)anthracene on epithelium in rats, 80–1121
effect of nicotine on contractility, 80–0072
effect of transplacental *N*-nitrosodiethylamine on metabolism in fetus in hamsters, 80–1134

TRACHEAL MUCOSA
effect of adrenergic agonists in bronchitics, 80–0950
effect of dimethylbenz(*a*)anthracene on histopathology in rats, 80–1660, 80–1661

TRACHEAL NEOPLASMS
(*See also* **RESPIRATORY TRACT NEOPLASMS)**
dynamics of carcinogenesis of dimethylbenz(*a*)anthracene in rats, 80–0065
induced by benzo(*a*)pyrene in rats, 80–1120
induced by dimethylbenz(*a*)anthracene in rats, 80–0065, 80–0729, 80–1660, 80–1661

TRACHEOBRONCHIAL EPITHELIUM
(*See also* **BRONCHIAL EPITHELIUM)**
carcinogenicity of benzo(*a*)pyrene and diol-epoxide I in rats, 80–0741
effect of benzo(*a*)pyrene in hamsters, 80–0281
effect of dimethylbenz(*a*)anthracene in rats, 80–0729
effect of formaldehyde in hamsters, 80–0281
effect of smoke inhalation in guinea pigs, 80–0717
multinucleated cells in smoking vs. nonsmoking neoplasm patients, 80–0758

TRIGLYCERIDE LEVELS
(*See also* **FATTY ACIDS)**
effect of coronary disease intervention program, 80–1446
effect of smoke inhalation in baboons, 80–1649
effect of smoking, 80–1218
in smoking vs. nonsmoking adolescents, 80–0364
in smoking vs. nonsmoking diabetics, 80–1232

ULCER, PEPTIC
(*See also* **GASTROINTESTINAL DISEASES)**
effect of cimetidine in smokers vs. nonsmokers, 80–0139
effect of cimetidine and pirenzepine on healing rate in smoking vs. nonsmoking duodenal ulcer patients, 80–1482
effect of smoking on healing rate in patients treated with cimetidine and sucralfate, 80–1744
gastritis in smoking vs. nonsmoking patients, 80–0806
pepsin 1 secretion in smoking vs. nonsmoking patients, 80–0993
site in smokers vs. nonsmokers, 80–1489
smoking and, 80–1746
smoking and incidence in psychiatric clinic patients, 80–1049

URANIUM
(*See also* **METALS)**
determination of content in Indian cigarettes, 80–1596
and smoke inhalation, effect on pulmonary disease incidence in animals, 80–0284
in tobacco varieties in Yugoslavia, 80–0692

URIC ACID LEVELS
- effect of coronary disease intervention program, 80–1446
- effect of smoking, 80–1624

URINE
- cadmium levels in smokers vs. nonsmokers, 80–1105, 80–1634
- determination of cotinine and nicotine levels, 80–0219
- effect of high- vs. low-nicotine cigarettes on catecholamine levels, 80–1505
- effect of smoking and alcohol consumption on 17-ketosteroid content, 80–1488
- effect of smoking and pH on psychomotor performance, 80–1530
- effect of smoking on cotinine and nicotine levels in males vs. females, 80–1656
- effect of smoking on mutagenicity in *Salmonella typhimurium*, 80–0283, 80–1140
- estrogen:creatinine ratio in prediction of fetal complications in smokers vs. nonsmokers, 80–1737
- mutagens in smokers vs. nonsmokers vs. cirrhotics, 80–1347
- nicotine content in post-mortem samples, 80–0061
- thioether levels in smokers vs. nonsmokers, 80–1140

UROGENITAL NEOPLASMS
- effect of smoking on incidence in phenacetin abusers, 80–1400

UROGENITAL SYSTEM
- (*See also* **PROSTATE; SPERMATOZOA; UTERUS**)
- effect of nicotine on enzyme activity in epididymis and testes in rats, 80–1340
- effect of nicotine on testes in rats, 80–0905
- effect of smoking and alcohol consumption on testes, 80–1488

UTERINE NEOPLASMS
- smoking habit and socioeconomic status in cervix neoplasm patients, 80–1407

UTERUS
- benzo(*a*)pyrene metabolism in myometrium and ovaries, 80–1139
- effect of nicotine on acidosis and hypoxia incidence in fetus in monkeys, 80–1384
- effect of nicotine on blastocyst implantation in rats, 80–0256
- effect of nicotine on blood circulation and pressure in monkeys, 80–1384
- effect of nicotine on blood circulation in pregnant rats, 80–1338
- hysterectomy prevalence in smokers vs. nonsmokers, 80–1231

VASCULAR DISEASES
- (*See also* **ARTERIAL DISEASES; CARDIOVASCULAR DISEASES**)
- smoking and, 80–0755
- smoking habit in myocardial infarct patients and, 80–0794

Ventilatory function tests *See* **RESPIRATORY FUNCTION TESTS**

VIRUSES
- effect of potato virus Y infection on quality and yield of Burley tobacco, 80–0209, 80–0210
- effect of smoke inhalation on immunity to respiratory tract infections in mice, 80–1639
- interactive effect with cigarette smoke in lung neoplasm etiology, 80–0285

VISION
- (*See also* **EYES**)
- effect of age in smokers vs. nonsmokers, 80–1483
- effect of carbon monoxide on visual-manual performance, 80–1131
- effect of nicotine on flicker fusion, 80–0064
- effect of stimulation on electroencephalogram in smokers vs. nonsmokers vs. abstainers, 80–1632

Vital capacity measurements *See* **RESPIRATORY FUNCTION TESTS**

VITAMIN A LEVELS
- effect of smoke inhalation on larynx in hamsters, 80–0901
- effect on relative risk for bladder neoplasms, 80–0088
- effect on relative risk for lung neoplasms in heavy vs. light smokers, 80–0089

VITAMIN C LEVELS
- in cessation of smoking, 80–0667
- in treatment of nicotine withdrawal symptoms, 80–0664

VITAMIN LEVELS
- nicotinamide, pyridoxine, and thiamine in cessation of smoking, 80–0667
- vitamin B in treatment of nicotine withdrawal symptoms, 80–0664

WITHDRAWAL CLINICS
- (*See also* **CESSATION OF SMOKING; REDUCTION OF SMOKING**)
- Atlanta Lung Association program, 80–0432
- programs in hospitals, 80–1284
- programs in the workplace, 80–1252

Withdrawal symptoms *See* **BEHAVIOR; CESSATION OF SMOKING; REDUCTION OF SMOKING; WITHDRAWAL CLINICS**

WOMEN
  (*See also* **SEX RATIO)**
  attitudes toward health risk of smoking, 80–1792
  bronchial neoplasms in smokers vs. nonsmokers in Hong Kong, 80–1670
  cadmium, lead, and mercury levels in placenta in smokers vs. nonsmokers, 80–0386
  cardiovascular disease mortality in Switzerland, 80–0076
  effect of cessation of smoking methods, 80–0166
  effect of cessation of smoking on body weight, 80–1485, 80–1722
  effect of role stress on smoking habit in academic psychologists, 80–0144
  effect of smoking, alcohol consumption, and oral contraceptives on phenytoin metabolism in epileptics, 80–1342
  effect of smoking and aging on cardiovascular disease risk in oral contraceptive users, 80–1719
  effect of smoking and coffee consumption on myocardial infarct risk, 80–1727
  effect of smoking and employment status on coronary disease risk, 80–0787
  effect of smoking and occupation on relative risk for neoplasms, 80–0082
  effect of smoking and oral contraceptives on hemorrhagic stroke risk, 80–0803
  effect of smoking on age at onset of menopause, 80–1230
  effect of smoking on body weight, 80–1485
  effect of smoking on body weight during pregnancy, 80–0387
  effect of smoking on breast neoplasm mortality ratio in Japan, 80–0302
  effect of smoking on coronary disease incidence in Great Britain, 80–0983
  effect of smoking on placental characteristics in blacks vs. whites, 80–0391
  effect of smoking on subarachnoid hemorrhage risk in oral contraceptive users, 80–0128
  high- vs. low-tar cigarette preference in lung neoplasm patients, 80–0930
  hysterectomy prevalence in smokers vs. nonsmokers, 80–1231
  lung neoplasm mortality in Sweden, 80–0303
  myocardial infarct risk in smoking vs. nonsmoking oral contraceptive users, 80–1718
  risk factors for coronary disease, 80–0117
  smoking and hyperlipidemia in oral contraceptive users, 80–1199
  smoking and lung neoplasms, 80–0182
  smoking and oral contraceptives in myocardial infarct patients, 80–0346
  smoking and other risk factors for myocardial infarct in menopausal women, 80–1704
  smoking as risk factor for coronary disease, 80–0977
  smoking habit during pregnancy, 80–0818, 80–1470
  smoking habit in hospital employees in the United States, 80–1516
  smoking habit in planned vs. unplanned pregnancy, 80–1524
  smoking habit in the United States, 80–1569
  smoking habit in young angina pectoris and myocardial infarct patients, 80–1724

U.S. GOVERNMENT PRINTING OFFICE : 1981 O - 344-691

best copy available

U.S. DEPARTMENT OF
HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health
Rockville, Maryland 20857

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK

Public Health Service Bibliography Series No. 45

MAY 1981